PDF hosted at the Radboud Repository of the Radboud University Nijmegen

The following full text is a publisher’s version.

For additional information about this publication click this link.
http://hdl.handle.net/2066/80259

Please be advised that this information was generated on 2019-03-24 and may be subject to change.
Cardiopulmonary Resuscitation: Are two hands (really) enough?

A modeling approach to CPR

Gerrit J. Noordergraaf
Cardiopulmonary Resuscitation: Are two hands (really) enough?

A modeling approach to CPR
Colophon

Dissertation University Medical Center Nijmegen, St Radboud, Nijmegen, The Netherlands
ISBN: 978-90-9024557-7

The studies presented in this dissertation were supported by (alphabetical order): Abbott NV. (Louvain-la-Neuve, B); Health Affairs, Ltd. (Berkhamsted, UK); MediScore (Bodegraven, NL); Laerdal, Inc (Brussels, B); North Med Syst and Consultancy (Hilvarenbeek, NL) Philips Research (Eindhoven, NL); Zoll Medical (Chelmsford, MA, USA);


© 2009 G.J. Noordergraaf, Diessen, The Netherlands
No part of this publication may be reproduced, stored in a retrieval system, or transmitted in any form or by any means without prior permission of the author, or, when appropriate, of the publisher of the Journal.

Cover Illustration: Don Quixote and The Mirrored Knights. From The Man of LaMancha. Photograph (Ms. Judy Potter) used with written permission by the Willows Theater Company, 2005.

Printing: DekoVerdivas, De Posthoornstraat 9, 5048 AS Tilburg, Telefoon: 013 -463 13 35
Cardiopulmonary Resuscitation: are two hands enough?
A modeling approach to CPR

(met samenvatting in het Nederlands).

Een wetenschappelijke proeve op het gebied van de Medische Wetenschappen

Proefschrift
ter verkrijging van de graad van doctor aan de Radboud Universiteit Nijmegen op gezag van de
rector magnificus prof.mr. S.C.J.J. Kortmann volgens besluit van het college van decanen.

In het openbaar te verdedigen op vrijdag 2 oktober 2009 om 13.00 uur precies
door

Gerrit Jan Noordergraaf

geboren op 7 oktober 1959 te Zeist
Promotores:

Prof.dr. G.J. Scheffer  
Prof.dr. A. Noordergraaf, University of Pennsylvania, Philadelphia, PA USA

Manuscriptcommissie:

Prof.dr.ir. H.A. van Swieten  
Prof.dr. J.G. van der Hoeve  
Prof.dr. J.A. Roukema, University of Tilburg, & St. Elisabeth Hospital, Tilburg.

Promotiecommissie:

Prof.dr. H.J.J.M. Berden, University of Tilburg, & St. Elisabeth Hospital, Tilburg  
Dr. J. Lerou  
Prof.dr.ir. W.H.A. Schilders, Technical University Eindhoven, Eindhoven  
Prof.dr. J. Smeets  
Prof.dr.ir. P. Woerlee, Philips Research. Biomedical Sensor Systems, Eindhoven

Paranymphs:

P.F.J. van Berkom RN, MSc  
Ms. A. Venema, RN
Table of contents
# Table of contents

<table>
<thead>
<tr>
<th>1</th>
<th>Challenge and mission statement</th>
<th>p.V-IX</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.1</td>
<td>Introduction and relevance</td>
<td>p.1</td>
</tr>
<tr>
<td>1.2</td>
<td>The challenge</td>
<td></td>
</tr>
<tr>
<td>1.3</td>
<td>Mission statement</td>
<td></td>
</tr>
<tr>
<td>1.4</td>
<td>Outline of the dissertation</td>
<td></td>
</tr>
<tr>
<td>1.5</td>
<td>Endpoints for the dissertation</td>
<td></td>
</tr>
<tr>
<td>1.6</td>
<td>References</td>
<td></td>
</tr>
</tbody>
</table>

## PART I: FUNDAMENTALS IN CPR

<table>
<thead>
<tr>
<th>2</th>
<th>Technical issues in and around CPR</th>
<th>p.11</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.1</td>
<td>Introduction</td>
<td></td>
</tr>
<tr>
<td>2.2</td>
<td>Practical CPR anno 2007</td>
<td></td>
</tr>
<tr>
<td>2.3</td>
<td>Data and evidence in resuscitation and neurological outcomes</td>
<td></td>
</tr>
<tr>
<td>2.4</td>
<td>Moving blood in CPR: introductory aspects and theory</td>
<td></td>
</tr>
<tr>
<td>2.5</td>
<td>Measurements and monitoring: physiology in CPR</td>
<td></td>
</tr>
<tr>
<td>2.6</td>
<td>Monitoring techniques</td>
<td></td>
</tr>
<tr>
<td>2.7</td>
<td>Summary and conclusion</td>
<td></td>
</tr>
<tr>
<td>2.8</td>
<td>References</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>3</th>
<th>A historical, mechanistic, approach to chest compressions: facts and polite fiction</th>
<th>p.41</th>
</tr>
</thead>
<tbody>
<tr>
<td>3.1</td>
<td>Introduction</td>
<td></td>
</tr>
<tr>
<td>3.2</td>
<td>The later part of the first era: Egypt to 1960</td>
<td></td>
</tr>
<tr>
<td>3.3</td>
<td>The second era: 1960-1962 (rediscovery and application)</td>
<td></td>
</tr>
<tr>
<td>3.4</td>
<td>The third era: 1963-1968 (skepticism)</td>
<td></td>
</tr>
<tr>
<td>3.5</td>
<td>The fourth era: 1969-1976 (acceptance and complacency)</td>
<td></td>
</tr>
<tr>
<td>3.6</td>
<td>The fifth era: 1976-1990 (return of OCCR, redirection and refinement)</td>
<td></td>
</tr>
<tr>
<td>3.7</td>
<td>The sixth era: 1991-2004 (formalization and evidence-base)</td>
<td></td>
</tr>
<tr>
<td>3.8</td>
<td>The seventh era: 2005 – (return to core task)</td>
<td></td>
</tr>
<tr>
<td>3.9</td>
<td>Adjuvant techniques to generate flow</td>
<td></td>
</tr>
<tr>
<td>3.10</td>
<td>Mechanical chest compressions</td>
<td></td>
</tr>
<tr>
<td>3.11</td>
<td>Summary</td>
<td></td>
</tr>
<tr>
<td>3.12</td>
<td>References</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>4</th>
<th>Thoracic CT-scans and cardiovascular models: the effect of external force in CPR</th>
<th>p.89</th>
</tr>
</thead>
<tbody>
<tr>
<td>4.1</td>
<td>Introduction</td>
<td></td>
</tr>
<tr>
<td>4.2</td>
<td>Methods and materials</td>
<td></td>
</tr>
<tr>
<td>4.3</td>
<td>Results</td>
<td></td>
</tr>
<tr>
<td>4.4</td>
<td>Discussion</td>
<td></td>
</tr>
<tr>
<td>4.5</td>
<td>Conclusions</td>
<td></td>
</tr>
<tr>
<td>4.6</td>
<td>References</td>
<td></td>
</tr>
</tbody>
</table>
PART II: MODELING

5 Models and modeling and their potential in circulatory research p.105
  5.1 Introduction
  5.2 Modeling and simulations
  5.3 Simple terms in modeling
  5.4 Early models in cardiovascular research
  5.5 The development of cardiovascular system models based on sophistication
  5.6 Experiments on animals models of different kinds
  5.7 Models in CPR: under appreciation of a potential source of insight
  5.8 Summary
  5.9 References

6 Layout, initial conditions and description of the Donders models p.119
  6.1 Introduction
  6.2 Anatomical and symbolic layout of the Donders model (DII)
  6.3 The cardiac chambers
  6.4 Valvular opening and closure
  6.5 Arterial system
  6.6 Venous system
  6.7 Controls
  6.8 Adaptations for CPR
  6.9 Initial conditions
  6.10 Summary
  6.11 References

7 Modeling in cardiopulmonary resuscitation: Pumping the heart p.133
  7.1 Introduction
  7.2 Methods and procedures
  7.3 Results
  7.4 Discussion
  7.5 Conclusion
  7.6 References

8 The Donders model of the circulation is normo- and pathophysiology p.155
  8.1 Introduction and motivation
  8.2 The development of cardiovascular modeling
  8.3 Impedance defined (Z) flow
  8.4 The Donders model
  8.5 Results
  8.6 Discussion
  8.7 Conclusions
  8.8 References

9 Supporting the use of modeling in CPR: things (not) in evidence p.189
  9.1 Introduction
  9.2 Qco: a non issue
  9.3 Stroke volume in relation to compression force
  9.4 Volume sequestration
PART III: PRACTICAL APPLICATIONS OF MODELING MECHANISMS IN CPR

10 Physical modeling in CPR: simple and complex  p.205
  10.1 Introduction
  10.2 The physical model
  10.3 Are there requirements for manikins and how they impact skills
  10.4 Integrated resuscitation simulators should retain basic options
  10.5 Is the model needed for skills?
  10.6 The manikin and the model
  10.7 Conclusion
  10.8 References

11 Training needs and qualifications  p.221
  11.1 Abstract
  11.2 Introduction
  11.3 Materials and methods
  11.4 Results
  11.5 Discussion
  11.6 Conclusions
  11.7 Acknowledgements
  11.8 References

12 Applicability of insights in force and pressure: A CPR-Ezy study  p.233
  12.1 Introduction
  12.2 Materials and methods
  12.3 Results
  12.4 Discussion
  12.5 Conclusions
  12.6 Acknowledgements
  12.7 References

13 Work and force in CPR: a practical discussion  p.247
  13.1 Introduction
  13.2 Materials and methods
  13.3 Results
  13.4 Discussion
  13.5 Conclusions
  13.6 References

14 The effects of mattresses and backboards on in-hospital CPR  p.257
  14.1 Introduction
  14.2 Materials and methods
  14.3 Results
  14.4 Discussion
  14.5 Conclusions
  14.6 References
<table>
<thead>
<tr>
<th>Chapter</th>
<th>Title</th>
<th>Pages</th>
</tr>
</thead>
<tbody>
<tr>
<td>15</td>
<td>The accelerometer as a device in CPR</td>
<td>p.273</td>
</tr>
<tr>
<td>15.1</td>
<td>Introduction</td>
<td></td>
</tr>
<tr>
<td>15.2</td>
<td>Materials and methods</td>
<td></td>
</tr>
<tr>
<td>15.3</td>
<td>Results</td>
<td></td>
</tr>
<tr>
<td>15.4</td>
<td>Discussion</td>
<td></td>
</tr>
<tr>
<td>15.5</td>
<td>Conclusions</td>
<td></td>
</tr>
<tr>
<td>15.6</td>
<td>References</td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>Conclusions and perspectives:</td>
<td>p.283</td>
</tr>
<tr>
<td>16.1</td>
<td>Introduction</td>
<td></td>
</tr>
<tr>
<td>16.2</td>
<td>Chest compressions: this dissertation</td>
<td></td>
</tr>
<tr>
<td>16.3</td>
<td>An adjunct to developing thinking and practice</td>
<td></td>
</tr>
<tr>
<td>16.4</td>
<td>Future perspectives</td>
<td></td>
</tr>
<tr>
<td>16.5</td>
<td>Conclusions</td>
<td></td>
</tr>
<tr>
<td>16.6</td>
<td>References</td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>Summary</td>
<td>p.291</td>
</tr>
<tr>
<td>18</td>
<td>Samenvatting (summary in Dutch)</td>
<td>p.299</td>
</tr>
</tbody>
</table>

Acknowledgements p.307
Curriculum vitae p.311
List of publications p.315

Appendices
Challenge and mission statement
Chapter 1: Challenge and mission statement

Table of contents

1 CHALLENGE AND MISSION STATEMENT ................................................................. 1

1.1 Introduction and relevance .................................................................................. 3
1.2 The challenge ...................................................................................................... 3
1.3 Mission statement ............................................................................................... 5
1.4 Outline of the dissertation .................................................................................... 6
1.5 Endpoints for this dissertation .............................................................................. 6
1.6 References ......................................................................................................... 8
1.1 Introduction and relevance

Sudden collapse, from circulatory or respiratory arrest has become an accepted fate of life in the Western world even though it often culminates in immediate death. In the Netherlands alone more than 2000 people year\(^{-1}\) million\(^{2}\) inhabitants suffer such an abrupt cardiovascular collapse.\(^{2}\)

The causes of these collapses are legion and include principal pump failure (acute myocardial infarction, arrhythmias), respiratory collapse (asphyxia), trauma, and disturbances in the internal environment (such as electrolyte disorders),\(^{3}\) and were even suggested to be influenced by lunar activity.\(^{4}\) Those who do not die immediately require prompt, adequate and intensive therapy in order to have a chance at survival. A procedure in this immediate care process, which has become an entity in and of itself, is known as cardiopulmonary resuscitation (CPR). Two hands were suggested to be enough to save a life. CPR has been further structured in a ‘chain of events’ each of which, independent of the others, may play an essential role in the overall outcome.

Since the 1960’s CPR has been deemed teachable to and applicable by health care providers as well as by lay persons. Involvement of lay people has also been claimed to improve the chance of survival by a margin of about 50% in comparison with those not receiving bystander CPR, both when ventricular fibrillation is present as well as in general.\(^{5,6,7,8}\) Treatment of sudden cardiac death with cardiopulmonary resuscitation, however, suffers from an impressive failure rate which has remained disconcertingly stable from early reports to the current day.\(^{9}\)

The relevance of interest into the etiology, prevention, management and treatment of sudden cardiopulmonary collapse needs little elaboration. With 1.2 million deaths year\(^{-1}\) in the United States and Europe alone, sudden cardiac death (SCD) has become a primary cause of early death, surpassing deaths due to trauma and cancer.\(^{10}\) Fully 50% of the first presentation of coronary artery disease is sudden death.\(^{11}\) Over the whole population with SCD group, researchers have suggested that two-thirds received some form of resuscitation attempt.\(^{12}\) While success rates of treatment were quoted as 70% to 94% in the 1960’s\(^{13,14}\) work up through 2000 cites about 30% as being a realistic outcome for initial survival and less than 15% at hospital discharge.\(^{15,16}\) However, since 2000, systematic reviews of definitive survival suggest percentages of up to 15% survival if found in ventricular fibrillation,\(^{17}\) with this being only 5-10% survival overall for those treated by emergency medical personnel.\(^{18,19}\) When projected on an adult, in-hospital population, a survival to discharge of 17% has been reported.\(^{20}\) While saving ‘hearts too good to die’ is the potential for good CPR, those hearts too poor to survive escape objectification.

While the equivalent of billions of US dollars has been spent worldwide on animal, clinical and didactic research, many aspects of CPR remain unclear. In this context, and strongly implemented as of the 1992 Standards and Guidelines,\(^{21}\) evidence is being sought to explain the success and failure of CPR.\(^{9}\) Consistently, however, research has focused on ‘observing’ the conduct of the circulation in animal experiments and applying this to humans. In line with classical investigation, pressure gradients such as the coronary perfusion pressure (CPP) instead of flows have been used. Modeling, both physical and mathematical, has only recently achieved some acceptance as an important tool in making choices in CPR, with work by Babbs et al.\(^{22}\) leading to one of the central choices in the 2005 guidelines.

1.2 The challenge

The central challenge in CPR is to understand how to make oxygenated blood move towards and through vital organs despite cardiac arrest. Surprisingly little is understood about how and why
Empirically, emphasis has shifted to ‘less is more’ and simplicity in the skills needed for CPR. Over the last decade, this seemed to move to the background. Mathematical scrutiny and many features remain speculative; although performing chest compressions using a given technique may support one theory more than the other. In practice, many suffice with the comment that these mechanisms exist and will ‘come and go’ during the course of a CPR attempt. Even more strongly, the discussion about pump theories has, over the last decennia, seemed to move to the background.

CPR is supposed to work. Doctrine as well as contradictions prevail, some of which seem intrinsic to the issue at hand.

Some examples:

- Bystander CPR has been suggested to improve the probability of survival by 50% while at the same time the quality of bystander and professional basic life support skills, such as chest compressions, is demonstrably poor.
- The basic philosophy and practice (of chest compressions) in CPR have remained unchanged for more than 45 years. While different factors, such as age, body mass index (BMI), time since arrest and comorbidity may affect the way the circulation acts, a single, strictly applied CPR technique has been consistently advocated for both laymen and professionals for out-of-hospital as well as in-hospital settings.
- Laboratory techniques, including mechanical compression technology, fail in the field while excelling during bench and animal testing.
- The principal goal of CPR for the different partners in the chain of survival seem subject to debate: is this to create a circulation, achieve ‘return of spontaneous circulation’, or for all, ultimately reduced to achieving good, definitive survival.
- “Teachability” of skills and interventions has become a principal operator in choices for treatment strategies, but does not seem to impact on the quality issue. Has the ‘two hands’ philosophy offered by Kouwenhoven been usurped or undermined or was it too simplistic?

Focusing further on the intent of creating an adequate circulation following sudden cardiac arrest, the total research effort has produced two main, interacting theories regarding the effect of chest compressions and their task in restoration of an organized pump function. Both have led to a considerable number of variations.

These two theories are the cardiac pump theory (1960) with its focus on compression of the heart itself, and the thoracic pump theory (1976) with its focus on creating a general intrathoracic pressure gradient to move blood volume out of the thoracic cavity into the arterial tree. The two theories have common elements and, thus, may not be as distinctly separable as was thought originally. Neither theory has been subjected to detailed, fundamentally oriented, mathematical scrutiny and many features remain speculative; although performing chest compressions using a given technique may support one theory more than the other. In practice, many suffice with the comment that these mechanisms exist and will ‘come and go’ during the course of a CPR attempt. Even more strongly, the discussion about pump theories has, over the last decennia, seemed to move to the background.

Empirically, emphasis has shifted to ‘less is more’ and simplicity in the skills needed for CPR. Basic life support (BLS) and more precisely, the effects of pressures and forces on the chest wall, have once again become recognized as the essential subject in attempting survival. Practical studies, however, continue to demonstrate the poor quality of skills and adjuncts to manual compressions have remained consistently unable to supply evidence of definitive outcome. A procedural approach instead of a step towards a more mechanistic approach has been the result for those less fundamentally oriented.

Are we missing a fundamental link in the Chain of Survival? Has vision on the principal issue been lost, just as the knight who threw himself on his horse and rode away in 36 directions at the same time? Is it possible that the (manual) methods used for chest compressions are so suboptimal that oxygenation of cardiac and cerebral tissue is generally inadequate, except for a lucky few having an unrecognized specific state of being? Why is it that in a patient with a ‘heart too poor to save,’ the circulation cannot be adequately ‘sustained’ and how can this be measured? Even with the recognition from animal and human models that circulatory collapse has dynamic
properties which relate to the condition of the circulation and to the heart itself, no changes have been seen in the technique of basic life support.

Perhaps we are missing a window of opportunity: a thought not unique to research in CPR. Perhaps we should focus on what might be best in humans, working towards a time variable effort during CPR by understanding what may work in driving the circulation with 'restarting a heart that is good enough to restart' as its effect.

1.3 Mission statement

Recognizing that many of the pressure, flow and volume phenomena in CPR are unexplained while most theoretical explanations are poorly correlated with results in humans, this project sets out to query accepted practice by rational analysis of facts in evidence.

The project is designed to return from a procedural to a more analytical modeling approach to application of pressures and forces to the chest and the vascular structures.

This study reaches back to the historical framework and its development over time, evaluating and summarizing these. The manuscript addresses the plethora of literature with specific interest in (hand) position, the depth or force of compression, the compression-relaxation ratio, and the frequency of compressions. These aspects are supposed to lead towards a basic understanding of the underlying mechanisms so as to make clinical suggestions which may improve treatment. These aspects are some of the central psychomotor aspects of CPR: are two hands actually enough? This extensive, but not exhaustive, analysis looks towards handholds in studies directed towards the transmission of pressures and forces into and onto the chest towards the vasculature. It avoids the aspects of outcome and ventilation as autonomous factors but includes adjuvant CPR techniques.

Using these historical references, the dissertation project is designed to develop and apply a mathematical model using modestly lumped parameters with differential-integral-algebraic equations relating variables and parameters, founded in physiology and specifically suitable for CPR investigations. It will integrate fundamental insights, despite their low level of evidence classification (level 7 for modeling, see chapter 2), into what determines whether the circulation can be driven manually, allowing viable outcome after the heart has stopped. Specific attention is focused on the model on the role of compression and of circulatory valves. It researches and discusses aspects of the venous return related to impedance defined circulation under external cardiac compressions. Both the cardiac pump and the thoracic pump theories and their variations are analyzed using the techniques offered by the model. In this dissertation, cardiopulmonary collapse, limited to adult specifications, will be examined as an extreme case within the continuum of circulatory and respiratory states.

The methodology of research will be scrutinized for physiological, experimental and clinical research, as well as for mathematical models. Using these considerations as a foundation, it progresses to evaluation using modern means.

A number of the theoretical insights, which may offer opportunities for (lay) caregiver application, such as using force applied to the chest as a marker for patient-driven resuscitation (PDR), will bring some of the theory to the level of clinical applicability. Central to these studies will be an analytical, critically realistic, modeling approach in quality: what are we (actually) doing, as opposed to what are we purporting to do. The development of feedback as a strategic tool in force to compression quality links the highly theoretical to clinical application.
A consistent effort will be made to understand why treatment thus far has failed to enjoy more success and, if it proves possible, improve success rate.

1.4 Outline of the dissertation

This dissertation consists of three main parts:
Part 1: In this section, the reader is introduced to cardiopulmonary resuscitation, both in its up-to-date pre-hospital and clinical applications (chapter 2). This foundation information will tantalize the reader by its clarity and simplicity, begging the question of the dissertation goals. In chapter 3 this clarity will be challenged, as an array of studies and evidence is reviewed to bring the focus to chest compressions. In this extensive, but non-exhaustive review of circulatory research into CPR, the compression or circulatory aspects may sow seeds of concern. In chapter 4, a study of an at random sample of thoracic CT scans is presented which voices questions regarding the most fundamental assumptions in CPR ‘pump theory’ as well as demonstrating the diversity of compression force issues in current CPR.

Part 2: Introduction of the reader into mechanical and mathematical modeling and its suitability for CPR research as a potential investigative and strategic tool (chapter 5). This chapter lays the groundwork for the introduction and initial conditions (chapter 6) and development of the Donders model as a physiological, open pump (chapter 7) and as a model for CPR (chapter 8). Chapter 9 focuses on the potential usefulness of the model in analysis of effects of compressions, and offers provocative hints of further opportunities.

Part 3: The reader is reintroduced to clinical medicine, with chapter 10 returning the reader to chest compressions as a skill involving manikins as models and simulators. The fact that skill perception and objective measurement of the same may lead to disappointing results is presented in chapter 11. If projection can be reduced by feedback allowing for better effects, it is demonstrated in chapter 12 with the CPREzy as a device. It introduces the effect of patient based feedback supporting the suggestions of limitations, and with chapter 13 allows an analysis to be made of the potential ‘costs’ involved in feedback, both from a modeling point of view. Chapter 14 reports on the impact of another factor on the forces involved in chest compressions: the mattresses used during in-hospital resuscitation and their complexity, and concludes with chapter 15 which introduces the other clinically available feedback device.

In chapter 16 conclusions are brought together from the three parts and the reader is offered a limited number of perspectives and handholds as the 2010 guidelines approach. Chapter 17 presents a brief structured summary of each chapter.

Finally, in the appendices, tables and other general information are provided.

1.5 Endpoints for this dissertation

At the end of this dissertation, the reader should have become convinced that cardiopulmonary resuscitation is a developing, functional, and useful medical treatment strategy, which as a young subspecialty presents itself simply but deals with the most complex patients imaginable. It will enhance understanding of a small segment in resuscitation medicine, the ‘basic’ step of applying forces directly or indirectly to the vasculature in the hope of moving oxygenated blood.

While not directly the goal, improvements or the potential for such improvements is sought. A number of theoretical insights, opportunities for (lay) caregiver application of modeled insights, such as force applied to the chest, will bring theory to the level of clinical applicability. The
development of feedback, i.e. the CPREzy, a device geared towards correlating feedback in force to compression quality, and the PocketCPR, an accelerometer, link theoretical features to clinical application.

This dissertation will focus on the circulation of the adult: the exclusion of outcome studies, medication variables, the extremes of age, are recognized as limitations but also support the opportunity offered by a modeling approach, in that complicating factors can be controlled.

In most final sense, this dissertation sets out to allow the clinician to recognize factors which may influence outcome, and strive to implement this knowledge to improve survival.
1.6 References

16 Parish DC, Dinesh Chandra KM, Dane FC. Success changes the problem: why ventricular fibrillation is declining, why pulseless electrical activity is emerging, and what to do about it. Resuscitation 2003; 58: 31-35.
Chapter 1: Challenge and mission statement

26 Descartes R. Meditationes de prima philosophia, in qua Dei existentia et animae immortalitas demonstratur. 1641.
Technical issues in and around CPR

Chapter 2: Technical issues in and around CPR

This chapter is designed to supply definitions, lay the groundwork, offer insights to the other chapters, and will start out to discuss CPR as it is taught following the 2005 ILCOR guidelines. The reason to offer a "brief course" in CPR is that the manuscript entertains a diversity of technical issues with CPR: suggested to have a large impact of the chance of survival. This chapter will focus on adult life support and limit itself to the main stream of sudden cardiac death, excluding specific cases such as choking, drowning and trauma. While the emphasis is on the teachability of CPR, the reader may be most comfortable, having been "taught" the techniques and procedures without complicating discussions. Initially, it will resemble a basic life support course. In the second part of the chapter, the emphasis will change to an analysis of the pathophysiology during CPR. The concept of impedance defined flow will be introduced as concepts of measurement and monitoring are presented. This from both a clinical as well as a theoretical (modeling) point of view.

Ref: Figure 1: Chain of survival emphasizing both the importance and independence of each link as an uninterrupted nature of chest compressions, causing the emphasis in course work to change.

The (patho-)physiology of the airways & pressure in the thorax

Ultrasound and echocardiographic investigations

Radionuclide injection

Direct or invasive pressure monitoring

The macrostructure of the (human) chest as it relates to CPR

The (patho-)physiology of the airways & pressure in the thorax

Distribution of blood in the circuit

Units in physiology and CPR

Levels of evidence, class of recommendation

Data collection and registration

Measurement and monitoring: Physiology in CPR

Current practice in teaching in BLS

Practical CPR anno 2007

Introduction

The incidence of sudden cardiac death grows to 1.89 per 1000 person-years, with a large variation of up to 30-fold, between clinical populations. Many times each day attempts at resuscitation are performed both by professionals and by laypeople. Just the intervention of performing CPR has been suggested to have a large impact of the chance of survival. Cardiopulmonary resuscitation (CPR) is taught the world over. The relevance of CPR is increasing as the chain of survival. (Figure 1) Emphasis changed to focus on the quality, frequency and...
2.1. Introduction

Cardiopulmonary resuscitation (CPR) is taught the world over. The relevance of CPR is increasing as the incidence of sudden cardiac death grows to 1.89 per 1000 person-years, with a large variation of up to 30-fold, between clinical populations.1 Many times each day, attempts at resuscitation are performed both by professionals and by laypeople. Just the intervention of performing CPR has been suggested to have a large impact of the chance of survival.2

This chapter is designed to supply definitions, lay the groundwork, offer insights to the other chapters, and will start out to discuss CPR as it is taught following the 2005 ILCOR guidelines.3 The reason to offer a ‘brief course’ in CPR is that the manuscript entertains a diversity of technical issues with CPR: the reader may be most comfortable, having been ‘taught’ the techniques and procedures without complicating discussions. Initially, it will resemble a basic life support course. In the second part of the chapter, the emphasis will change to an analysis of the pathophysiology during CPR. The concept of impedance defined flow will be introduced as concepts of measurement and monitoring are presented. This from both a clinical as well as a theoretical (modeling) point of view.

This chapter will focus on adult life support and limit itself to the main stream of sudden cardiac death, excluding specific cases such as choking, drowning and trauma. While the emphasis is on basic life support measures, and more exactly on those mechanisms involved in circulatory motion, the scene is set to include other relevant aspects for basic and professional level CPR.

2.2. Practical CPR anno 2007

The most recent guidelines were introduced in 2005 and broke with more classic doctrine by adapting the chain of survival. (Figure 1) Emphasis changed to focus on the quality, frequency and uninterrupted nature of chest compressions, causing the emphasis in course work to change.

![Figure 1: Chain of survival emphasizing both the importance and independence of each link as well as of the whole](image)

Adult basic life support (BLS) has been defined as “those activities performed to maintain an airway and support breathing and the circulation without the use of equipment other than a protective device”.4 It holds with the original premise by Kouwenhoven et al.5 that “all you need to save a life is two hands”. Even today, basic life support is still performed with a minimum of materials and should be initiated as soon as possible after any sudden collapse. In the in-hospital setting, however, materials such as self-inflating bags have become accepted for use.
Basic life support retains the suggestion that it is designed to maintain the minimum of circulation required for vital organ viability. While for many years it was suggested that the basic aspects of resuscitation were aimed at creating time for therapeutic interventions initiated by physicians and requiring medication, monitors and the like, current thinking has changed, elevating BLS to a therapeutic measure in its own right. Advanced (cardiac) life support (ACLS) may add directed care, but adequate BLS must be maintained until return of spontaneous circulation (ROSC). The essence of CPR and its basic life support aspects, as exemplified by the “Airway-Breathing-Circulation” approach, has remained basically unchanged since the earliest definition. In the 2000 standards and guidelines, emphasis was focused for the first time on a specific item. This focus was on airway and breathing aspects in CPR, and suggested strongly that morbidity and mortality could be avoided by decreasing pressure and flows during ventilation, while in 2005 the focus on the airway was replaced by emphasis on compressions.

### Table 1: Summary of the A-B-C instructions in the Guidelines 2005

<table>
<thead>
<tr>
<th>Prime factor</th>
<th>Global order</th>
<th>Placement of hands</th>
<th>Depth: C/R</th>
<th>Freq. Rhythm</th>
</tr>
</thead>
<tbody>
<tr>
<td>1960</td>
<td>Airway</td>
<td>A-B-C</td>
<td>Lower half</td>
<td>1.5 – 2 inch</td>
</tr>
<tr>
<td>1974</td>
<td>Airway</td>
<td>A-B-C + precord thump</td>
<td>Lower half 1.5 inch p.x.</td>
<td>1.5 – 2 inch 1:1 C/R</td>
</tr>
<tr>
<td>1980</td>
<td>Airway</td>
<td>A-B-C</td>
<td>Lower half</td>
<td>1.5 – 2 inch 1:1 C/R</td>
</tr>
<tr>
<td>1992</td>
<td>EMS/ABC</td>
<td>A-B-C</td>
<td>Lower half</td>
<td>1.5 – 2 inch 1:1 C/R</td>
</tr>
<tr>
<td>2000</td>
<td>Chain of survival</td>
<td>A-B-C C-A-B</td>
<td>Lower half</td>
<td>1.5 – 2 inch 1:1 C/R</td>
</tr>
<tr>
<td>2005</td>
<td>Chain of survival Chest compression</td>
<td>A-B C B</td>
<td>Middle of chest</td>
<td>1.5 – 2 inch 1:1 C/R</td>
</tr>
</tbody>
</table>

The ‘chain of survival’ concept:
- The recognition of distress, acute myocardial infarction and stroke;
- Early entry into the ‘chain of life’ or emergency medical system;
- Primary support of respiration in victims of respiratory arrest (rescue breathing), including recognition and relief of foreign body airway obstruction (FBAO);
- Primary support of respiration and circulation in victims of cardiopulmonary arrest (basic life support with rescue breathing and chest compressions);
- Defibrillation attempts. This involves the use of an automatic external defibrillator (AED).
- Early advanced cardiac life support and post resuscitation care, including mild therapeutic hypothermia to restore the quality of life.

In a practical sense, confusion may exist in what BLS entails and who is performing it. While conforming to the definition above, the general tendency is that the emphasis in BLS lies on chest compressions, and decreasingly on the airway measures. BLS can be performed by laypeople. Not only during the initial period until professional help arrives, but often also after this, as the first team
arriving may consist of only two caregivers. If they judge the BLS (i.e., the chest compressions) to be adequate, typically the chest compressions may be ‘farmed out’ to the lay person.12

2.2.1 Current practice in teaching in BLS
With the presentation of the 2005 Standards and Guidelines,3 basic life supports, as taught to laymen and professionals, may be summarized as listed below. Teaching emphasizes the one-caregiver situation, suggesting that the chance of having two qualified lay caregivers at one scene is small. In effect, this means that the one caregiver must diagnose, alert, ventilate and perform the chest compressions. It also recognizes that the majority of resuscitations are performed on strangers.13 For simplicity’s sake, the description is for a one-caregiver scenario.

- Check for safety: The caregivers are reminded that there is a need to maintain (personal) safety as well as to watch for the safety of other caregivers or patient. The issue of safety has extended itself to include concerns about transmission of (viral) diseases from the patient to the caregiver, as has been anecdotally reported for tuberculosis14 as well as for SARS, even though the use of a bag-valve device was implicated in the latter case.15 Nonetheless, the fear of acquiring an infectious disease, most notably HIV/AIDS, has pervaded BLS extensively. There have been only older case-oriented reports of disease transmission via manikins to (other) trainees during training, such as Neisseria,16 hepatitis B virus,17 herpes simplex virus,18 with strict guidelines for hygiene in place.19 This despite the millions being trained annually.

- Assess responsiveness: The candidate is instructed to approach carefully and to verbally and with gentle shaking check to see if the patient is responsive. Until recently, the caregiver was instructed to pinch in the M. Trapezius on one or both sides.9
If there is no reaction, this is enough to call for help. The caregiver is taught to turn the patient onto their back if the patient is unresponsive and in such a position which disallows or hinders access to airway and circulation. The caregiver remains with the patient during this phase: the “call” should be seen as a literal exclamation (as opposed to going for a phone). Emphasis is placed, however, on the importance of early entry into the emergency medical system (EMS). The general availability of cell phones has resolved this issue which caused concern in many lay rescuers.

The EMS in the Netherlands allows for a “free-call” activation. This means that any layperson may call for and have sent out a police, fire or EMS response without validation by a professional. This means that after anyone determines that a respiratory arrest exists, or, as in others systems, as early as the lack of reaction to the verbal commands or the shaking, the activation can occur. Current practice has experienced (retired) EMS personnel monitoring the calls, while in other countries questionnaire-based queries determines the type or urgency of the response. The high incidence of agonal breathing has, as mentioned above, caused concerns about misdiagnosis in the algorithms in use at EMS dispatch.20

- Following the call for help, the caregiver assesses the airway. This remains difficult as agonal breathing makes this observation ambiguous for both laypeople as professional caregivers.21 The classical approach using the head-tilt/neck-lift method (Figure 2) to open the airway has been supplanted by the head-tilt/chin-lift or the jaw thrust technique. The latter has been indicted for causing cervical spine movement and as being difficult.22,23 The intention is to clear the upper airway of obstructions due to unconsciousness (i.e., move the tongue base out of the oropharynx with opening of the epiglottis).24 The 2000 Standards and Guidelines recognized that manual finger sweeps of the mouth could be counterproductive and discouraged their use.25,9
The assessment of the circulation is no longer advised for lay persons, and with care for professionals, since the Guidelines 2000 suggested that the accuracy of 65% left too large a margin for serious error.

Figure 2: Freeing the airway.

- Assess airway and breathing. The frequency and amplitude of regular breathing patterns is demonstrated as well as recognition and treatment of foreign body airway obstruction (FBAO) as well as options in clearing and maintaining the airway, including the rescue position. After the airway has been cleared, the caregiver must judge whether the patient is breathing or not. This breathing can be divided into: (a) absent, (b) agonal or insufficient, and (c) normal. While discussion abounds about limiting the frequency of ventilation and the relevance of ventilation during CPR, the importance of the presence of an adequate airway and breathing is taught. Suggested time for the evaluation of the airway and breathing within 5-10 seconds, motivated by the base respiratory frequency of 8–15 min⁻¹.

- Start chest compressions when the patient is unconscious and not breathing without further checks. The caregiver should (Figure 3):
  - Place their hands on the center of the patient’s chest. As there is no evidence to support the position of the lower two-thirds of the chest in adults, this has changed since the 2000 guidelines.
  - Compress the chest directly down for 4-5 cm in ventral-dorsal direction. This should be done with their shoulder in line with and above the sternum, with elbows as straight as possible and then by allowing their body weight to push down.
  - Perform the compressions at a gross rate of 100 min⁻¹
  - Maintain a duty cycle (compression to relaxation ratio) of 50%. The concept of a duty cycle suggests that the compression/decompression portions of the cycle follow each other continuously.
  - Do this for 30 compressions. Pause briefly to allow two ventilations. The caregiver will have to move his hands to clear the airway by head-tilt/chin-lift technique, close the nose, seal their mouth over that of the victim, and exhale into the victim’s mouth, simulating positive pressure ventilation. The ventilation period should not exceed 5 seconds, with one-second ventilations of 400-500 ml per ventilation. As soon as this is done the caregiver must (rapidly) return his hands to the sternum and continue with compressions.

Figure 4: Positioning of the focal point of compression classically involved using two fingers to

*Note that the guidelines suggest that the size of victim may require adjustment of depth.*
This series of actions is carried out until help arrives, the caregiver is incapable of performing the activities, or the victim is declared dead. Each minute the caregiver should stop to check for responsiveness and breathing in the patient, but allow only a few seconds for this.

A number of aspects of one-rescuer BLS have changed with the 2005 guidelines. The American Heart Association (AHA), as well as in-hospital teams have retained the rescue breaths before the initiation of chest compressions. Attempt five with a goal of at least two adequate breaths of 6-7 ml kg\(^{-1}\) if no oxygen supplement is used.\(^8\) Each breath should take circa 2 seconds, allowing low airway pressures. Rescue breaths are performed immediately following each other.

The hand position, described by Kouwenhoven\(^{31}\) (Figure 4) puts the hand “just cephalad of the xyphoid” and has remained unchanged from his original suggestion up to the 2005 guidelines when it was dropped. This skill was physically trained by using two finger-widths to space off the xyphoid process and could be time consuming. The hand position has now changed to ‘the middle of the chest’, simplifying and quickening placement.

\(^8\) Note that 400-600 ml is advocated by the European Resuscitation Council (ERC). The 6-7 ml kg\(^{-1}\) is an ERC directive, the 10 ml kg\(^{-1}\) is an ILCOR standard.
measure off the xyphoid process. The guidelines 2005 simplified this to “a hand position in the middle of the chest”.

This has been simplified to just ‘in the center of the chest.’ One manuscript has described the importance of having the dominant hand under the non-dominant hand and this has been added to the guidelines. The 1.5–2 inches has remained constant after initial adaptation from Kouwenhoven’s 3-4 cm. The caregiver is instructed to press straight down, with interlocking hands and straight elbows on the sternum. Current guidelines continue to advocate a 50% compression to relaxation ratio.

With the 2000 Standards and Guidelines the breathing interval had implicitly been lengthened as emphasis was placed on low ventilations flows and pressures geared towards limiting the risk of gastric insufflation. More recently, this interval has become the focus of discussions due to the excessive length of time required, with one study showing times of up to 16 seconds, as opposed to the theoretical 5 seconds needed. Berg et al. demonstrated that coronary perfusion pressure falls during breathing intervals, and associated this with negative outcome in a swine model. Noordergraaf et al. in a computer model went on to demonstrate that ventilation, with their impact on coronary perfusion pressure not only interfered with flow, but also caused flow to turn around due to ventilatory impact on right atrial pressure.

Cardiopulmonary resuscitation has emphasized the pulmonary and circulatory aspects of the arrest. This has been expressed in the rhythm of alternating compressions and ventilations during basic life support. Kouwenhoven et al. initially suggested 5 compressions followed by a ventilation, but added a two-rescuer option of 15:2 by 1965. This has remained unchanged until challenged on the basis of disinclination of caregivers to perform mouth-to-mouth and suggestions of extensive ventilation to perfusion mismatch, supported by animal experiments, in the later part of the 1990s. This ratio will change, in part, based on a computer model, to 30 compressions to 2 ventilations in 2005.

The teachability of CPR

When BLS became “commonly” available, respiratory and compression aspects were defined, primarily to make teaching these skills easier for both the trainee and the instructor. Standardized courses were created as early as 1965 to make the skills and knowledge needed to apply the skills available. Manikins were added quickly as the training needs grew and became the basis for skills courses were created as early as 1965 to make the skills and knowledge needed to apply the skills available. Manikins were added quickly as the training needs grew and became the basis for skills training.

Teaching the activities will be done, in our case, using a Resusci Anne manikin. Laerdal Inc. (Stavanger N) supplies a wide range of manikins world wide, and has done so for decennia.

While teachability was initially not considered at issue, it has become one of the points of critique in the development of recent guidelines. This recognizes that while the application of BLS has been shown to have a positive effect on survival, and, perhaps surprisingly, that some BLS may be better than no CPR at all, much of the basic life support being performed was far suboptimal. Methodological concerns in training have long since been raised and reiterated over time, where a number of candidates active during a training (i.e., the number of persons trained in relationship to the number of manikins and instructors/teaching aids, using a short period of time for the training), as well as the quality of teaching have implicated a rapid loss of skills and to a lesser degree the lack of knowledge retention.

During the development of training, the so called psychomotor skills were developed. In any number of papers these aspects have been designated as an “all or nothing” series. This implies that if one’s skill is ineffective, the relative chance of a successful outcome is reduced to zero. This is not universally supported, and perhaps contradicted by the suggestions that ‘some’ BLS seems to improve outcome. The skills described above, also judged as “able to be scored” during training, allowing the quality of the caregiver to be “measured” independently of the personal emphasis of the instructor.
The placement of the hands on the thoracic wall;
Compression rate; (*)
Compression depth; (*)
Compression to relaxation ratio (C/R ratio or duty cycle)(*)
Breathing interval (i.e., the no compression time, hands-off time, no-flow time);
Ventilation volume;
The rhythm (i.e., the ratio of compressions to breaths).

Each skill, which all could be read directly from the written reports generated by a Laerdal manikin, were given points (Figure 6) to allow for recognition of minor as well as major errors, even though there is little evidence of the relativity or cumulative nature implied.

Teaching CPR, however, turned out to be more difficult than expected, with retention of skills and knowledge being brief, independent of the group to which it was being taught and the medium used. This is, in part due to the fact that the skills are discontinuous, discrete processes which, are learned in a short, intense period, and thereafter not considered or used until they are, unexpectedly, needed. Similarly, invasive airway skills and, for example, amateur diving skills suffer from the same limitations. Riding a bike, however, does not; after many years of inactivity, most people will be able to climb on an unknown bicycle and ride away. Interestingly, Devices designed to replace the ‘human’ activity in chest compressions have, to date, been unable to demonstrate improved good outcome hospital discharge: because of the quality of what the “two hands” are doing, because of design limitations since the device must do what humans are supposed to do, or because of an other, potentially fundamental, flaw in resuscitation thinking, is unknown.

In Chapter 3 research into the items marked with an (*), compression rate, depth and ventilation volumes will be presented as they have the largest impact in training, experimentation and in clinical use. The duty cycle will be addressed here briefly, as it pertains to evaluation of mechanical versus teachable manual techniques and is an example of research progression.

The duty cycle, or the compression to relaxation ratio (C/R ratio) refers to the period during which the sternum in being lowered or held down in relationship to the period during which there is no (external) pressure on the thorax and it is either rebounding (passively) or is at rest in the neutral position. These periods may be termed CPR systole and CPR diastole, respectively. Kouwenhoven et al. described the duty cycle as a discontinuous curve, compressing, holding and quickly rising again. Their suggestion was that the brief period of continued pressure would result in increased blood movement. Current teaching is based on the physical limitation of manual compression.

2.3. Data and evidence in resuscitation and neurological outcomes

The end point of all efforts made in CPR is restoration of life (i.e., leave hospital to return to an active, independent, productive life); however, many authors have suggested that to measure cardiopulmonary resuscitation sub-measures in terms of process steps should be made since the ‘real’ outcome was seldom seen.

A clear relation has been shown between “down time” and potential for outcome. Useful definitions in (BLS) CPR are the concept of “down time” which is a period (totally) free of treatment, non-compression time (NCT) and other concepts. The basis for the discussion of survival and its quality can be traced back to the initial reports by Kouwenhoven et al. where more than 70% definitive and good survival was reported. However, in his follow up, Jude et al. differentiated the survivors by cause and location of the collapse and showed that perhaps 30% of their acute myocardial infarction patients survived initially. This subset in their population, however, consisted of in-hospital cases.

A broad range of outcomes after out-of-hospital resuscitations has been documented. The range lies from 0–87% depending on any number of variables, with the ERC Guidelines 2005 suggesting that 5–
10% of all out-of-hospital arrests survive to hospital discharge, with this approaching 15% if the initial rhythm is ventricular fibrillation. Mullie et al. retrospectively described 4548 patients with 1-25% long-term survival (LTS) after cardiac arrest; if the arrest was at home a lower end outcome of 5% was found. The best results were achieved in arrests of non-cardiac origin, such as sepsis. For in-hospital resuscitation 17% is expected. However, even without basic life support, 5-50% survival has been described. Guzy et al. described a 10% overall survival at discharge, and contrasted this with 6% initial survival in a population of 368 cases if bystander CPR was not performed. They suggested that the only important variable in outcome was the five “intervention free” minutes required for the paramedic response.

Surprisingly, outcome has remained constant for the last 40 or more years, reflecting the lack of gain expected from increased training, primary or clinical research and technical improvements. Also interesting is the fact that closed-chest CPR has remained acceptable, applied preferably over open-chest CPR, even though little or no hard evidence has been generated to support the former’s (protracted) use. This may, in part, be attributed to the confusing manner in which data has been collected.

2.3.1 Data collection and registration
To improve on the quality of data, as well as to allow for improved comparison between data, Utstein templates for reporting out-of-hospital, laboratory research, and in-hospital resuscitation were developed. An update and simplification has recently been presented, as well as similar systems for more general use. These structured, descriptive systems were developed to offer a degree of standardization in reports. Much has been said about CPR and survival after it. These definitions, part of the Utstein system, are important when comparing quality and similarity between caseloads. Table 2 describes accepted points of evaluation.

<table>
<thead>
<tr>
<th>Table 2: Utstein definitions for outcome.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Description</strong></td>
</tr>
<tr>
<td>ROSC</td>
</tr>
<tr>
<td>Initial or survived event</td>
</tr>
<tr>
<td>24 hours</td>
</tr>
<tr>
<td>72 hours</td>
</tr>
<tr>
<td>Discharge from hospital</td>
</tr>
<tr>
<td>6 months after arrest</td>
</tr>
</tbody>
</table>

The Utstein reporting is complemented by different scoring systems used to evaluate neurologic outcome after resuscitation for clinical use. Most common is the CPC score, while others are geared towards assessment of potential outcomes in order to support decisions to continue treatment. These scoring systems range from the use of the Glasgow Coma Scale to the system of neurologic deficit...
developed by Safar, including a 0-500 detailed system, also suitable for animals. Currently the scoring via the CPC (Cerebral Performance Scale, 0-5) has preference (Tables 3, scoring systems).

**Table 3a: Glasgow coma scale.**

<table>
<thead>
<tr>
<th>Glasgow Coma Scale</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
</tr>
<tr>
<td>2</td>
</tr>
<tr>
<td>3</td>
</tr>
<tr>
<td>4</td>
</tr>
<tr>
<td>5</td>
</tr>
<tr>
<td>6</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Eyes</th>
<th>Does not open eyes</th>
<th>Opens eyes in response to painful stimuli</th>
<th>Opens eyes in response to voice</th>
<th>Opens eyes spontaneously</th>
<th>N/A</th>
<th>N/A</th>
</tr>
</thead>
<tbody>
<tr>
<td>Verbal</td>
<td>Makes no sounds</td>
<td>Incomprehensible sounds</td>
<td>Utters inappropriate words</td>
<td>Confused, disoriented</td>
<td>Oriented, converses normally</td>
<td>N/A</td>
</tr>
<tr>
<td>Motor</td>
<td>Makes no movements</td>
<td>Extension to painful stimuli</td>
<td>Abnormal flexion to painful stimuli</td>
<td>Flexion / Withdrawal to painful stimuli</td>
<td>Localizes painful stimuli</td>
<td>Obey commands</td>
</tr>
</tbody>
</table>

This general score has a moderate inter-observer variability, but is very common and often incorporated in other scoring systems. A GCS > 13 at discharge has been suggested as a surrogate for good neurological (CPC-1 to 2).

**Table 3b: CPC: cerebral performance categories scale.** Also known as the Glasgow-Pittsburgh CPC

| CPC 1 | Good cerebral performance: conscious, alert, able to work, might have mild neurologic or psychologic deficit. |
| CPC 2 | Moderate cerebral disability: conscious, sufficient cerebral function for independent activities of daily life. Able to work in sheltered environment. |
| CPC 3 | Severe cerebral disability: conscious, dependent on others for daily support because of impaired brain function. Ranges from ambulatory state to severe dementia or paralysis. |
| CPC 4 | Coma or vegetative state; any degree of coma without the presence of all brain death criteria. Unawareness, even if appears awake (vegetative state) without interaction with environment; may have spontaneous eye opening and sleep/awake cycles. Cerebral unresponsiveness. |
| CPC 5 | Brain death: apnea, areflexia, EEG silence, etc. |

**Table 3c: EPS** Early prediction score: Developed in Leuven (B). A five factor based system

<table>
<thead>
<tr>
<th>Add one point if</th>
<th>Scorable items:</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Type of cardiac arrest is VF</td>
</tr>
<tr>
<td>1</td>
<td>Type of respiration at initiation of CPR is gasping</td>
</tr>
<tr>
<td>1</td>
<td>Pupil reaction: unequal, present (slow or normal)</td>
</tr>
<tr>
<td>1</td>
<td>Swallowing is present</td>
</tr>
<tr>
<td>1</td>
<td>The cardiac arrest was witnessed</td>
</tr>
</tbody>
</table>

EPS during resuscitation results in a comparable amount of information, whether used to predict success, alive and conscious 14 days post-CPR or no-success. EPS early (10 min) after initially successful resuscitation is more effective in predicting no-success than success. EPS during CPR does not allow decision making as far as stopping or continuing CPR efforts. EPS early after CPR does
Table 4: Levels of evidence (as applies to cardiopulmonary resuscitation)\textsuperscript{69}

<table>
<thead>
<tr>
<th>Evidence level</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Randomized, controlled, clinical studies, or meta-analysis of multiple clinical trials with substantial treatment effects</td>
</tr>
<tr>
<td>2</td>
<td>Randomized clinical trials with smaller or less significant treatment effects</td>
</tr>
<tr>
<td>3</td>
<td>Prospective, controlled, non-randomized cohort studies</td>
</tr>
<tr>
<td>4</td>
<td>Historic, non-randomized cohort or case-controlled studies</td>
</tr>
<tr>
<td>5</td>
<td>Case studies</td>
</tr>
<tr>
<td>6</td>
<td>Animal studies levels A (better) or B (moderate), mechanical models</td>
</tr>
<tr>
<td>7</td>
<td>Extrapolation, theoretical modeling or analysis</td>
</tr>
<tr>
<td>8</td>
<td>Rational conjecture, common sense</td>
</tr>
</tbody>
</table>

Table 5: Class of recommendation (as applies to cardiopulmonary resuscitation) Adapted from the S&G 2000, p1-5.

<table>
<thead>
<tr>
<th>Recommendation class</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Strong evidence of desired effect. Standard of care</td>
</tr>
<tr>
<td>2a</td>
<td>Interventions are acceptable, safe and useful. Considered standard of care, intervention of choice by majority of experts</td>
</tr>
<tr>
<td>2b</td>
<td>Interventions are acceptable, safe and useful. Considered within the ‘standard of care’, but as a reasonable option for prudent physicians,</td>
</tr>
<tr>
<td>Indeterminate</td>
<td>Evidence suggesting strong potential for harm. Intervention not suitable for general use</td>
</tr>
</tbody>
</table>

2.3.2 Levels of evidence, class of recommendation
Levels of evidence have been introduced in cardiopulmonary resuscitation research during the mid 1990s and extensively used in the standards and guidelines since then.\textsuperscript{9} The level of evidence criteria are defined as listed in Table 4. The purpose of defining levels of evidence is perhaps indicative of the need for careful control of expert opinion. The class recommendations are summarized in Table 5. However, as the absence of evidence is not entirely the same as the evidence of absence (of effect), this system caused many accepted practices to become questioned as no studies, were available to support them. In Chapter 3 this will be commented on further.

2.4 Moving blood in CPR: introductory aspects and theory

Maintenance of a normal internal milieu (i.e., life) requires a large number of physiological pillars. The focus here remains on the circulatory aspects. Sudden failure of cardiac function (i.e., asystole of fibrillation) means that oxygen and metabolic transport are reduced to inadequate levels, which after only a short time interval forces the muscular part of the respiratory system to fail. This will also occur if respiration fails. Assuming for a moment that circulation can be promptly restored to normal function, such action does not guarantee long-term survival: if the heart suffered serious damage from the original insult, restoration of blood flow may not occur or this failure is likely to repeat itself, leading, finally, to death. Clinically, this poses a great challenge: which “hearts are too good to die”?\textsuperscript{70}
Prompt restoration of the circulation is not possible under many conditions under which arrest may occur. The only workable approach appears to be an attempt to sustain a circulation, even at a subnormal level, until professional help allows invasive or diagnostic choices to be made.

In the paragraph on teaching CPR, the mechanism by which the circulation is sustained is implied. By pressing the sternum downward, the heart is compressed and pumps blood into the pulmonary and systemic circulations. This, most likely, is a strong simplification of reality and also the central issue in this manuscript. To promote a basic understanding, mechanistic descriptions of important theories and principles are presented below.

Initially, resuscitation, being limited to in-hospital and more specifically the operating suite setting, was performed as a surgical procedure. Under open-chest cardiac resuscitation (OCCR), the mechanism by which the circulation was supported was clear. Compression of the ventricles by the hand enabled replacement of the ventricular pump to a large degree. The heart is supported by a hand. Compression was directed at the ventricles, and the hand kept below the annulus fibrosus, to avoid impact on the competence of the cardiac valves. This concept has not been contested, and remains accepted today. It remains unchanged today, with the incision being made in the fourth intercostal space, requiring only seconds. The caregiver is instructed to avoid pressure on the coronary arteries. Even Stephenson noted that failure was often due to lack of practice in an animal lab or on a suitable model, which he stated as actually being available and in use in 1953.

However, opening the chest was deemed unsuitable for general lay or out-of-hospital cardiac arrest. Kouwenhoven et al. in 1960, suggested that resuscitation could be done equally effectively without opening the chest. Over time, two main theories on how this actually worked were developed. The central goal of both is to describe the mechanism which moved blood around the two parts of the closed circulatory loop and thereby allowing a window of opportunity to improve on the technique.

When Kouwenhoven introduced closed-chest cardiac resuscitation (CCCR) in 1960, his initial description of the mechanism was, in large part, analogous to the mechanism in OCCR. (See Figure 2 in chapter 9.3) He suggested that the ventricle(s) were compressed between the sternum and the vertebral column during CPR systole. This caused the ventricular pressure to be higher than that in the atria, closing the atrioventricular valves and allowing atrial refilling during CPR systole. With a venous-to-atrial pressure gradient forward flow around the cardiovascular system was guaranteed. The implicit assumption is that the ventricles contain sufficient blood to generate an adequate stroke volume, so that the ventricular valves will open and close normally. He described this as a physiological process with a potential for 40-60% of normal cardiac output and a potential survival time of up to an hour.

The core of the competing theory is, clinically, attributed to Criley et al. This theory held that the heart becomes a conduit during CCCR. The principle revolved on the movement of blood through the left heart, from the pulmonary circulation during CPR systole requiring that left-sided mitral and aortic valves remained open. The pulmonary capillary bed became a functional valve and the right heart valves (both) close. Blood moves from the high pressure intrathoracic vascular to the lower pressure extrathoracic structures. This theory refocused interest on the role valves fulfilled in CPR.

An essential aspect in this theory was the demonstration of Niemann’s valve, a functional valve at the thoracic outlets. This valve which protects the valveless cerebral circulation from the backflow pressure, allows the extrathoracic arteriovenous pressure gradient to generate forward flow. This theory also assumes an adequate supply of blood, but defines the reservoir as blood being in the thoracic cavity as a whole, with emphasis on the thoracic veins, the pulmonary vasculature and the right heart. The same chest compression is now presumed to compress all vascular structures in the cavity, including the heart, forcing forward blood movement as the pressure gradient ensues.
Both theories sprouted several variations. An innovative concept allows generalized integration when viewing the issue of an external pressure on the visceral contents of the chest. Impedance defined flow offers a generalization of Harvey’s teaching and can be applied to the CPR pump theory. Harvey held that the heart, as pump, alone is responsible for steady flow around a closed circuit as well as the pulsations that occur in it. Steady flow can, however, also be generated around a closed circuit that is free of valves providing certain conditions are satisfied. Such steady flow may also be accompanied by pulsations.

The mechanism is visualized as period alternation between disturbing the (dynamic) equilibrium in a compliant system and allowing the system to regain a (new) equilibrium. (Figure 5) A state of equilibrium may be disturbed in several ways, such as local compression and relaxation, acceleration or deceleration of the system, or modification of the gravity vector. The disturbance selected requires expenditure of energy on the system and results in alteration in the distribution of blood, hence in an equilibrium state, involving another redistribution of blood and the generation of flow. In an asymmetric circuit like the cardiovascular system, the two generated flows will generally not compensate one another. In particular, when the design is such that the disturbance runs its course faster than the return of equilibrium, the flows, selecting the paths of least impedance, may occur in the same direction around the circuit, rather than in opposite directions. A pertinent consideration here is that, since the frequency content of disturbance and recuperation is different, while most impedances are frequency dependent, impedances may have different values during the two phases, resulting in effects not expected at first sight, like the two flows occurring in the same direction.

Examples, similar to CPR, may be worked out for linear and nonlinear forms. These include pumping by the ventricles, where the valves play the role of time-varying impedances, small when open, large when closed. Neither the presence of a discontinuity, nor of nonlinearity is a general requirement for the production of impedance defined flow. The mechanism involved is compression of large or small veins by augmentation of pressure external to that vein. The increase in ambient pressure raises the internal venous pressure regionally or locally to a higher level, which will move more blood out of the compressed section. The outflow will follow a direction and a distribution defined by the impedances as ‘seen’ by the compressed section: more flow in the direction of the lower impedance and lower pressure. When the compression is passed, the veins will refill, again from the direction of the lower impedance, but under the new conditions.

Other theories, both attempting to incorporate the different mechanisms into one theory or to rule a theory out, have been developed. These will be presented in Chapter 3 as part of the development of thinking in CPR. Here it is sufficient to note that these two major lines of thinking both involved a
closed-chest to which an external pressure was applied. Low survival rates after resuscitation, despite these mechanistic theories, provided a powerful stimulus for research. Answers have been sought, and continue to be sought in several directions. For example, which, if either, of these pump theories is valid at which moment in time, and which actions may allow optimal affects on the pump theory active at that point in time.

Receiving the core aspects, it would seem straightforward to make a basic choice by measuring in patients which (pair of) cardiac valves function approximately normally. This has been attempted, but failed to provide convincing, reproducible, answers. Although experiments in humans have the allure of studying reality, the object is difficult to define adequately. Anesthesia may also have introduced unknown changes.

Such considerations led to a significant number of life science investigators to execute animal experiments as living models of actual patients (Chapter 6). This decision has attractive features in that more quantities can be measured in the animal, though the new object exhibits anatomical and physiological characteristics at variance with the human object. Inanimate models can avoid two classes of difficulties in patient measurements and animal experiments. These are that all properties of the inanimate model are known or can be determined and there is no issue about unpredictability as time progresses as may occur in living models. However, derivation of classical laws in physics (i.e., Ohm’s law, Maxwell’s law, Navier-Stokes equation) was possibly based on physical models. Such successes made it appear to natural scientists to carry the methodology over to the life sciences and confront more intricate issues such as CPR. This decision immediately exposed barriers to the execution of such plans; deficiency of knowledge of natural scientists in the life science areas, and where life scientists invaded the natural sciences similar problems were encountered.

2.5 Measurements and monitoring: Physiology in CPR

There is a wealth of investigations into the conduct of the circulation in a plethora of conditions. In this light it has been judged as acceptable that the circulation, even under the extreme conditions imposed by circulatory collapse, may be approached as a physiologic whole. In the following paragraph, aspects involved in measurements and monitoring of the circulation during CPR will be explored as a basis for understanding the gradual of history. Its principal task is to maintain an internal environment within a close range of pH and to effectively transport oxygen, nutrients and waste products to allow the peripheral cell to maintain its integrity.

2.5.1 Units in physiology and CPR

There are many different systems of units involved in the monitoring and description of the circulation, the airway and the performance of CPR. Confusingly these systems are sometimes used in an intermingled form. Newtons Law (Force equals mass times acceleration) lies at the basis of this unit system.

Pressure equivalents which may be found are: mm Hg = 1.36 cm H2O; 1 kPa = 7.5 mm Hg; 1 atmosphere (ATM) = 760 mm Hg. The mm Hg as a unit is not incorporated in any system of pressure. There is a range of uses for Newtons as a unit of force. This unit has replaced dynes. If an area is associated, the correct nomenclature is the pascal N m⁻². In an older system kilograms were used as the unit of force, and this is still done in CPR research. (Chapters 4, 12)

The relationship between pressure and flows, as opposed to pressure and force, is defined by the Navier-Stokes equation, including partial derivatives. Pressure differences across a system are related to the flow through that system, taking into account any obstruction or resistances to flow (i.e., stenosis). In CPR, an example might be coronary perfusion pressure (CPP). This is the pressure in the aortic root minus that in the right atrium at diastole, and is suggested to represent coronary blood flow. In cardiac arrest, some form of impedance is a factor. As a pressure difference drives flow, much of CPR research incorporates arithmetic differences between values. In example:
\[ Q = \Delta P / R \]  

(eq. 2.1)

Where \( Q \) is the flow, \( \Delta P \) is the difference in pressure between two points and \( R \) is the resistance between them. This formula, however, may be of little help owing to the clinical difficulty in measuring \( R \).\(^{10} \) Also, delayed compliance, or stress relaxation, defines the ability of a vessel to adapt to abrupt increases of stress by increasing size (over time) by elastic distention.\(^{10} \) Determinants in the resistance to flow are the viscosity of the fluid \( \eta \) (i.e., blood), the length of the blood vessel involved (\( L \)), the radius of the vessel (\( r \)) and a constant (\( 8/\pi \)) such that:

\[ R = (\eta Lr^4) (8/\pi) \]  

(eq. 2.2)

Under physiological conditions viscosity is not subject to rapid change, while this has been described under marathon runners, as well as after lengthy resuscitation.

The normal cardiac cycle incorporates diastole and systole. Diastole is defined as the period during which there is no ventricular contraction, and during CPR is defined as the period starting with the release of pressure to the initiation of new (external) pressure. Systole is defined as beginning with the ventricular contraction (isovolumetric with the closure of the mitral valve and continuing until closure of the aortic valve). Venous return to the right heart is a complex process involving ventricular compliance, heart rate, skeletal muscle pump function, atrial contraction, cardiac contractility and autonomic tone.\(^{81} \)

A typical electrical cardiac cycle, at a frequency of 70 beats min\(^{-1}\) involves a systolic period of circa 250 msec and a diastolic period of circa 550 msec. During this period 300 msec is passive ventricular filling supplemented by 110 msec of atrial contraction. During rest some 80% of ventricular filling is achieved before atrial contraction. During sympathetic and hormonal stimulation the cardiac cycle becomes shorter as rate goes up. A large part of the decreased time available impacts on ventricular diastole, affecting filling time, and placing a greater dependence on atrial contraction to ensure adequate ventricular filling. Ventricular contraction may shorten by 20% as a function of increasing force, with both rate of contraction and relaxation being equally involved.

The arterioles, under baroreceptor and neural control, influence the vascular resistances. During CPR, however, the sympathetic control rapid decreases, causing direct reduction of arteriolar regulation despite high vasopressor concentrations. This has the effect of creating diastolic values to fall more than the systolic values. Mean blood pressure defined as:

\[ \frac{\{P_{\text{systolic}} + (2 \times P_{\text{diastolic}})\} / 3}{3} \]  

(eq. 2.3)

suffers from this fall, creating low perfusion pressures and tissue hypoxia.

2.5.2 Distribution of blood in the circuit

The circulation, when discussing CPR, is compared to a closed system with two pumps in series. The lymphatic circulation is generally ignored as is third space volume shifts. The circulatory volume can be divided\(^{10} \):

- 39% large veins and venous reservoirs, with an additional 25% in the other parts of the venous system.
- 9% of the volume may be found in the pulmonary vessels.
- 8% in the large (central) arterial system.
- 7% in the heart itself.
- 7% in small arteries and arterioles.
- 5% in the capillaries.

During normal hemodynamic states the peripheral veins may serve not only as a conduit, but also as a blood reservoir, containing as much as 75% of the circulating volume. Autonomic reflexes may alter
R = \left( \frac{\text{blood vessel involved (L), the radius of the vessel (r) and a constant (8/} \pi}{\text{to abrupt increases of stress by increasing size (over time) by elastic distention.}} \right) \frac{\eta}{L/r^4} \left( \frac{8}{\pi} \right) \text{ (eq. 2.2)}

\text{Determinants in the resistance to flow are the viscosity of the fluid measuring R. Also, delayed compliance, or stress relaxation, defines the ability of a vessel to adapt and with the size of the liver. After a forced expiration the right cupola is on a level in front with the fourth costal cartilage; at the side with the fifth, sixth, and seventh ribs, and behind with the eighth rib; the left cupola is a little lower than the right. Halls Dally states that the absolute range of movement between deep inspiration and deep expiration averages in the male and female 30 mm. on the right side and 28 mm. on the left; in quiet respiration the average movement is 12.5 mm. on the right side and 12 mm. on the left. It appears that the position of the diaphragm in the thorax depends upon three main factors, (a) the elastic retraction of the lung tissue, tending to pull it upward; (b) the pressure exerted on its under surface by the viscera; this naturally tends to be a negative pressure, or downward suction, when the patient sits or stands, and positive, or an upward pressure, when he lies; (c) the intra-abdominal tension due to the abdominal muscles.}

The heart is suspended by the great vessels and held by the pericardial fold. It lies as a cone form, from cranially (right) at the level of the third costal, to caudal (left) at the level of the sixth intercostal space for a distance of 15-16 cm. The inferior and superior venae cavae and the free wall of the right atrium can be projected just to the right of the sternum in the ventral-dorsal axis. Dorsal of the sternal corpus, the right atrium and right ventricle with the right and left outflow tracts may be found. The free wall of the left atrium and most of the left ventricle lay to the left of the sternum. The apex cordis lays is the sixth intercostal space in the midclavicular line (during expiration) and this may move caudally to the medial margin of the eighth rib as well as medially during deep inspiration.

Dorsal of the heart, the aorta and the esophagus cross over the vertebral column from the right to their definitive position next to or lateral of the vertebral column.

To apply open-chest cardiac resuscitation (OCCR) little anatomical knowledge is required. A longitudinal intracostal incision is made from the lateral side of the lateral clavicular line to the posterior axillary line in the fourth intercostal space (i.e., one rib below the nipple). This allows the pericardium to be opened, if this was desired, and both ventricles to be enclosed (below the fibrotic annulus) and rhythmically compressed. Typically, the heart lies “in” the hand. It was also possible to compress the heart against the anterior chest wall, or even the vertebral column. Lung tissue is only encountered indirectly, allowing visual inspection of respiration but without losing pulmonary volume for gaseous exchange.

For closed-chest cardiac resuscitation (CCCR) the lower part of the sternal corpus is moved dorsally, with the intention of compressing the heart. Functionally, however, this would most likely compress the right atrium, the left outflow tract, the ascending aorta, and the right ventricle. The fibrotic annulus is also compressed, potentially altering its shape and, also potentially, the competence of the atrioventricular valves.

2.5.4 The (patho-)physiology of the airways & pressure in the thorax

Under spontaneous ventilation, the chest cavity is enlarged drawing air in via the upper airways by moving the chest wall out (to a maximum of about 120%) during maximum inspiration, with the diaphragm moving downward (caudally) in the abdomen.

Ventilation and oxygenation may be separated for logistic reasons; the purpose of oxygenation being to maintain aerobic metabolism, and ventilation to maintain CO₂ removal in order to maintain pH. Ventilation volumes are typically maintained at 7-8 ml kg⁻¹, at ventilation rates of 10-14 min⁻¹.
Anatomical dead space has been estimated at 1-2 ml kg\(^{-1}\), and increases with the human in a supine position. Functional residual capacity has been shown to decrease by some 30% in a healthy population when the patient moves from a horizontal to a supine position. Depending on metabolism, CO\(_2\) concentration in blood will increase with 3-5 mm Hg min\(^{-1}\) with a proportional shift in pH of 0.1 point per 5 mm Hg CO\(_2\).

Important pressures and pressure relationships, useful in CPR are:

- \(P_{TA} = P_{TAW} - P_{A}\) (transairway pressure = pressure difference between the opening of the airway (mouth and the alveolus), representing the pressure gradient moving air and defines airway flow resistance.
- \(P_{a} = P_{TR} = P_{A} - P_{AS}\) (transthoracic pressure = pressure difference between the pressure in the alveoli as compared to that outside the body surface). Represents the pressure needed to fill the lungs and simultaneously move the chest wall out.
- \(P_{I} = P_{TR} = P_{A} - P_{b}\) (transpulmonary pressure = the pressure difference between the inside alveoli of the lung and the pleural space). It may be noted as the alveolar distending pressure and is responsible for maintaining alveolar insufflation.
- \(P_{ex} = P_{TWO} - P_{ex} = (P_{A} - P_{b}) + (P_{aw} - P_{A})\) The transrespiratory pressure is the pressure difference between the opening of the airway and the body surface, and represents that needed to inflate the lungs during positive pressure ventilation.

During spontaneous ventilation the intrapulmonary pressure (alveolar pressure, \(P_{A}\)) can be described as a sine curve starting a 0 and moving slightly negative to -1 to -5 cmH\(_2\)O\(^{85}\) (inspiration) to neutral when inspiration becomes expiration, to slightly positive during expiration. The \(P_{A}\) may increase to -80 to +100 mm Hg during maximal inspiration and expiration efforts against a closed glottis. The intrapleural pressure (\(P_{pl}\)), under spontaneous respiration, goes from ± –5 cmH\(_2\)O at the beginning of inspiration to ± –10 cmH\(_2\)O at end inspiration, and then decreases to ± –5 cmH\(_2\)O again.\(^{82}\)

Under mechanical ventilation, typically IPPV (intermittent positive pressure ventilation by manual or mechanical means via an endotracheal tube (ETT) or mask), additional measurements can be defined.

- \(P_{PEAK} = P_{IP} = P_{TA} + P_{a}\) It represents the sum of the pressure needed to move the air through the resistance of the airway (i.e., a small ETT), and the pressure of filling the alveolus. It is the highest pressure recorded during a cycle or 0.5 – 1.5 seconds during which diffusion can occur.
- \(P_{PEAK} = P_{TA} = P_{MO} - P_{MID} = P_{TC} = P_{WT} = P_{m} = P_{m} = P_{TA} - P_{PEAK}\) - this equation provides a measurement of the inspiratory and expiratory pressures required.
- \(C = V_{T} / (P_{m} - P_{EEP} = 1/e = \Delta V / \Delta P).\) Compliance or the inverse of elasticity. Compliance is the ease of a structure to change shape defined in ml/cmH\(_2\)O. Absolute values may vary by type of ventilation and gender. In spontaneous ventilation 50–170 ml/cm H\(_2\)O are normal, varying to 40-50 ml/cmH\(_2\)O in males and 35–45 ml/cmH\(_2\)O in females under IPPV conditions. As these measurements are performed under no-flow conditions they are termed static compliance values.
- \(R_{aw} = P_{TA} / \text{flow} = \text{airway resistance to flow} (\text{cm H}_{2}\text{O/L/Sec}).\)

Definitions particularly relevant in cardiopulmonary resuscitation are:

- Coronary perfusion pressure CPP (also known as myocardial perfusion pressure MPP) = mean diastolic Ao (mm Hg) – mean diastolic RA (mm Hg).\(^{84,85}\) These are also related to the myocardial blood flow (MBF),\(^{86}\) which should be more than 20ml/min/100g of tissue to supply nutrients to the fibrillating heart or about one-third of normal. CPP of 18 mm Hg is required to perfuse the epicardium and 28 mm Hg for the endocardium, in normal hearts. Swenson states that a CPP of > 15 mm Hg is routinely associated with survival, and <15 mm Hg almost never.\(^{87}\)
- Cerebral perfusion pressure = mean carotid artery pressure (mm Hg) – mean intracranial pressure (mm Hg).\(^{84}\)
- Cerebral blood flow (BBF): Normally this is 60-70 ml/min/100g with MVO\(_2\) (ml/min/100g) of 8.1 – 9.2.\(^{86}\)

The diastolic pressure (with its principal determinant the compliance) tends to be low (into the single digits) during CPR due to a very low peripheral resistance. Compliance became smaller as (pulse)
pressure become higher in a non-linear fashion. Systolic pressure is a measure of the quality of the pump. Intravascular pressure in a supine human decreases through the circulation as the oscillations decrease. Resistance in the arteries up to the small arterioles is small. By the time the capillaries are reached, the oscillations have decreased to a minimum and the pressure to about 30 mm Hg. At the venous end of the capillaries the pressure is 10 mm Hg. The pressure gradient continues to decrease, reaching approximately zero at the right atrium.

However, these assumptions apply to the normal circulation and normal circulatory tonus. Volume in the lungs may vary from -50% of normal to + 250% depending on PA gradients. During cardiac arrest arterial tonus (i.e., the peripheral resistance) decreases with a parallel fall of diastolic pressures.

During CPR it has been suggested, but until 1990 never formally investigated, if venous return under CCCR conditions is pressure gradient dependent. The peripheral venous to right atrial pressure difference during CCCR diastole is the driving force. Venous return (to the thorax/right heart) has been demonstrated in dogs (19-30 kg, Pento model), to be dependent on (subdiaphragmatic) diastolic and mean inferior vena cava (IVC) pressure, and carotid blood flow, but not on the IVC to RA pressure gradient.

The central aspect of CPR remains that sufficient blood must be moved to supply oxygen to (vital) tissues and to remove sufficient metabolic wastes that the internal environment of the cells remains viable.

2.6 Monitoring techniques

In order to understand the different monitoring systems used in CPR research, the mainstream techniques are briefly reviewed below.

2.6.1 Direct or invasive pressure monitoring

Historically, mercury in a glass tube was used to measure blood pressure oscillations. This system was suitable for slow response frequencies and moderately accurate. It was replaced by fluid-filled transducers in the 1970s, and finally by solid-state systems in the 1990s. The early systems, as well as some in current clinical use, were notably slow, because each system had its own characteristic frequencies which might affect monitoring. Clinicians, looking at the pulse pressures may assume that these are indicative of flow, forgetting that flow is better represented by the area underneath the arterial wave curve, as well as that forward and retrograde flow could be present. The use of the Swan-Ganz catheter has been suggested to allow for pressure and flow descriptions. However, placement of the system requires coordinated flow of blood, and positioning recognizable pressures and curves. Newer automatic systems, no longer requiring fluid boluses, also require adequate and reproducible flows to avoid local overheating, suggested to be an one L min⁻¹. The use of central venous monitoring encounters the same resonance and response time difficulties as arterial monitoring does, but must also cope with the closure of the (large) veins due to the changes in transmural pressure.

2.6.2 Radionuclide injection

Radioisotopes have been used as an attractive method to estimate flows during CPR, but requires specific materials not generally available. The use of C-14 iodoantipyrine demonstrated that radionucleotide injected into the right atrium may be inaccurate due to 'sloshing' as opposed to an injection into the left ventricle, and even then required a competent mitral valve. The use of radio-labeled microspheres has been advocated, but opponents have suggested the repeated use may cause a bias due to obstructions.

2.6.3 Ultrasound and echocardiographic investigations

A consistent limitation to clarification of what is happening in the chest is the inability to directly visualize the internal organs during chest compressions and more in particular to visualize the status
and movement of cardiac valves. The development of ultrasound seemed to create a new window, allowing real time evaluation and potentially allowing the mechanism of CCCR to be resolvable with the introduction of two-dimensional transthoracic echocardiography as well as with transesophageal echocardiography. Work was done by Schluter et al. who, in their careful work describing the possibilities and limitations of the transesophageal approach to echocardiography. As a point of departure, Kouwenhoven suggests (Table 6) that the underlying mechanism involved in CCCR as strongly approaching physiology. Note that, while there are figures describing this in the original manuscripts, these drawings do not demonstrate the atria.

### Table 6: Cardiac pumping as proposed by Kouwenhoven et al.

<table>
<thead>
<tr>
<th>CPR systole</th>
<th>CPR diastole</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitral: closed</td>
<td>Mitral: open</td>
</tr>
<tr>
<td>Ao: widely open</td>
<td>Ao: closed</td>
</tr>
<tr>
<td>Tricuspid: closed</td>
<td>Tricuspid: open</td>
</tr>
<tr>
<td>Pulm: open</td>
<td>Pulm: closed</td>
</tr>
<tr>
<td>$P_{out} &gt; P_{in}$ during CPR systole: AV valves close during compression; Art to venous pressure gradient; RA inflow during CPR systole</td>
<td></td>
</tr>
</tbody>
</table>

Furthermore, the opening or closing during CPR systole of the mitral valve has long been held as the determining factor between the two theories. A closed mitral valve during the compressions (the systole phase) in combination with forward transmitral flow during the release (diastole) phase held to be clear evidence of the cardiac pump theory. An open mitral valve with forward transmitral flow during compression and an unchanging left ventricular volume during the compression phase of chest compressions have been cited as being indicative of the thoracic pump theory.

Presenting the work done in CPR with echocardiography, in chronological order, allows interesting results to be found. The first study, by Rich et al. (Table 7) was done in a convenience sample of in-hospital arrest, with manual CPR initiated early and the echocardiography done within 20 minutes of arrest.

### Table 7: Transthoracic echocardiography finds in (4) humans

<table>
<thead>
<tr>
<th>CPR systole</th>
<th>CPR diastole</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left ventricle size: mean decrease of 2.9% (AP), lateral movement seen. Right ventricle size: max size decrease in outflow tract (up to 73%) from 16% at tricuspid valve. LA and Ao root: up to 32% decrease.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PA cath: antegrade and retrograde flow. Over pulm. antegrade flow only.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Interestingly, the investigators find that the right ventricular outflow tract seems to be obstructed during CPR systole and suggest that the forward flow of blood must then be caused by the pressure gradient between the extra- and intrathoracic systemic (central) veins during the relaxation phase, using Rudikoff et al. as the source of this idea. Rich et al. also points out that arterial peak pressures are not representative for flow, and note most specifically that there was an almost 180 degree phase shift between the peak pressure and flow in their observations.

Werner et al. (Table 8) describes a series of five patients coming to cardiac arrest and manual CPR on the coronary and intensive care units with by oblique echocardiography during manual...
CCCR. Interesting is that four patients had the same investigation under a spontaneous cardiac rhythm during the previous 24hrs, and that Swan-Ganz catheters as well as other invasive monitoring devices were already in situ. The principle goal of this investigation was to evaluate the movement of the (atrio-ventricular) valves.

Table 8: Transthoracic echocardiography findings in (5) humans (Werner et al.101,102)

<table>
<thead>
<tr>
<th>CPR systole</th>
<th>CPR diastole</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tricuspid: * (almost) closed. Pulm: closed, but also closes for IPPV only, compression only as well as compression + IPPV.</td>
<td>Tricuspid: opens. Pulm: open.</td>
<td>PA cath: * ante &amp; retrograde flow. * antegrade flow only. The tricuspid is most inconsistent in movement with ante- and retrograde flow.</td>
</tr>
<tr>
<td>Left ventricle size: unchanged (cautious conclusion).</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Werner concluded in the basis of this data that only the thoracic pump model seems effective. While recognizing this, the investigators also suggest that harder and deeper compression might augment antegrade flow (e.g., the mitral valves open further), and that the advantage of this flow mechanism is that the pulmonary blood volume far exceeds that of the left ventricle. They conclude that volume supplements during CPR might also improve outflow.

Feneley et al.103 (Table 9) working in a dog model, and interested in high impulse CPR, investigated valvular motion and flow in an eloquent dog model with both chronically instrumented and fresh specimens. This group attempted to adjust for prior comments by inducing ventricular fibrillation both with potassium, as well as with alternating current as well as (manual) lateral and sternal compressions in different variations with and without ventilation of compressions.

Table 9: Transthoracic echocardiography findings in (9) mongrel dogs in VF (Feneley et al.103)

<table>
<thead>
<tr>
<th>CPR systole</th>
<th>CPR diastole</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitrail: close rapidly with all forms of compression, except if (a) low velocity compressions or (b) compression not adjacent to heart. Ao: opens rapidly.</td>
<td>Mitrail: open. Ao: closes/approximates rapidly upon release of pressure. Approximation only at low velocity.</td>
<td>Without compression valves stay open. Notes retrograde flow from LA during compressions. No antegrade flow during compressions over mitral valve. Notes LV deformity in direct relationship to (direction of) compression, but no entrapment seen.</td>
</tr>
</tbody>
</table>

Feneley103 finds consistently higher (systolic) pressures in the left ventricle than in the left atrium. The group suggests that the discussion is not whether or not there is a thoracic pump mechanism, but whether this model accurately describes blood flow in their observations. Optimization requires this understanding, they suggest, as cardiac pump would benefit from frequency adaptation and thoracic pump from compression ratio alterations. Remarkling on Rich and Werner, Feneley comments that they were unable to find deterioration of valvular motion after the first five minutes as described by Deshmukh and co-workers.
Deshmukh et al.,104 (Table 10) four years later, aware of both the earlier studies by Rich99 and Werner105 and also aware of the more recent work by Maier105, supported the cardiac pump theory (in the high impulse CPR subgroup), in chronically instrumented dogs, performed a new experiment in 13 mini pigs (20-30 kg) using mechanical CPR (compression depth $\approx 25\%$ AP thoracic diameter) as well as for OCCR.

Table 10: Transthoracic echocardiography findings in (13) mini pigs in VF (Deshmukh et al.104)

<table>
<thead>
<tr>
<th>CPR systole</th>
<th>CPR diastole</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ao: widely open.</td>
<td>Ao: closed.</td>
<td></td>
</tr>
<tr>
<td>Minor tricuspid regurgitation noted.</td>
<td>Tricuspid: opens.</td>
<td>Using contrast study: contrast moves from RA to RV during compression diastole, and to RV-outflow during compression systole.</td>
</tr>
<tr>
<td>Pulm: open.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left ventricle size: decrease of $&gt;25%$ during CPR systole (5 animals).</td>
<td></td>
<td>Posterior cardiac movement during compression with return in decompression.</td>
</tr>
</tbody>
</table>

Deshmukh et al.104 also performed OCCR in these (end-stage) mini pigs while observing the valves transthoracically. He noted that both left sided valves remained competent, if the heart is not luxated anteriorly. This group concludes that valvular competence may decrease over time and support a cardiac pump model. The authors suggest that the differences in results with Werner105 and Rich99 may be due to timing (the delay of at least 20 minutes) in the echocardiogram.

In 1989, Deshmukh et al.98 (Table 11) repeated the main points of their earlier study104 in a larger population (27 pigs, 25-35kg), incorporating successful and non-successful resuscitations with hemodynamic data. In this experimental set, the Thumper was used at 60 cpm, with sufficient force to decrease the chest diameter by 25% and hyperventilation (180 ml/kg). 17 pigs were resuscitated and 5 died, with mean CPPs at 15 and 4 mm Hg, respectively; CPR was performed for 12 minutes.

Table 11: Echo and hemodynamic results in pigs (Deshmukh et al.104)

<table>
<thead>
<tr>
<th>CPR systole</th>
<th>CPR diastole</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitral: closed (competent).</td>
<td>Mitral: open wide.</td>
<td>Note that after 5 min the AV-valves remained open in all animals without ROSC.</td>
</tr>
<tr>
<td>Ao: widely open (20 animals).</td>
<td>Ao: closed (20 animals).</td>
<td></td>
</tr>
<tr>
<td>Tricuspid: closed (competent) with 11 mm Hg gradient shown in all animals with ROSC.</td>
<td>Tricuspid: open wide.</td>
<td>FA cath: n/a.</td>
</tr>
<tr>
<td>Pulm: n/a.</td>
<td>Pulm: n/a.</td>
<td></td>
</tr>
<tr>
<td>Left ventricle size: decrease of $\pm 25%$.</td>
<td></td>
<td>Pressure gradient: $\pm 19$ mm Hg peak syst. over tricuspid valve</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Aorta to RA gradient reported (animals later ROSC), of $&gt;20$ mm Hg(?)</td>
</tr>
</tbody>
</table>

Deshmukh concludes that cardiac pump is the mechanism involved as already confirmed by Feneley et al.103 working in a dog model in 1987. This group also points out the Niemann’s valve prevents regurgitant flow from the superior vena cava, and name both jugular and subclavian vessels in this regard.

In 1990, Hackl et al.106 evaluated the mitral valve function during CCCR in young pigs (19-24 kg) in ketamine-Thumper model, with life support occurring at 60 cpm with a 50% duty cycle, beginning after one minute of ventricular fibrillation, describes 15%, 20% and 25% reduction of the anterior-posterior chest wall diameter with 200, 350 and 500 Newton (N). They found that mitral valve closing force dependant with 16% in 200 N, 68% with 250 N and 95% with 500 N actually closing during CPR systole. As an aside they note that only at 500 N did the CPP gradient reach a mean of 15 mm Hg.
This group addresses the ‘impulse theory’ in CPR: with mass x velocity initial is the energy transferred to the chest and, indirectly, to the heart. They suggest that the thoracic pump theory is demonstrated by their own experiments at the low end of 200 N, and are the first paper to accept that multiple pump mechanisms may be responsible for blood flow during different times, and under different conditions. However, they remain advocates of ‘cardiac pump’ when possible. These conclusions were revalidated in 1990, by Higano et al. using anecdotal data from humans.

Ma, noting that earlier studies generated conflicting results, performed a follow up TEE study in an unique, convenience sample of 20 adult humans presenting with cardiac arrest (without pre-hospital therapy) or developing cardiac arrest in the Emergency Department. They look specifically at the pulmonary flow aspects under CPR conditions in addition to mitral valve movement. They postulated that if the thoracic pump dominated, then pulmonary venous flow (PVF) would occur during CPR systole (i.e., from the pulmonary vein into the left atrium). In normal patients, PVF has been demonstrated to consist of:109,110

- Mono or biphasic forward flow during systole.
- Monophasic forward flow during diastole.
- Small reverse flow during atrial contraction.

During this study Ma et al.108 (Table 12) compared this to those found during CPR performed with manual compressions at 90 cpm, a 50% duty cycle, with epinephrine every five minutes. They divided their findings up into three groups.

Table 12: Transthoracic echocardiography findings in (17 of 20) humans (Ma et al.108)

<table>
<thead>
<tr>
<th>CPR systole</th>
<th>CPR diastole</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitral:</td>
<td>Mitral:</td>
<td>Peak flow: (across mitral valve)</td>
</tr>
<tr>
<td>• Gr-1: closed in 5 of 17</td>
<td>always open 17/17</td>
<td>During release phase</td>
</tr>
<tr>
<td>• Gr-2: opened or widened in 12/17</td>
<td>Ao: n.d.a.</td>
<td>During compression phase</td>
</tr>
<tr>
<td>Ao: widely open with flow 17/17</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PVF:</td>
<td>PVF:</td>
<td>Concludes: All survivors (3/17) in Gr-1.</td>
</tr>
<tr>
<td>• Gr-1: backwards (5/17)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Gr-2a: 8/17 forward</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Gr-2b: 4/17 backwards</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Ma et al.108 also suggests that within the old thoracic (open mitral during compression) and cardiac (closed mitral) theories, a third, time sensitive model should be introduced. This would add left atrial flow as a subgroup in the thoracic pump group, with backward flow over an open mitral valve during compression pointing towards the left atrial pump mechanism. They further suggest that this finding could explain some of the discrepancies between echocardiographic studies, as Rich99 noted left atrial and right ventricular compression. While not powered for survival, none of the patients were discharged and only three achieved ROSC (not indicated which patients these were). The investigators conclude that there is a strong suspicion that there is a time sensitive element in blood flow mechanisms during cardiac arrest.

Klouche et al.111 described a series of investigations in pigs using transesophageal echocardiographic Doppler. They observed that the left atrium remains contractile during the first (seven) minutes of untreated VF, and reported ventricular chamber deformity during precordial compression and relaxation, in combination with mitral valve opening and closing. Finally, Pernat et al.112, working in heavy pigs, reported confirmation of the cardiac pump theory, when using echocardiographic Doppler, as well as that they could demonstrated quantifiable stroke volume.

2.6.4 The use of capnography in CPR

The use of capnography or end-tidal CO\textsubscript{2} (ETCO\textsubscript{2}) has gained general acceptance for monitoring of patients who are being mechanically ventilated. It correlates well with metabolic or ventilation to
diffusion needs if the circulation is not a limiting factor, or significant ventilation to perfusion mismatch exists. Both the numeric value as the waveform itself offer information.

Its introduction as a monitoring device within CPR has been, in part, dependent on the training of physicians involved; anesthesia or critical care physicians have an affinity for these devices. The device has also been limited in its use since a formal airway (i.e., intubation) is required in order to avoid measurement artifacts. Different authors have shown it to be a surrogate for CPP: Sanders, working in a large dog model, and referring to earlier work by Kaleda et al., demonstrated that ETCO\(_2\) correlated well (r =0.78, p = 0.01) with CPP and went as far as to suggest that ETCO\(_2\) might be used as a feedback device. The use of end-tidal carbon dioxide (ETCO\(_2\)) has increased dramatically over the years. While the 2005 Guidelines recommend its use as an early indicator of ROSC, they do not support it as a device to offer real time suggestive feedback to guide chest compressions, but this seems to be a coming reality. Other monitoring devices are summarized in table 13.

**Table 13: Some miscellaneous monitors/comments about monitoring in CPR**

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1984</td>
<td>Maier</td>
<td>Dogs</td>
<td>n.a.</td>
<td>Intrathoracic pressure, an unprotected manometer in the chest or mediastinum and/or esophageal balloons are NOT suitable for monitoring CPR: This due to impact artifacts in the manometers: these are proportional to the kinetic energy (i.e., product of mass and velocity squared). Normally irrelevant, in CPR a major effect.</td>
</tr>
<tr>
<td>1985</td>
<td>Martin</td>
<td>20 dogs</td>
<td>Correlation between CVO(_2) and MVO(_2) during OCCR</td>
<td>Central venous and mixed venous oxygen content may correlate with effectiveness of CPR. Using SG and central venous lines, correlation was sought but not found.</td>
</tr>
<tr>
<td>1985</td>
<td>Martin</td>
<td>24 dogs</td>
<td>Correlation between pH(<em>A) and pH(</em>{C_v}) to steer Na-Bic therapy</td>
<td>pH can be determined from arterial and central venous lines during CPR. They found that a strong correlation exists between pH(<em>A) and pH(</em>{C_v}) (r=9771, p&lt;0.000) with a difference of 5.2 mm Hg before and 15.5 mm Hg during CPR.</td>
</tr>
<tr>
<td>1988</td>
<td>Gudipati</td>
<td>22 Swine (20-35kg)</td>
<td>Evaluate prognostic value of ETCO(_2)</td>
<td>Strong correlation between cardiac output and CO(_2) in both OCCR and CCCR. Strong marker for ROSC.</td>
</tr>
<tr>
<td>1989</td>
<td>Heyworth</td>
<td>15 human patients arriving in A&amp;E</td>
<td>Evaluate the use of C(_O_2) as indicator for CPR quality</td>
<td>C(_O_2) normally is 50-60 mm Hg. If less than 20 all patients died. CCCR produced little improvement in values. Suggest that OCCR may be better.</td>
</tr>
</tbody>
</table>

### 2.7 Summary and conclusions

This chapter set out to position the reader as a skilled provider and offer a broad base as well as some detailed insights into what is happening and can be measured in basic life support and, more specifically, in aspects of importance to chest compressions. The steps and the psychomotor skills are clear, terms have been listed and an introduction to the underlying theory of cardiac and thoracic pump has been presented. How different monitoring techniques may support or impact outcomes have been listed, with special attention to the ultrasound investigations of the heart during CPR, since this pulls together all the different aspects "visually". It makes clear that it is not the lack of trying which has failed to clarify an optimally effective strategy.
Chapter 2: Technical issues in and around CPR

2.8 References

97. [ID-32] SEE ALSO ref 5!!


Noodergraaf GJ. Modeling the effect of interrupted compressions: less is better. Presented at the ERC-UK annual meeting 2005.


Chapter 2: Technical issues in and around CPR

...
Chapter 2: Technical issues in and around CPR


Facts and polite fiction: the report of a saga in research

(A historical, mechanistic approach to chest compressions: facts and polite fiction)
Chapter 3: A historical, mechanistic approach to chest compressions: Facts or polite fiction

3.1 Introduction ................................................................................................................................43
  3.1.1 Facts and polite fiction: the report of a saga in research...............................................................41
  3.1.2 The development in basic life support (circulatory aspects) .................................................................45
3.2 The later part of the first era (Egypt to 1600 BCE) .................................................................................46
3.3 The second era: 1960-1962 (period of rediscovery & application) .....................................................48
3.4 The third era: 1963–1968 (skepticism in blood flow) .................................................................................54
3.5 The fourth era: 1969–1976 (acceptance and complacency) .................................................................57
3.6 The fifth era: 1977–1990 (refinement, redirection & OCCR's return) ..................................................58
3.7 The sixth era: 1991–2004 (formalization and evidence based) ............................................................69
3.8 The seventh era: 2005–2022 (the return to core task) ............................................................................71
3.9 Adjuvant techniques to generate flow in basic life support .................................................................72
  3.9.1 The use of airway pressure: ....................................................................................................................72
  3.9.2 The use of abdominal binding: .............................................................................................................74
  3.9.3 Vest or cough CPR: ..............................................................................................................................75
  3.9.4 Prone CPR: .........................................................................................................................................76
  3.9.5 pGz CPR: .............................................................................................................................................76
  3.9.6 Studies of different "adjuvants" techniques in one model: .................................................................76
  3.9.7 Conclusions in technique in BLS: .........................................................................................................77
3.10 Mechanical chest compressions: a resolution? .................................................................................77
3.11 Chest compressions: conclusions in facts and polite fiction .............................................................79
3.12 References ..................................................................................................................................81
3.1 Introduction

Resuscitation after sudden cardiac or after sudden respiratory death is intended to support the vital organs by producing a form of effective circulation and oxygenation with as end point restoration of normal perfusion before irreparable damage is done. This fundamental understanding has not been at issue in the long history of attempts to resuscitate humans.

Clinically, the neonate may be a highly demonstrative model of this concept of resuscitation. During the birth process, if oxygenation is not maintained, finally, the heart will stop. The rescuer must open the airway, supply oxygen and perform chest compression to move the oxygen from the capillary circulation in the lungs to the coronary circulation to effectively restore the cardiac function. Only a few centimeters of effective forward flow are needed to achieve the desired purpose. However, even in this simple concept the fetal heart may already be too damaged to restore effective pumping.

In general, this Chapter approaches the history of cardiopulmonary resuscitation (CPR) from the angle that the underlying concept did remain unchanged, but that the approaches to the physiology and mechanics involved has undergone changes. Specifically, the focus will be, without striving for an all-encompassing listing, to review and analyze the theoretical and experimental work done in understanding the physiognomy involved in CPR.

3.1.1 Facts or polite fiction: resuscitation over time

Texts from the peak period of ancient Egyptian culture (prior to circa 1600 BCE) demonstrate awareness of airflow into the lungs and transfer of vital spirit into the blood of the lobes of the lungs. In the Torah, or Pentateuch, a younger Hebrew document probably written in the late fifth century BCE with new editions being formulated over the next several centuries, the concept is broadened to include the beginning of life (Genesis 2:7). Vivid descriptions of revitalization appear in the subsequent historical books (1 Kings 17:17-23 and 2 Kings 4:34-35).

In the scientific world, Vesalius (1555 CE) discovered a close relationship between respiration and cardiac pumping when he applied artificial respiration to animals and observed that the heart converted from its quiescent state to pumping. During the 18th and subsequent centuries, a wide variety of techniques were developed in an effort to improve the outcome of revitalization efforts. Although the primary interest was artificial ventilation, some of the techniques may have caused vigorous chest compression as a by-product (such as the prone victim supported crosswise over the back of a trotting horse), thereby likely providing compression of the thoracic cavity, including potentially the heart and thus impacting the circulation as well. The transition from compressing the thorax as a function of interest in causing respiration, and consequently at the respiratory frequency, to a compression frequency normal to the heart was the result of desperation, in a successful effort by Maas (1892), to save the life of a nine year old boy subsequent to chloroform administration in preparation for repair of a palatine fissure. He refers to Kraske (1887) as first to recognize implicitly of a "thoracic" pump model (i.e., blood moves due to increased intrathoracic pressure via pressure changes induced using the airway). Maas, using what could be described as a modification of Koenig's method, actually described his technique as "... in the excitement very fast and powerful. . .".

---

*a BCE: Before the Current Era: Reference to the Christian Calendar, while avoiding the specificity of the Christianity. “BC” (before Christ). Introduced (retrospectively) by the Scythian monk, Dionysius Exiguus, who, in or about AD 525, presented his “Easter table” with the new calendar to the representatives of Pope John I.*
Closed-chest resuscitation, in which the heart or the thoracic vasculature in general, is, directly or indirectly, compressed as well as open-chest resuscitation, in which the chest is surgically opened and the heart itself is massaged, were applied starting in the second half of the 19th century by Balassa in 1858,7 Schiff in 1874,8 Boehm in 1878,9 Koenig in 1883,10 and Ingelsrud in 190111 respectively. An anecdotal report is given by Sherwin in 1786, a surgeon of Enfield, reporting that "the surgeon should go on inflating the lungs and alternately compressing the sternum."12 Interest had been stimulated by the introduction of inhalation anesthesia, in 1848.13 The underlying philosophy was that open-chest resuscitation imitates the natural contraction and ejection of the two ventricles into the pulmonary artery and the aorta, by means of hand-supplied rhythmic massage. Initial reports of different applications of OCCR were all disappointing, such as that reported by Tuffier in 1898, with initial but no definitive success,14 while closed-chest massage imitates open-chest massage by compressing the heart between the sternum and the vertebral column as a fluid filled bag with one-way valves at its entrance and exits, gained footholds.15 This latter became known as the cardiac pump theory, and is solidly embedded in Harveyan teaching.16 It was brought to the attention of Kouwenhoven et al.17 in 1960. Physically, the heart was suggested to be compressed between sternum and vertebral column. Formal criteria, later described to be the essence of cardiac pump thinking is that:18

- Intraventricular pressures greater than intra-atrial pressures during CPR systole.
- Closure of the AV-valves during CPR systole with a development of a ventriculo-atrial gradient.
- An arterio-venous gradient to facilitate perfusion.
- Synchronous outflow from the right and left heart.
- Right heart inflow due to arterial to central venous pressure gradient during CPR systole.

The principle that the heart could be the functional pump, (the "cardiac pump theory" as a term did not appear until the 1980's), was challenged almost immediately in that same year by Weale et al.19 with concerns about the potential for bidirectional flows, with the suggestion that the whole of the thoracic cavity may act as the essential pump. It was Criley who, in 1976, coined the term "thoracic pump".20 This heralded a long period of interest in and of focus on what was moving the blood as formulated by Rudikoff et al., in 1980,21 more so than what type of intervention might move blood best. Hence, the current state envisions two, potentially competing or possibly complementary, theories why application of CPR, using the principle of closed-chest compressions, should benefit a victim of cardiac and respiratory standstill. Despite the fact that there are at least two theories of how the circulation is supported during CPR, little attention has been focused on the physiognomic mechanisms, even though the survival rate after CPR remained less than 25%.22

Both principles recognize that chest compression, not unlike normal expiration, increase intrathoracic pressure. Release of compression permits intrathoracic pressure to drop. Two relevant aspects must be considered to support or repudiate any of the available theories. First, in the spirit of Donders,23 that increasing intrathoracic pressure should promote filling of the right ventricle and therefore increase its outflow, since the right ventricle is subjected to the same increase in intrathoracic pressure. This argument alone has been advanced frequently in support of the thoracic pump theory. However, it presupposes adequate availability of blood volume in the central veins and does not resolve the issue of bidirectional flow in these central veins.24

The resulting scrutiny exposed some surprises. For the cardiac pump theory to apply effectively, some investigators determined that the mitral valve must be closed during CPR systole, but observed that the cardiac valves remain open throughout the CPR cycle, while others found that the tricuspid valve opens and closes in a systolic/diastolic cycle.25 Experimental work in animals,26 and in humans27 disclosed inconsistencies, such as equilibrium between the arterial and
venous pressures. Later work showed that the ventricles may barely be compressed by reducing the distance between the sternum and the spinal column, owing among other reasons, to their position outside the line of compression. In addition, it was observed that the recommended magnitude of chest compression (4-5 cm) may regularly be too small to cause compression of the ‘rubber ball’ of the ventricles even if they were in the line of compression. With regard to the thoracic theory, there was doubt about the actual availability of venous blood, especially after the first several compressions, leading to the idea that the venous return must be enhanced. It has been demonstrated that the respiratory system increases venous return during exercise in the normal human by lowering the intrathoracic pressure. More recent work has rediscovered a disassociation between the right and left circulations as a factor in inappropriate volume distribution and suggested that this is a dynamic component in CPR.

Closed-chest cardiac resuscitation has remained the popular method of choice owing to the strong emphasis on its teachability to lay persons and the desire to train a large number of people to perform rescue operations. This can be traced to the original work by Kouwenhoven, ”All that is needed is two hands.” While instruction of CPR is not the goal of this Chapter, a useful, if theoretical, definition of seven psychomotor aspects of basic life support (BLS) CPR have been developed.

In this Chapter, using the psychomotor skills involved in chest compressions and using recent historical references, evidence will be sought and discussed from the viewpoint of hemodynamics and mechanics. While interesting, specific outcome studies will be avoided as much as possible for reasons that will gradually become clear. The animal, bench and human studies will be reviewed semi-chronologically. Care will be taken to address only basic life support activities and to avoid complicating factors such as extrapolation of principle work to outcome studies, spin-off studies and unusual models.

As end point, this Chapter will offer the reader a basis in critical analysis of the body of evidence available to date, tempering the Guideline approach and supporting the feeling that fundamental understanding may still be the major missing key.

3.1.2 The development in basic life support (circulatory aspects)
Of the seven essential psychomotor skills in basic life support, four of these, hand position, compression depth, compression frequency, and compression-relaxation (duty cycle) directly impact the pumping mechanism. While discussed as if completely independent, all aspects must function in order to expect good outcomes remains implicit. While use of the airway (i.e., opening or closure, insufflation during compression) also may impact the circulation, this aspect will be spotlighted only when the issue at hand is aimed at the circulation itself. Each section will present a collection of general points, present the four components, and offer a summary with an analysis of the above. It remains the central contradictory aspect in chest compressions that while everyone knows that the quality of this activity is poor, patients still seem to survive. Is this despite that doing "anything" is (already) good enough and are scientists needlessly exited about the poor quality, or is this poor quality the bane of resuscitation medicine?

In light of the plethora of data available, an index system suggested by Criley organizes this focus into eras of interest and research. However, the pre-1960 research will also be described as a fifth era, as here the foundation, as well as some of the later limitations, of CPR may be found.
3.2 The later part of the first era (Egypt to 1960)

In 1906 Green wrote, with case reports, what is accepted as the first scientific report of OCCR in humans, though this is disputed. It is interesting to note that, in Keen's description of Crile's work of circa 1904, a pneumatic suit was used exclusively as compression device, that reaching a coronary perfusion pressure of 30-40 mm Hg is named as the essential difficulty, and the role of epinephrine was pointed out. Open-chest cardiac resuscitation (OCCR) became an accepted practice, as resuscitation was an in-hospital activity. Its principles and practice were described, in a textbook setting, as early as 1916.

The principle is not complex:

- Open the chest between the fourth and fifth ribs from just medially of the nipple to the mid-axillary line on the left side. Caution is needed to avoid the internal mammary artery which may bleed after successful resuscitation.
- The hand may be inserted with a rib spreader being used later.
- The hand should enclose both ventricles, but remain under the level of the annulus fibrosus.

Introduction of the hand through or under the diaphragm is also acceptable and may be done preferentially if the abdomen is open. Arrival to start of massage may be as quick as 10-15 seconds, as bleeding is not an initial issue. Opening the pericardium is possible but not required. An 94% overall definitive success rate is reported if OCCR is started within 4 minutes, decreasing to 6% is the massage is delayed longer than 4 minutes.

During the early 1930's, interest into creating movement with the vasculature developed secondary to support of the respiration and can be highlighted by the work done by Eve. In his 1932 paper, he describes fixing the patient to a table that can rock from -45° to +45° from the horizontal plane, using the weight of the intestines, the hydrostatic column formed by the great vessels and gravity to move the flaccid diaphragm in an effort to ventilate the patient after drowning or paralysis. (Figure 1a-b) In 1947 he reported that resuscitation of those drowned required both attention for the respiration and the circulation. His technique, involving four second head-down and three second head-up cycles allowed aortic-coronary flow and pulmonary flow, as well as venous return to the heart, respectively. He also calculated pulmonary artery pressures of 78 mm Hg based on the formula:

\[ H(\text{in}) \cdot \sin 45° \cdot (SG \text{ blood}/SG \text{ mercury}) \cdot 25.4 \text{ mm Hg} = PA \text{ and AO pressure} \]  

\text{(eq 3.1)}

i.e., H is the length of the column of blood in inches, "SG" the specific gravities of blood and mercury, and the sine the angle of horizontal with head down. These experiments were among the first to separate ventilatory from circulatory support, as well as to suggesting that adequate influence on both is required for survival.
In 1906 Green wrote, with case reports, what is accepted as the first scientific report of OCCR in humans, though this is disputed. It is interesting to note that, in Keen's description of Crile's work of circa 1904, a pneumatic suit was used exclusively as compression device, that reaching a coronary perfusion pressure of 30-40 mm Hg is named as the essential difficulty, and the role of epinephrine was pointed out. Open-chest cardiac resuscitation (OCCR) became an accepted practice, as resuscitation was an in-hospital activity. Its principles and practice were described, in a textbook setting, as early as 1916.

The principle is not complex:

- Open the chest between the fourth and fifth ribs from just medially of the nipple to the mid-axillary line on the left side. Caution is needed to avoid the internal mammary artery which may bleed after successful resuscitation.
- The hand may be inserted with a rib spreader being used later.
- The hand should enclose both ventricles, but remain under the level of the annulus fibrosus. Introduction of the hand through or under the diaphragm is also acceptable and may be done preferentially if the abdomen is open. Arrival to start of massage may be as quick as 10-15 seconds, as bleeding is not an initial issue. Opening the pericardium is possible but not required. An 94% overall definitive success rate is reported if OCCR is started within 4 minutes, decreasing to 6% if the massage is delayed longer than 4 minutes.

During the early 1930’s, interest into creating movement with the vasculature developed secondary to support of the respiration and can be highlighted by the work done by Eve. In his 1932 paper, he describes fixing the patient to a table that can rock from –45° to +45° from the horizontal plane, using the weight of the intestines, the hydrostatic column formed by the great vessels and gravity to move the flaccid diaphragm in an effort to ventilate the patient after drowning or paralysis. In 1947 he reported that resuscitation of those drowned required both attention for the respiration and the circulation. His technique, involving four second head-down and three second head-up cycles allowed aortic-coronary flow and pulmonary flow, as well as venous return to the heart, respectively. He also calculated pulmonary artery pressures of 78 mm Hg based on the formula:

\[ \text{H(in)} \times \text{sine 45°} \times \left( \frac{\text{SG blood/SG mercury}}{25.4 \text{ mm Hg}} \right) = \text{PA and AO pressure} \]

As early as 1946, Thompson et al. demonstrated that resuscitative efforts directed at treating asphyxia (with positive-negative ventilation) could also help move the circulation, by using dogs (14-20 kg, dead 20 minutes) and radioactive sodium, with the counting being performed at the carotid artery.

The statement, "... a heart too good to die", attributed to Beck (in 1947), was used to motivate internal defibrillation and OCCR, and has been expanded to include all aspects of CPR. Its inverse: a heart to poor to save” may have become a major confounder in resuscitation medicine.

**Compression frequency:**

During this period (up to 1960), no papers addressed compression frequency as principle end point. However, a number of papers raised interest in this area. Boehm, in his landmark paper (1878) describing closed-chest (contralateral) cardiac resuscitation CCCR in cats, says nothing on this subject. Note, however, that a normal heart rate for cats is circa 120-160 min⁻¹, with a stroke volume of 3-4 ml kg⁻¹ beat⁻¹. Koenig describing OCCR in a surgical textbook (more explicitly...
chloroform related cardiac depression) also did not make a point of compression frequency, although the implication was that normal (slow) compression frequencies were optimal. Maas suggested compression frequencies of ≥ 120 CPM, based on two case reports of CCCR in children, without motivating this choice or offering an opinion on the mechanism. Tournade, writing in French in the Compt Rend Soc de Biol. (1934), performed experiments with dogs and described transthoracic CCCR, but does not explain details of the procedure including any details on compression frequency.

Russian researchers, Gurvich and Yuniev, pointed out at the end of WW II, in a report of extensive experiments on 650 dogs, sheep and goats, in a study into defibrillation, that a potentially critical time limit is 90 seconds for successful defibrillation. No data on frequency was given.

Generally, while recognizing that a form of artificial respiration and circulation could be created by direct as well as closed compression of the heart, and that the two were both needed, little interest had been developed into the mechanisms involved. Based on the instructions by Stephenson, no compression pause was used to facilitate ventilation during OCCR and the clinically used compression rate was circa 60-85 compressions minute⁻¹. Since the chest was open, compression and ventilation were both autonomous.

Compression depth / force:

Julian, in a manuscript describing resuscitation in the 18th century, points to a quote by John Sherwin (in 1786, writing in the proceedings of the Royal Humane Society) that the surgeon should “...inflate the lungs and alternately compressing the sternum...” without saying how much or why. Boehm compressed the thoracic cavity of the cats side to side, but makes no mention of depth or force.

Duty cycle and hand position: No papers discuss these issues either as end point or indirectly.

Summary (up to 1960)

At the end of the 1950’s clinicians were comfortable performing OCCR, principally in the clinical setting. Successful cases had been reported both in and outside the operating rooms with up to 58% success rates in retrospective studies. Careful mention of closed-chest resuscitation being performed can be found, but little research had been done into its principles or mechanism.

3.3 The second era: 1960 – 1962 (period of rediscovery & application)

In 1960, the world of resuscitation changed completely. The focus changed from opening the chest as a clinical procedure done by surgeons and anesthesiologists, often due to iatrogenic complications, to a technique suitable for use by lay persons. Recognition of the circulation as central, including the effect of venous refilling became clear. Kouwenhoven et al., while working on dogs in a defibrillation study suggested that a structured approach to closed-chest resuscitation was possible, making the technique not only as good as open-chest resuscitation but also more generally applicable.

---

b Chloroform [Goodman & Gilman, The Pharmacological Basis of Therapeutics, 4th Edition, 1970. Macmillan Co. New York, NY., USA. Pages 83–84], known since 1831, used from 1847, more pleasant than diethyl ether. It causes dose-related arterial hypotension with abrupt bradycardia in a late stage. It is a myocardial depressant and reduces the arterial tone. It sensitizes the ventricular electrical system to tachyarrhythmias by increasing the sensitivity to catecholamine.

c Note that up through this date there were no reports of deaths secondary to the technique of resuscitation as such.
He suggested that 70% permanent survival was obtainable, while survival in OCCR was limited to 10-52% (mean 28%) overall, with restoration of an intact circulation in 56% of cases. Only in small part was death due to infection following the lateral thoracotomy (i.e., a rate of 4.7%). Kouwenhoven et al. suggest some of the mechanisms involved in creating flow:

- The heart is restricted in its movement in the thoracic cavity.
- It can be compressed by moving the sternum down 3-4 cm. This is done with the weight of the rescuer and causes an artificial circulation out of the heart during compression.
- Compression works best if the patient is lying on a firm surface.
- Artificial ventilation is required.
- Refill follows compression by releasing all pressure on the sternum.

The manuscript included pressure graphs and carotid artery flows, and was hailed as a landmark. Jude et al. followed up the initial report by Kouwenhoven in 1961, with a review of their first 118 patients at Johns Hopkins. In order to effectively continue the discussion, additional, technical details, as presented by Jude are noted here as:

- Compression frequency 60-80 minute⁻¹.
- Compression depth of 1-1.5 inches.
- Compression on the lower one-third of the sternum.
- Window to start resuscitation of 3-5 minutes should be respected.
- Patient physiognomy may impact force required.

Of these cases, while 78% had achieved return of spontaneous circulation (ROSC), 24% survived to leave the hospital. (Figure 2) When the cardiac cohort (24 patients) is inspected further, (age 40-82 years old, with CPR for 2-120 minutes) 20% reached ROSC but only 13% left the hospital. These papers became a ‘standard for comparison’.

Figure 2: Results compiled from Jude et al. These results demonstrate the location and outcomes. Of these 118 patients, 30% overall VF (EKG in 54% of patients), 78% had ROSC but 60% return of neurologic status, 24% leaves hospital. If separated to an AMI group (24 patients); these are then 40- to 82-year-olds, CPR 2-120 minutes and 20% achieve ROSC but 13% survive.

Kouwenhoven describes the “classic” population (asphyxia, hypovolemia, anesthesia gas and cardiac etiology), with ages between 2 months and 80 years and CPR duration of 1 minute to 65 minutes, both in and outside the operating rooms. Interestingly, he notes only three ventricular fibrillation cases.
As a word of caution, as early as 1961, Weiser et al.\textsuperscript{57} followed by Nachlas\textsuperscript{58} in the next era, suggest that experiments in dogs for reasons such as their size, weight and anatomy, might result in poor representation model of humans in experimental CPR.

**Compression frequency:**
Starting with the change to closed-chest resuscitation, the discussion of compression frequency started.

**Table 1: Compression frequency in second era**

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1960</td>
<td>Kouwenhoven\textsuperscript{32}</td>
<td>100 dogs (5-24 kg) (20 patients).</td>
<td>To extend time for successful defibrillation.</td>
<td>• Frequency used, 60 min\textsuperscript{-1}.</td>
</tr>
<tr>
<td>1961</td>
<td>Baringer\textsuperscript{59}</td>
<td>Retrospective patients.</td>
<td>NA.</td>
<td>• Frequency used, 60-90 min\textsuperscript{-1}.</td>
</tr>
<tr>
<td>1961</td>
<td>Dotter\textsuperscript{60}</td>
<td>Case reports.</td>
<td>Description validation of mechanical device.</td>
<td>• Frequency used, 80–100 min\textsuperscript{-1}. No supporting data supplied. Notes 60 CPM for mechanical device.</td>
</tr>
<tr>
<td>1961</td>
<td>Jude\textsuperscript{61}</td>
<td>Prospective patients.</td>
<td>Descriptive.</td>
<td>• Freq. used 60-80 min\textsuperscript{-1}</td>
</tr>
<tr>
<td>1961</td>
<td>Kouwenhoven\textsuperscript{62}</td>
<td>NA.</td>
<td>Descriptive.</td>
<td>• Frequency used 60–80 CPM. No supporting data.</td>
</tr>
<tr>
<td>1962</td>
<td>Birch\textsuperscript{63}</td>
<td>24 dogs and baboons in “V” cradle and sandbags.</td>
<td>Extend CCCR to pre-hospital by means of a mechanical device.</td>
<td>• Output = f (freq. 60 min\textsuperscript{-1} optimal)\textsuperscript{9}.</td>
</tr>
<tr>
<td>1962</td>
<td>Rivkin\textsuperscript{64}</td>
<td>27 dogs (10-20 kg) (32 patients).</td>
<td>Efficacy.</td>
<td>• Frequency of 30–40 per minute.</td>
</tr>
<tr>
<td>1962</td>
<td>Rivkin\textsuperscript{65}</td>
<td>90 dogs (70 patients).</td>
<td>Efficacy.</td>
<td>• Freq. 50-40 min\textsuperscript{-1} but no data given for this choice.</td>
</tr>
</tbody>
</table>

Kouwenhoven describes, complete with the historical references, the idea of closed-chest cardiac massage. No specifics on the dog experiments are given and neither are survival statistics presented. The authors suggest that up to 30 minutes of resuscitation is feasible, and note as a matter of course that the compression frequency is 60 CPM. In the 5 (of 20) cases described, the exact role of the CCCR may be subject to discussion. Baringer et al.\textsuperscript{59} report on the results from Massachusetts General hospital. While stating that the lower sternum should be sharply compressed 60-90 times minute\textsuperscript{-1}, this is not motivated. Nixon, writing in the Lancet, describes a patient successfully treated with a variation of OCCR, (80-90 CPM), who had gross mitral regurgitation, and was able to show arterial curves as an indwelling catheter had been placed pre-arrest.\textsuperscript{64}

Birch described the first study where mechanistic aspects of CCCR are controlled with output (i.e., blood pressure), in an experimental series in baboons and dogs.\textsuperscript{64} The device being tested could perform 60-90-100 cycles min\textsuperscript{-1} (CPM) while also allowing changes in the duration of systole from 30-70%, the force applied to the chest and the relationship between ventilation and compression in dog and baboon experiments. These experiments were performed as part of a validation of a mechanical compression device. They demonstrated that increasing the frequencies from 60 to 120 CPM with up to 60% duration of artificial systole improved arterial blood pressure. However, they conclude that the combination of a slow rate (60 CPM), with a long systolic period, generated the best arterial blood pressure in this model. Compression

\textsuperscript{4} In combination with long compression phase at 60-pound force.

50 Chapter 3: A historical, mechanistic approach to chest compressions: Facts or polite fiction
frequency and compression duration were inversely related. The group notes, as a matter of course, that positioning of the venous cannula was peripheral in order to avoid the high pressures seen in the vena cava. Rivkin et al.\textsuperscript{63} describing his conclusions as coming from both 90 dog experiments and 70 human cases, made in their first year of experience. He names his preference as 30-40 compressions min\textsuperscript{-1} without giving any experimental data to support this preference. He does state that deviation from the technique will cause death and that higher frequencies may cause acceleration injury and fatigue, but that they do not improve output.

Compression depth and/or force:
Movement of the sternum, strictly in an anterior-posterior direction, is the benchmark for chest compressions. During this, the hands or fingers should not contact the lateral chest, to avoid fractures outside the costochondral or costosternal borders. The discussion of impression depth, force, its units as well as the consequences for caregiver work, were also initiated immediately after the start of closed-chest compressions.\textsuperscript{65}

Table 2: Depth and force in compression in the second era

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1960</td>
<td>Kouwenhoven\textsuperscript{32}</td>
<td>100 dogs (5-24 kg) [20 patients].</td>
<td>Extend time for successful defibrillation.</td>
<td>Depth of 3-4 cm. No force listed. No data offered to support this choice.</td>
</tr>
<tr>
<td>1961</td>
<td>Harkins\textsuperscript{56}</td>
<td>NA.</td>
<td>Validation of a mechanical device.</td>
<td>Depth not listed. Force of 60-75 pounds (27-34 kg). No data offered as support.</td>
</tr>
<tr>
<td>1961</td>
<td>Dotter\textsuperscript{57}</td>
<td>Case reports.</td>
<td>Description and mechanical device.</td>
<td>Depth as 2 inches.</td>
</tr>
<tr>
<td>1961</td>
<td>Jude\textsuperscript{58}</td>
<td>Prospective patients.</td>
<td>Descriptive.</td>
<td>Depth used 1-1.5 inches. No force listed. No data to presented as support.</td>
</tr>
<tr>
<td>1961</td>
<td>Kouwenhoven\textsuperscript{32}</td>
<td>NA.</td>
<td>Descriptive.</td>
<td>Depth of 1-1.5 inches, achieved by weight of rescuer.</td>
</tr>
<tr>
<td>1962</td>
<td>Baringer\textsuperscript{60}</td>
<td>Retrospective patients.</td>
<td>NA.</td>
<td>Depth of 3-4 cm. No force listed.</td>
</tr>
<tr>
<td>1962</td>
<td>Beck\textsuperscript{59}</td>
<td>NA</td>
<td>NA</td>
<td>Depth? Force given as 90-120 pounds.</td>
</tr>
<tr>
<td>1962</td>
<td>Birch\textsuperscript{49}</td>
<td>24 dogs and baboons in 'V' cradle and sandbags.</td>
<td>Validate force and rate in mechanical compression.</td>
<td>Depth not listed. Force described in 40-80 lbs causing increasing arterial blood pressure. (90 CPM, duty cycle 40%).</td>
</tr>
<tr>
<td>1962</td>
<td>Rivkin\textsuperscript{62}</td>
<td>27 dogs (10-20 kg) [32 patients].</td>
<td>Efficacy.</td>
<td>Depth 3-5 cm. Force of 80 pounds (in the dogs at left side of sternum). No supporting data.</td>
</tr>
<tr>
<td>1962</td>
<td>Rivkin\textsuperscript{63}</td>
<td>90 dogs (10-20 kg) [70 patients].</td>
<td>Efficacy.</td>
<td>Depth is 3-5 cm. Force of 70-90 pounds. No supporting data presented.</td>
</tr>
</tbody>
</table>

If the heart is to be compressed by external compression, (i.e., between sternum and vertebral column) and sufficiently compressed to cause ventricular emptying, the issue of compression depth can not be understated.\textsuperscript{28} Kouwenhoven et al., describing dog (5-24 kg)\textsuperscript{6} experiments notes adequate circulation for \( \leq 30 \) minutes, and states as a matter of course that the compression depth

\begin{itemize}
  \item Optimal is at 60-pound force in combination with long compression phase.
  \item Note that weight, breed and size of dogs will become an issue later.
\end{itemize}
Duty cycle:

Duty cycle, or compression to relaxation ratio (C/R), the relationship during which pressure was build up, and even held, versus the release of pressure, including wait period, functioned as a

Figure 3: Poster developed by the California Medical Society (after Rivkin et al. 62,63). Note its similarity of current instruction.

Warltier is reported as noting that manual compression (i.e., the 70 pounds expected) is physically exhausting, and that the pulse obtained varies from stroke to stroke dependent on the length of time the rescuer has been active and their zeal.68 He developed a mechanical device (with a lever arm lock nut to limit range) for this purpose.

Duty cycle:

Duty cycle, or compression to relaxation ratio (C/R), the relationship during which pressure was build up, and even held, versus the release of pressure, including wait period, functioned as a

is 3-4 cm. Baringer et al., in the clinical case review from Massachusetts General hospital, applies a force suitable for 3-4 cm of compression depth as well as an important incidence of complication, including two to eight ribs in 33% of the 46 (of 84 in the series) autopsied patients. Harkins and Bramson start from a completely different premise, suggesting that accurate and reproducible compressions can only be done mechanically. They describe a compressed air-driven piston device, and note that "it is estimated that . . . a pulsating force of 60-75 pounds." The device (early Thumper™) is constructed to optimize the factors compression depth, C/R ratio (i.e., duty cycle) and force.66 They recognize operator fatigue. The 27-34 kg are unsupported in the manuscript which contains no experimental or clinical data.

Rivkin et al.62,63 suggested that the dorsal displacement of the sternum should be 3-5 cm as a result of 70-90 (80) pounds of force applied to the lower third of the sternum. He described two sets of 32 and 70 human experiments as well as 27 and 90 dog experiments in three separate studies, retrospectively, in two manuscripts. In his patient population, survival to discharge was 15/70 (21%) with only 5 requiring defibrillation. No motivation in the form of data is given for the stated depth or force. The California Heart Association prepared a poster (Figure 3) suitable for instruction and general dissemination.
point of interest in those developing mechanical devices, as ratios under manual compressions seemed difficult to control.

Table 3: Duty cycle in the second era

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1961</td>
<td>Harkins et al.</td>
<td>NA.</td>
<td>Validation of a mechanical device.</td>
<td>• Suggest that duty cycle is an important variable. No data offered as support.</td>
</tr>
<tr>
<td>1961</td>
<td>Jude et al.</td>
<td>Prospective, patients.</td>
<td>Descriptive.</td>
<td>• Implies a duty cycle of 1.</td>
</tr>
<tr>
<td>1962</td>
<td>Birch et al.</td>
<td>24 dogs and baboons in &quot;V&quot; cradle and sandbags.</td>
<td>Validate force and rate in mech. comp.</td>
<td>• Describe 30-70% systole with improving pressure with longer systolic period. Best at 60 CPM, 60 pounds, and 0.7 C/R ratio.</td>
</tr>
<tr>
<td>1962</td>
<td>Rivkin et al.</td>
<td>27 dogs (10-20 kg) {32 patients}.</td>
<td>Efficacy.</td>
<td>• Impression time 0.5 sec. At rate of 30 - 40 compressions min⁻¹, this is a duty cycle of 0.3 (long pause between compressions). No supporting data.</td>
</tr>
<tr>
<td>1962</td>
<td>Rivkin et al.</td>
<td>90 dogs (10-20 kg) {70 patients}.</td>
<td>Efficacy.</td>
<td>• Duty cycle 0.5 (at rate of 30-40 CPM).</td>
</tr>
</tbody>
</table>

Birch et al.\(^{61}\), working in animal models, found that 70% compression cycle supported better pressures in the femoral artery than shorter compressions (i.e., direct mention of 120 CPM, 60 pounds and 0.25 duty cycle). They concluded that slow rates and the long impression was optimal in this cardiac pump model. None of the other manuscripts approach this issue directly, and Rivkin, in the first manuscript,\(^{62}\) introduces the discussion of keeping the chest compressed (but not moving) as well as leaving the chest, unmoving, in its neutral position as the end phase of decompression.

Mechanistic and modeling aspects (1960-1962)

In one of the first studies comparing OCCR and CCCR, Weiser et al.\(^{57}\), using a dog model (22 dogs, 9-30 kg), introduced cardiac index monitoring (indocyanine green dye method) as well as the pathophysiology of coronary occlusion (microspheres) in a complex experimental setup and found low flows. This group also pointed out the danger in using pressure for flow calculations due to 'spiked' systolic ejection period. They conclude that OCCR produced better cardiac outputs (61% baseline, at lower frequencies of 30-60 CPM) than CCCR (21% of baseline), and that the anterior-posterior diameter of the dog's chest was a factor. In closed chest, the rates of 60-120 produced comparable outputs and pressure (dogs > 14 kg). They conclude that they, in line with Redding and Cozine,\(^{69}\) were unable to demonstrate superiority or even equality in closed-chest resuscitation when compared to OCCR. A long series of case reports appeared as well as concerns about complications.

Brief summary of the second era

This period may be characterized as working with the new technique of closed-chest resuscitation, and recognition that artificial ventilation should be combined with circulatory interventions. Little direct validation was, as yet, sought and even less interest in the mechanics or modeling. One group, Birch, et al.,\(^{61}\) looked at the cardiac pump model and attempted to optimize details. Many of the psychomotor skills are as yet not recognized. Jude mentions the importance of cardiac valves,\(^{56}\) but does not mention Gallet (who wrote in French) in expressing concern that manual compression of the heart would "only produce an ebb and flow movement."\(^{70}\) The 70% definitive success rate mentioned by Kouwenhoven was not repeated by others, while some poor outcomes (4% of 84 patients) are described.
An important aspect in this period is that the focus of treatment has shifted away from the operating rooms to patients with acute myocardial infarctions/acute collapse syndromes.

### 3.4 The third era: 1963–1968 (skepticism in blood flow)

Immediately after the initial "popular" response to CCCR, many reports were published questioning the indication (to leave OCCR), the outcome, the mechanism by which CCCR should work and reporting on significant incidence and severity of complications when using this technique.

This era distinguishes itself in, on one hand, acceptance of the basic principles (i.e., technical skills) described and the beginning of the search as to why the original successes could not be reproduced. This led, initially, to a brief period of complex comparative studies between OCCR and CCCR.

**Compression frequency:**

In 1963, acceptable practice was a slow, (circa 60 CPM) tempo of compressions. Ventilations were interposed.

#### Table 4: Compression frequency in the third era

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1963</td>
<td>Del Guercio</td>
<td>Humans, case reports</td>
<td>Objectify actually blood flow in CCCR.</td>
<td>• Reports 88 and 120 CPM (accidentally used, with invasive pressure and CO measurements in three case studies. Suggests 120 CPM appropriate for children.</td>
</tr>
<tr>
<td>1963</td>
<td>Nachlas</td>
<td>55 dogs (9-21 kg) in &quot;V&quot; cradle.</td>
<td>Construction of a mechanical compression unit.</td>
<td>• 30-52 CPM. No outcome differences (use scoring system). Also correct for percent of resting heart rate. Suggest ( \geq 25% ) of control.</td>
</tr>
<tr>
<td>1963</td>
<td>Warltier</td>
<td>NA</td>
<td>Construction of a mechanical device.</td>
<td>• 60 CPM. No supporting data given.</td>
</tr>
<tr>
<td>1964</td>
<td>Himmelhoch</td>
<td>65 human patients.</td>
<td>Descriptive.</td>
<td>• Finds no benefit for slower rates than 80 CPM.</td>
</tr>
<tr>
<td>1964</td>
<td>Jude</td>
<td>NA</td>
<td>Extensive review</td>
<td>• States that dog more difficult for CCCR than the human. The rate should be of normal cardiac contraction (child 100-120, adult 60-80 CPM). No supporting data supplied</td>
</tr>
<tr>
<td>1967</td>
<td>Harris</td>
<td>14 dogs (8-12 kg).</td>
<td>Optimal combination of compression to ventilation.</td>
<td>• Rates of 48-60-72 and 120 CPM. Carotid flow measure with rotameter. Strong improvement with increasing number of CPM. Suggest that 70-80 CPM in realistically possible for rescuer.</td>
</tr>
</tbody>
</table>

Guercio et al., points out that pressures and flow should be carefully separated, from a circulatory point of view, stating that valvular incompetence, and brief ejection periods may limit
forward movement. He also notes that a bed board made of plywood (1.9 cm thick) is an appropriate support when resuscitating in bed. Nachlas et al.\textsuperscript{58} supports the point of view that flows and circulatory pressures become disassociated during CPR. Thomsen, in a clinical case series, directed principally at the left heart, described the measurements as 'fluctuations caused by CCCR', and wonders whether these are pulse waves, or ineffective shock waves. He notes that left atrial and femoral artery pressures were equal during the CCCR.\textsuperscript{75}

**Compression depth:**
Interest developed into the issue of compression depth in this period, 1-1.5 inches became more a point of departure then a goal.

**Table 5:** Compression depth and force in the third era.

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1963</td>
<td>Nachlas\textsuperscript{58}</td>
<td>55 dogs (9-21 kg) in &quot;V&quot; cradle.</td>
<td>Construction of a mechanical compression unit.</td>
<td>Note 74-104 pounds (90-130 pounds with manual) applied in animals of 8.6–21.6 kg. No force to arterial pressure relationship found.</td>
</tr>
<tr>
<td>1963</td>
<td>Warltier\textsuperscript{72}</td>
<td>18 human patients.</td>
<td>Construction of a mechanical device.</td>
<td>Depth of up to 10 cm (minimum at 2.5 cm), with no damage at autopsy. Force of 30–40 kg. No supporting data given.</td>
</tr>
<tr>
<td>1964</td>
<td>Jude\textsuperscript{9}</td>
<td>NA.</td>
<td>Extensive review.</td>
<td>1.5-2 inches (4-5 cm). Force of 60–100 pounds.</td>
</tr>
<tr>
<td>1964</td>
<td>Johansen\textsuperscript{96}</td>
<td>Humans, (8, anesthetized and apnic volunteers).</td>
<td>Depth-force relationship.</td>
<td>Depth of compression 0.8-2.0 mm kg\textsuperscript{-1} (average 1.3). Suggests depression proportional to force.</td>
</tr>
</tbody>
</table>

The tendency is towards an increase the depth of compression as well as the force involved. The number of studies with validation of mechanical compression devices supports the view that CCCR is hard work if done well.

**Duty cycle:**

**Table 6:** Duty cycle in the third era

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1963</td>
<td>Nachlas\textsuperscript{58}</td>
<td>10 dogs (9-21 kg) in &quot;V&quot; cradle.</td>
<td>Construction of a mechanical compression unit.</td>
<td>Report that most attempt 1/3 systole and 1/3 diastole. Under 30 CPM, for 30 minutes, ±30% was not different from ± 50% duty cycle.</td>
</tr>
<tr>
<td>1964</td>
<td>Jude\textsuperscript{9}</td>
<td>NA.</td>
<td>Extensive review.</td>
<td>States &quot;force rapidly down, stroke held for one-half a second.&quot; Then rapidly and totally release (0.7 duty cycle); ref. Birch.\textsuperscript{51}</td>
</tr>
</tbody>
</table>

Duty cycle seems to only apply in investigations involving mechanical compression devices.

**Mechanistic and modeling aspects (1963-1968)**
The role of the dog as a model is noted with concern by Nachlas et al.\textsuperscript{58} who point at the long thoracic cage, the high liver, and the mobile mediastinum as complicating factors in translating findings to humans, as well as the anterior-posterior chest depth. During this period, the first theoretical and computer-oriented models of the circulation became available.\textsuperscript{78,79,80}
Permutt and Riley, in 1963, brought the concept of critical closing pressure, Poiseuille's law and the vascular waterfall in arterioles for a mechanistic modeling point of view. They point out that not only in and outflow pressure determine flow, but that the critical closing pressure, anywhere in the collapsible vessel, may replace the outflow pressure in the equation.

**Adjuvant techniques** begin to be seen, whether in reaction to the low flow states or as a by-product of general investigation is not discussed. Wilder et al. in a carefully orchestrated experimental setup with dogs (10-15 kg), performed the first simultaneous compression-ventilation (SCV-CPR), implicitly applied this principle, and found the technique to be more than 30% better than just CCCR at 60 CPM. Even Harris et al. were unable to reproduce this in their experiments a few years later. The same group also presented the use of (static) abdominal binding (aAB-CPR) to augment CPR, as well as initial experiments with epinephrine and volume expansion as adjuvants geared to improve flow. Abdominal binding, while increasing intrathoracic pressure, impacted not only diastolic arterial pressure, but also on right atrial and central venous pressures adversely effecting CPP. This study, for other reasons, became the basis for the ventilation-compression 2:15 and 1:5 ratios.

Nachlas et al. became concerned about dog experiments and human cases where low blood pressures (i.e., 50/25 mm Hg) and low flow states persisted despite optimal CCCR. They suggest adjuvant techniques and, working in dogs, used aortic occlusion (effect), norepinephrine (seeming improvement), and intravascular diastolic pumping (technically difficult and little hemodynamic effect). Different mechanical devices (i.e., pistons) were developed and were in use to regulate chest compression techniques.

MacKenzie et al. opened the door (early) for the thoracic pump principle, as they observed equalization of pressures in all cardiac chambers, and reflects on similar findings by Weale and Rothwell-Jackson. Series of reports (cases) with rib fractures, bone marrow or fat emboli, topping the list as well as less frequent aortic dissection, esophageal, liver, and myocardial rupture, pneumothorax, spleen and stomach damage. The tendency is towards a decreasing incidence. Survival slides towards the 33% and to as low as 6%.

The role of acidosis and of epinephrine as a vasopressor during CCCR became a focus with Pearson and Harris et al. performing a series of experiments in dogs (8-12 kg) in comparison with a series using volume expansion. No reference is made to the concerns stated by Nachlas in 1963, that the use of epinephrine impacts the coupling of arterial pressure and flow during CPR.

**Brief summary of the third era**

In 1966 the first ad hoc committee on cardiopulmonary resuscitation published the first Guidelines and states that there is clear clinical evidence for the efficacy of external, manual chest compressions. The performance of the activities involved in resuscitation had been designated as emergency procedure, bringing it outside the medical domain in mid 1965. As standards, 80-120 pounds (1.5-2 inches), on a firm surface, at a rate of 60 CPM was given. A duty cycle of 1 was explicitly stated.

This statement is surprising in an era during which the process of discovery was still in full swing. Actual data supporting the choices can not be found in the Tables above, so one can only conclude that generalization of the technique to make it available outside the hospital was paramount.

---

Chapter 3: A historical, mechanistic approach to chest compressions: Facts or polite fiction
3.5 The fourth era: 1969–1976 (acceptance and complacency)

The fourth era can be characterized by extensive reports of the use of external or closed-chest massage. Some 45 case reports, descriptive manuscripts as well as comments and concerns about the technique can be found. The technique is also clearly becoming ‘institutionalized’ with manuscripts appearing which are concerned about responsibilities and practice at in-hospital settings, such as the developments of cardiac resuscitation teams.

Compression frequency:

Table 7: Compression frequency in the fourth era

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1976</td>
<td>Ohomoto</td>
<td>50 dogs (10-30 kg)</td>
<td>IAC-CPR or countermassagge</td>
<td>70-90 CPM. No data given.</td>
</tr>
</tbody>
</table>

Compression depth and force:

Table 8: Compression depth and force in the fourth era

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1971</td>
<td>Redding</td>
<td>Dogs (5.5-18.1 kg)</td>
<td>AC-CPR</td>
<td>Notes that force used is such as to allow 50-100 mm Hg aortic pressure. Other data not supplied.</td>
</tr>
<tr>
<td>1976</td>
<td>Ohomoto</td>
<td>50 dogs (10-30 kg)</td>
<td>IAC-CPR or countermassagge</td>
<td>3-4 cm sternal compression, 4 cm abdominal. Force: 1.9 kg/cm² for chest, 2.8 kg/cm² for counterpulse device and 0.05 kg/cm² for abdominal binder. No data given for choices.</td>
</tr>
</tbody>
</table>

Duty cycle:

Table 9: Duty cycle in the fourth era

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1976</td>
<td>Ohomoto</td>
<td>50 dogs (10-30 kg)</td>
<td>IAC-CPR or countermassagge</td>
<td>0.27–0.3, including brief ‘hold-compression’ phase. The study involved phase shifts with abdominal compression device.</td>
</tr>
</tbody>
</table>

Adjuvant techniques:

Table 10: Adjuvant techniques reported in the fourth era

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1971</td>
<td>Redding</td>
<td>155 dogs (5.5 – 18.1 kg)</td>
<td>Efficacy of CCCR (s)AB-CPR.</td>
<td>Abdominal binding: 150-200 mm Hg. Reports across the board (moderate). Notes that AC-CPR does not augment venous return improvements. Mechanical chest compressions.</td>
</tr>
<tr>
<td>1976</td>
<td>Criley</td>
<td>3 human patients</td>
<td>Descriptive.</td>
<td>Coughing maintains consciousness for &gt; 35 sec (as validation of cerebral flow) in cath room VF. Recognized the need for a competent aortic valve and SVR to have A-V flow in diastole.</td>
</tr>
<tr>
<td>1976</td>
<td>Ohomoto</td>
<td>50 dogs (10-30 kg)</td>
<td>IAC-CPR or countermassagge</td>
<td>Finds 1.4 to 2 fold increases of coronary and carotid flow, using flow sensors. No additional trauma.</td>
</tr>
</tbody>
</table>
Redding describes (static) abdominal binding (sAB-CPR), a mechanism intended to increase redirect blood flow, following earlier work by Harris et al. The principle is based in OCCR and may increase venous return. However, peripheral venous pressure, with retrograde flow are also (severely) increased, with negative effect on CPP, increased ICP and diminished cerebral flow content. He also notes a complete correlation in survival above and below aortic diastolic pressures of 40 mm Hg, supporting Crile.

Mechanistic and modeling aspects in the fourth era:
Interest in adjuvant techniques continued, with Anstadt et al. following up earlier reports of extended success in resuscitation of hearts with ventricular fibrillation using a direct (internal-external) ventricular compression device.

Perhaps the most striking development in the fourth era was the paper by Criley et al. reporting coughing as a pumping mechanism for non-perfusing heart rhythms. This initiated new enthusiasm as well as what almost became a major chasm among CPR scientists.

Brief summary of the fourth era
The years 1969-1976 brought little by way of innovation. AHA standards confirms the central role of the airway, changes the rate of compression to 80 CPM for one-rescuer and maintains 60 CPM for two-rescuer incidents. The direct involvement of the operating suite has become de-emphasized, even though the rate has not gone down (1:1,216, survival rate 42%). While manual (i.e., mechanical) as well as automatic chest compressors are acceptable, the Guidelines express concern in the length of time needed to place then the latter. OCCR remains an acceptable option if CCR is deemed ineffective (i.e., barrel chested patients).

Little fundamental experimental investigation was performed, although the serious adjuvant techniques continued to be developed and tested by investigators who were disappointed in flow, arterial pressures and survival.

3.6 The fifth era: 1977–1990 (refinement, redirection & OCCR’s return)

The 1980s enlivened the discussion in cardiopulmonary resuscitation greatly. Theory gained a foothold as the cardiac pump and thoracic pump principles threaten to divide thinking. New, fundamental as well as technical developments, such as echocardiography, offered insights. Models, such as the dog, for general resuscitation research were questioned. The complexity of the research achieved new heights as adjuncts continued to be developed in the face of disappointing outcomes.

However, as pointed out by Jackson and Freeman in a 1983 review, the rekindling of interest was at least in part due to the insight that OCCR produced, at best 30% of baseline cardiac output and only 10% of normal, while OCCR produced 50% of normal cardiac output and almost normal cerebral blood flows. While they assumed pure thoracic pump mechanism, clear recognition of the need for some form of improved pumping mechanism was strongly advocated for CCR.

In one of few studies in which hand position (i.e., compression point) is an explicit end point, Barsan et al. investigated cardiac output in relationship to hand position, using a thermodilution...
(SG) technique in large dogs with supported thoracic cavities in both OCCR and CCCR at 60 CPM. They note that there is no difference in side-to-side, mechanical or anterior-posterior compressions and can find only 13% of CO seen under control conditions. The OCCR generated 50% of baseline cardiac output. No force or depth in compressions of any of the three types is noted. OCCR was noted as superior to CCCR.

Not only hand position and rescuer fatigue in reaching 4-6 cm, but also the weight of the rescuer is recognized well ahead of their time by Bilfield and Regula who suggested, based on a manikin study by students, that leg-heel compression might be better in two-rescuer situations.96

In 1990, Chandra97 expresses deep concerns about the continued use of the (small) dog model in CPR, pointing to Barsan95, and relates this to the discussion about pump theory. Chandra, a proponent of the thoracic pump theory and the use of the abdomen in CPR, concludes that, despite clear anatomical differences, the large dog is suitable for laboratory use if confounders are carefully controlled.

The resurgence of OCCR:
Studies directed at the discussion between OCCR and CCCR. Little is known of the real survival from cardiac arrest after return of spontaneous circulation (ROSC) following OCCR during its early use.40,98 It was deemed too difficult to teach to lay persons. However, in 1984 Sanders (con),99 DelGuerico (pro)100 as well as Bircher101 argued about the relevance of open-chest resuscitation.

Table 11: OCCR vs CCCR in the fifth era

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1980</td>
<td>Bircher102</td>
<td>10 dogs (10-15 kg and flat chested).</td>
<td>Evaluation of neurological function.</td>
<td>OCCR done after lengthy CCCR (thumper) showed return of EEG activity. Difficult to interpret clinically, as realistic indicator of good neurological outcome.</td>
</tr>
<tr>
<td>1980</td>
<td>Byrne103</td>
<td>Dogs.</td>
<td>Cerebral perfusion.</td>
<td>Using radiolabeled microspheres to evaluate flow. Showed that CCCR was only 30% of control while OCCR was normal. Note that systolic blood pressure was standardized in these experiments.</td>
</tr>
<tr>
<td>1984</td>
<td>Arai104</td>
<td>15 dogs (8.5-15.5 kg).</td>
<td>Evaluate the efficacy of CPR on cerebral hemodynamics.</td>
<td>OCCR was compared to classic CCCR and SVC-CPR (also known as 'new' CPR) using dogs in lateral decubitus. Only OCCR was shown to produce flows perceived as adequate in this extensively instrumented study.</td>
</tr>
<tr>
<td>1984</td>
<td>Bartlett105</td>
<td>15 dogs (average weight 26.5 kg).</td>
<td>Compare CCCR with OCCR and mechanical OCCR device.</td>
<td>A crossover study design demonstrated that OCCR 71% of control is better than CCCR (flow at 19% of control) and that mechanical device is better still. Notes that a rate increase from 60 to 90 CPM using OCCR returns values to baseline. Also notes higher intracranial pressures in CCCR.</td>
</tr>
<tr>
<td>1984</td>
<td>Bircher106</td>
<td>Dogs.</td>
<td>Determine the</td>
<td>Very low CCCR outcomes versus</td>
</tr>
</tbody>
</table>

Barsan notes, as a matter of course, that they weigh more than 20 kg, as he considers smaller dogs unsuitable. He also points out anatomical difficulties (high liver, narrow sternum, unstable position in prone position.)
<table>
<thead>
<tr>
<th>Year</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1985</td>
<td>Bircher</td>
<td>32 dogs</td>
<td>Flow and neurological outcome.</td>
</tr>
<tr>
<td>1985</td>
<td>Raessler</td>
<td>10 dogs in Ketamine Pentobarbital model</td>
<td>OCCR versus CCCR.</td>
</tr>
<tr>
<td>1985</td>
<td>Sanders</td>
<td>32 dogs (17-27 kg)</td>
<td>Determine if OCCR after CCCR is relevant to survival.</td>
</tr>
<tr>
<td>1985</td>
<td>Babbs</td>
<td>Electrical model.</td>
<td>Study pump theory.</td>
</tr>
<tr>
<td>1985</td>
<td>Carden</td>
<td>14 dogs</td>
<td>Determine the time sensitive course of lactic acidosis in CCCR.</td>
</tr>
<tr>
<td>1985</td>
<td>Kern</td>
<td>29 dogs (20-25 kg)</td>
<td>Investigate survival differences between CCR and CCCR.</td>
</tr>
<tr>
<td>1985</td>
<td>Kern</td>
<td>NA</td>
<td>Determine time-limitations until which OCCR is useful.</td>
</tr>
<tr>
<td>1988</td>
<td>Raessler</td>
<td>63 mongrel dogs (7-32 kg)</td>
<td>Effect of technique on pressures.</td>
</tr>
<tr>
<td>1988</td>
<td>Henneman</td>
<td>16 human patients.</td>
<td>Development of acidosis in ongoing CPR.</td>
</tr>
</tbody>
</table>

- 28% discharge rate for OCCR. Low wound (< 9.1%) and iatrogenic cardiac damage (≤1.4%) reported.
- After 30 minutes of CCCR or OCCR, neurological outcome was good in only the OCCR group.
- Cardiac arrest induced with KCl. SG catheter in situ as well as sinus sagittalis catheter.
- Cerebral blood flow better in OCCR (baseline 42 to 18 ml/100g/min versus 38 versus 5 for CCCR.
- OCCR better than CCCR in this small series.
- In a pentobarbital and epinephrine, four groups were made, CCCR, and OCCR after CCCR and 15, 20, or 25 minutes of insufficient CPP. Generally, four-fold increases in aortic systolic and diastolic pressures were seen while CPP increased 10 to 30 fold. Concludes that "not last ditch" effort (even if after 20 minutes).
Enough interest resurfaced to suggest a study, performed by Barnett et al.\textsuperscript{117} into details, such as which different hand positions were most optimal. While no real differences in effectiveness were shown between internal techniques, all were found to be superior to CCCR.

Surprisingly, and despite the studies showing improvements while no studies showed a negative influence, in the formal sense, OCCR remains recommended for consideration in certain, specified situations (AHA Guidelines 1986\textsuperscript{118} and 1992\textsuperscript{119}).

**Compression frequency:**

The question of rate effects in CPR is essentially one of pump theory. If compression rate as opposed to compression duration is an effective influence on survival (as end point for better flows and pressures) this would support the cardiac pump thinking, while the latter would support the thoracic pump (i.e., recruitable volume) theory.

### Table 12: Compression frequency in the fifth era

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1977</td>
<td>Taylor\textsuperscript{120}</td>
<td>8 patients.</td>
<td>Effect of CPM and duty cycle on flow in carotid artery (Doppler).</td>
<td>Frequency of 40-60-80 CPM at 60% duty cycle did not impact flow.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Notes: only small backflow during CPR diastole.</td>
</tr>
<tr>
<td>1978</td>
<td>Taylor\textsuperscript{121}</td>
<td>80 patients.</td>
<td>Manual versus mechanical.</td>
<td>States frequency of 60 CPM. No supporting data given.</td>
</tr>
<tr>
<td>1981</td>
<td>Fitzgerald\textsuperscript{122}</td>
<td>20 dogs (6-12 kg)</td>
<td>Rate effects.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• CO = CR x SV\textsubscript{max} x {DC/(k\textsubscript{1} x CR + DC)} x {(1 – DC/(k\textsubscript{2} x CR + 1-DC))}.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• Based on rates of 20-140 CPM net or gross and compression duration of 10–90%.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• In graph peak output is reached at about 90 CPM. SV decreases steadily with increasing CPM. Notes SV\textsubscript{max} dependant on size of patient, force of compression, relative blood volume, venous filling pressure, and possible arterial collapse.</td>
</tr>
<tr>
<td>1984</td>
<td>Maier\textsuperscript{123} (2000 S and G)</td>
<td>24 chronically instrumented dogs, (25-35 kg) -uses lateral and supine positions as well as cradle.</td>
<td>Effect of compression rate on CO in CCCR (part of larger whole series of experiments, no allowance for time-sensitive course noted).</td>
<td>Notes that intrathoracic pressure (i.e., peak intrapleural pressure ≤ 20 mm Hg) was used a compression goal versus compression depth.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• Find that stroke volumes of ca. 7 ml are maintained between 60 and 150 CPM, with CO increasing strongly (2–3x) to with rate. MAP increases from 44–59 mm Hg.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• Coronary artery blood flow stays at 65–75% of controls.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• Supports cardiac pump theory.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• Some confusion is noted as to “net”</td>
</tr>
</tbody>
</table>

\textsuperscript{1} Definitions: CR = compression rate, DC = duty cycle, SV\textsubscript{max} (19 ml) effective volume of pumping chamber, k\textsubscript{1} = 0.00207 min, k\textsubscript{2} = 0.00707, respectively ½ of emptying and filling time for dogs. SV\textsubscript{max} for humans = 70 ml.
ventilations: (60 seconds/16 seconds) x 15 = 56 CPM netto.

Free periods for ventilation (i.e., 2 ventilations in 6 seconds: 10 sec for 15 compressions plus 6 seconds for 2 in 10 seconds): (60 seconds/10 seconds) x 15 = 90 CPM bruto. Netto is the frequency after correction for compression displaced.

Cardiac deformation was 4.6 ± 0.1 cm (in their dog model) This is almost the total max. distance the sternum is displaced!

Note that the rates and durations (duty cycles) chosen lie outside those demonstrated by Maier 123, 126 at maximum

* 1988 Halperin**23** Dogs, 21-36 kg - microspheres. Thoracic pump versus cardiac pump, with rate as indicator. • Thoracic pump (indicators: applied force and compression duration (duty cycle) but not compression rate effect output) versus cardiac pump (compression rate, force but less sensitive to compression duration).
  • Aware of the work by Maier 123, in effect sought a pure thoracic pump (vest-CPR) versus pure cardiac pump (OCCR) models. These outcomes were compared with specific sub-protocols. This study concludes that rate is NOT an important variable and that blood flow is produced by the thoracic pump theory.
  • Note Table 4 of their manuscript does not support their own conclusions.

* 1986 Kern**25** Dogs, (20-32 kg), three models - Morphine model, with use of thoracic splinting. Effect of high impulse CPR. • Could find no initial, 24-hour or definitive differences between high impulse (120 CPM, 20% C/R) and interposed abdominal compressions with 60 CPM as opposed to AHA Guidelines CPR of 60 CPM, 50% C/R ratio.

* 1986 Maier**28** HIP-CPR, chronically instrumented. Mechanism of HIP and rate in CCCR. • Has review characteristics.
  • Suggests that HIP shown only a 20 mm Hg intrapleural pressure surge.
  • Sown cardiac dimension changes.
  • Conclude return of consciousness at rates of 120-150 CPM.

* 1987 Fleisher**27** Dogs, 3-11 kg - cradle fentanyl /dopiridol model Effect of rate in child CPR. • Extensively instrumented with sinus sagittalis cath and LV cannula. N2O and microspheres used for CI measurements. KCl to induce arrest.
  • In a study designed for child CPR and using 40 or 120 CPM. When compression 30% diameter of the AP thoracic distance of the puppy, they found NO difference with rate change as all rates were inadequate.
  • "Arbitrary standards continue to dictate..."
  • Notes depth of compressions = 1.5–2 inches as 30% AP diameter.

* 1988 Kern**24** 60 dogs. Effect of compression. • Evaluated 6 defined methods in 60 dogs, but combined all values in an investigation geared to defining survivability based on myocardial blood flow. No data on frequency is

---

1. Bruto is the frequency of compressions during a compression block extrapolated to one minute (i.e., 15 compressions in 10 seconds): (60 seconds/10 seconds) x 15 = 90 CPM bruto. Netto is the frequency after correction for compression free periods for ventilation (i.e., 2 ventilations in 6 seconds: 10 sec for 15 compressions plus 6 seconds for 2 ventilations: (60 seconds/16 seconds) x 15 = 56 CPM netto.

2. Note that the rates and durations (duty cycles) chosen lie outside those demonstrated by Maier 123, 126 at maximum effect in circa 90 CPM and 50% C/R cycle.

3. Note that on pp. 546 of this manuscript: During OCCR (anterior chest wall removed), the direct antero-posterior cardiac deformation was 4.6 ± 0.1 cm (in their dog model) This is almost the total max. distance the sternum is displaced!
free periods for ventilation (i.e., 2 ventilations in 6 seconds: 10 sec for 15 compressions plus 6 seconds for 2 in 10 seconds): (60 seconds/10 seconds) x 15 = 90 CPM brutò. Netto is the frequency after correction for compression displaced!

Cardiac deformation was 4.6

Note that the rates and durations (duty cycles) chosen lie outside those demonstrated by Maier 123,126 at maximum effect in circa 90 CPM and 50% C/R cycle.

1986 Halperin 124 Dogs, 21-36 kg
1986 Kern 125 Dogs, (20-32 kg),
Kern 128 60 dogs. Effect of

Chapter 3: A historical, mechanistic approach to chest compressions: Facts or polite fiction 62

± 0.1 cm (in their dog model) This is almost the total max. distance the sternum is with use of - Morphine model, - Supine in cradle no allowance for time-sensitive

- Morphine model
- cradle fentanyl
- microspheres.

Mechanism of HIP Effect of rate in

Effect of high compression. Effect of rate in

Conclude return of consciousness at

Table 13: Compression depth and force in the fifth era

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1978</td>
<td>Taylor 121</td>
<td>80 patients</td>
<td>Manual versus mechanical.</td>
<td>States up to 7 cm with mechanical device. No data or supporting data given.</td>
</tr>
<tr>
<td>1979</td>
<td>Vallis 125</td>
<td>11 patients in Emergency Department</td>
<td>Determine force need for compressions</td>
<td>Using pressure sensing device, until a femoral pulse was felt. They found</td>
</tr>
</tbody>
</table>

In this study, initial success was defined as defibrillation with restoration of circulation and minimal systolic blood pressure of 60 mm Hg.
<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
</table>
| 1983 | Babbs 134 | 8 dogs (8-12 kg), chosen as young, compliant chests and AP to right-left diameter < 1.6:1, in cradle. | Compression depth threshold. | • Pentobarbital model.  
• Using mechanical (Thumper) CPR, but steered using esophageal pressure 20-80 mm Hg of pressure in increments of 10 mm Hg. Depression depth was then measured.  
• Suggests that CO is linearly related to compression depth by CO = a(x-x₀) where a is a slope of line and x₀ is a threshold value.  
• Notes threshold in these dogs at 2.3 cm, and effective improvements in flow up to 50 mm Hg esophageal pressure. |
| 1983 | Tsitlik 135 | 7 + 4 patients. | Evaluation of elastic properties of the human chest. | • Use the Thumper in a incremental study with steps of 20 N up for 0 to 310-540 N.  
• Suggests F = β Ds + γ Ds² where β = ± 92 N/cm², γ = ± 55 N/cm², and Ds = sternal displacement. However, the clinical data only goes to compression depth of < 5 cm.  
• 4 (older) patients had previous CPR; the rest were young. |
| 1984 | Maier 132 | 24 chronically instrumented dogs (25-35 kg).  
-Uses lateral and supine positions as well as cradle.  
"high-impulse CPR" | Validation of HIP-CPR. | • Chronically instrumented dogs (morphine model, and Swan-Ganz thermodilution with electromagnetic flow probes) investigated "high-impulse" CPR based on supposition CO = Freq x SV.  
• Also varied compression force (12-32 mm Hg intrapleural peak pressure) and found little improvement in stroke volume (middle was p<0.5 better than low). |
| 1988 | Swenson 136 | 9 human patients after 'lost case' force applied to reach 1.5-2 inch invasive catheters. | Compare SCV-CPR, HIP-CPR and Abdominal-binding CPR. | • States that force of compression is the largest determinant in the resulting aortic pressure, and that some improvement was seen with high-impulse CPR in studies in humans (for short periods). A systolic gradient of 49 ± 30 (16-118 mm Hg) was noted between the right atrium and the inferior vena cava (after passing the diaphragm). |

*In some papers, "high-impulse" CPR may mean high frequency and/or short duty cycle (30% as opposed to 50% compression-relaxation ratio).
Manikins, as the functional simulation for training purposes as well as a pseudo-model for humans, are pulled into the discussion about force and depth in CPR. As early as 1966, the ad hoc Committee on CPR suggested 80–120 pounds (36-55 kg) to compress 1.5–2 inches. The source of this instruction is unclear. Tsitlik was concerned with the elastic properties of the manikins in use during courses. He refers to the 1975 ERCI standard stating that 343–392 N (88 lbs) should produce a compression depth of 3.8–5 cm. In his convenience sample, he finds morphometric data (correlated with a mean weight of 75 kg, (range 55-109 kg in 10 patients), to fit with 19 cm (range 11-26 cm) substernal to supporting surface distance. He concludes that manikins are too stiff and act in a linear fashion, while the human chest is second or third order, non-linear. The compression force was later (1980) adapted by the ERCI to at least 294 N for a sternal depression of 4–5 cm. Clearly, recognizing that the current situation was unsatisfactory, Bankman et al. set out to model the mechanics of the chest wall based on the characteristics of a healthy human. The group modeled the chest including the internal organs based on elasticity (a spring), damping (a viscous fluid) and mass (by weight) using a system involving a module placed between the hands of the rescuer and the sternum which was attached to a force and acceleration sensing arm and described in the same journal. This group also suggests that the available models are strongly under engineered, as compression and relaxation have totally different characteristics.

Pinchak et al. investigated the force and chest wall acceleration with manual and mechanical CPR using a force measurement plate and an accelerometer. While their interest was the capability of rescuers to perform, they also introduced the DHO-CPR: down-hold-off version, and showed that Thumper CPR generated 5.2 G in downward acceleration at least 350% more than manual CPR.

Duty cycle:

**Table 14: Duty cycle in the fifth era**

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1977</td>
<td>Taylor</td>
<td>8 human patients.</td>
<td>Effect of CPM and duty cycle on flow in carotid artery (Doppler).</td>
<td>- Duty cycles of 30-60 were studied. Longer cycles improved flow and (less) pressure (&gt; 150% of control). - Used mechanical compressions, 4-6 cm impression depth, and 40% duty cycle. Instrumented with unidirectional flow probes to compare control with experimental settings during actual resuscitations. These observations were made within 30 minutes of start of CPR. - Notes that the hemodynamics of actual flow during CPR is different than under physiological conditions, with flow improving during the last phase of compression without a concomitant rise in blood pressure, while under normal conditions peak flow is during the upstroke of pressure and falls thereafter. - Suggest cycle length more important than frequency.</td>
</tr>
</tbody>
</table>
of the 100 patients none survived to discharge, while 24% had ROSC.

Findings include that no patient with a CPP of less that 15 mm Hg develops ROSC. Paradis143, working in 100 humans evaluated the correlation of CPP with ROSC in a carefully detailed study. Findings include that no patient with a CPP of less than 15 mm Hg develops ROSC, but a higher CPP is not a guarantee for survival (i.e., only a negative predictor). Note that of the 100 patients none survived to discharge, while 24% had ROSC.

Adjuvant techniques:
The fifth era did not differ for the earlier ones in that the moderately successful technique involving ‘only two hands’ seemed to need assist devices. This era was particularly fruitful in its developments. With the coordination of ventilation and compressions (Wilder, 1963)82, started a methodology which was continued in the fifth era. Table 15: Adjuvant techniques reported in the fifth era

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1977</td>
<td>Chandra144</td>
<td>7 dogs (no available weight).</td>
<td>SCV-CPR.</td>
<td>Increased airway pressures 70–110 cm H2O versus 40 cm H2O. Shows 100% increase in carotid flow, even at low (20 minutes') frequency.</td>
</tr>
<tr>
<td>1978</td>
<td>Bilfield96</td>
<td>Manikin.</td>
<td>Leg-heel CPR.</td>
<td>Found that light rescuers were more successful in giving compressions with leg-heel than with arm-hand.</td>
</tr>
<tr>
<td>1978</td>
<td>Taylor141</td>
<td>Patients, 50, convenience sample of a larger</td>
<td>To directly compare mechanical and</td>
<td>Patients after 10 minutes of manual CPR randomized to thumper (7 cm, 100 msec 60 CPM, 50% cycle time).</td>
</tr>
</tbody>
</table>

Outcome parameters:
Paradis143, working in 100 humans evaluated the correlation of CPP with ROSC in a carefully detailed study. Findings include that no patient with a CPP of less than 15 mm Hg develops ROSC, but a higher CPP is not a guarantee for survival (i.e., only a negative predictor). Note that of the 100 patients none survived to discharge, while 24% had ROSC.

### Table 15: Adjuvant techniques reported in the fifth era

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1977</td>
<td>Chandra144</td>
<td>7 dogs (no available weight).</td>
<td>SCV-CPR.</td>
<td>Increased airway pressures 70–110 cm H2O versus 40 cm H2O. Shows 100% increase in carotid flow, even at low (20 minutes') frequency.</td>
</tr>
<tr>
<td>1978</td>
<td>Bilfield96</td>
<td>Manikin.</td>
<td>Leg-heel CPR.</td>
<td>Found that light rescuers were more successful in giving compressions with leg-heel than with arm-hand.</td>
</tr>
<tr>
<td>1978</td>
<td>Taylor141</td>
<td>Patients, 50, convenience sample of a larger</td>
<td>To directly compare mechanical and</td>
<td>Patients after 10 minutes of manual CPR randomized to thumper (7 cm, 100 msec 60 CPM, 50% cycle time).</td>
</tr>
</tbody>
</table>

---

66 Chapter 3: A historical, mechanistic approach to chest compressions: Facts or polite fiction
developments. With the coordination of ventilation and compressions (Wilder, 1963), started a
detailed study. Findings include that no patient with a CPP of less than 15 mm Hg develops
Outcome parameters:

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1978</td>
<td>Taylor</td>
<td>121 Patients</td>
<td>50, Leg-heel CPR.</td>
<td></td>
</tr>
<tr>
<td>1978</td>
<td>Bilfield</td>
<td>96 Manikin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1984</td>
<td>Maier</td>
<td>123 24 chronically instrumented dogs (6-12 kg), dye, CO splinting - Morphine model,</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1981</td>
<td>Fitzgerald</td>
<td>122 20 selected dogs (20 minutes-1) frequency.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1980</td>
<td>Babbs</td>
<td>8 selected dogs (6-12 kg)# in cradle using a pentobarbital model with dye indicator.</td>
<td>Additive effect of interposed airway pressure to flow.</td>
<td>Investigated interposed high-pressure ventilation in CCCR and knew of the HAP-CPR work. Pressure ranged from 10–50 cm H2O were used and consistently generated 2 ml/kg. Alkalosis was achieved, but the trial period was very short. He suggests that interposed high-pressure ventilation may be useful.</td>
</tr>
<tr>
<td>1979</td>
<td>Chandra</td>
<td>7 dogs measures carotid flows.</td>
<td>Effects of SCV-CPR.</td>
<td>New CPR. Finds better output despite slowing the compressions rate to 20 CPM. Thoracic pump.</td>
</tr>
<tr>
<td>1979</td>
<td>Chandra</td>
<td>Dogs.</td>
<td>Negative airway pressure i.e.w. New CPR</td>
<td>Introduces as adjunct to the &quot;new CPR&quot;: simultaneous compression-ventilation with or without negative airway pressure (-20 to -40 cm H2O) augments carotid artery flow.</td>
</tr>
<tr>
<td>1980</td>
<td>Chandra</td>
<td>11 human patients as a convenience sample.</td>
<td>New CPR = simultaneous compression ventilation = CPR.</td>
<td>Set ventilator for synchronous ventilation compressions at 60-110 cm H2O, for alternating periods with conventional of 15-30 sec. Thumper CPR with 4.7-5 cm depth. Demonstrates that the airway pressure used has linear effect on output (radial and carotid arteries) was max 252% improvement. No carotid collapse observed.</td>
</tr>
<tr>
<td>1981</td>
<td>Chandra</td>
<td>10 human patients in convenience sample</td>
<td>Effect of abdominal binding</td>
<td>In-hospital study with mechanical CPR (6.25 cm, 60 CPM, 50%), with static (100 mm Hg) binding for brief periods during cardiac arrest (30 seconds to 4 minutes). Concludes better peak systolic and diastolic pressures. No injuries found, not geared to survival study.</td>
</tr>
<tr>
<td>1982</td>
<td>Ralston</td>
<td>10 dogs (15-26 kg), separating in small and large dog groups. Controls for dorsal-ventral ratio.</td>
<td>Dynamic abdominal binding (IAB-CPR).</td>
<td>A means to improve aortic diastolic pressure and thus CPP, with improvement of cerebral and myocardial blood flow, but requires a third caregiver. In a human study equal outcome was shown.</td>
</tr>
<tr>
<td>1984</td>
<td>Niemann</td>
<td>8 dogs, 16-24 kg Ketamine and Pentothal. Abdominal binding CPR.</td>
<td>Looks to venous valves and effects on timing of subdiaphragmatic blood flow.</td>
<td>Finds better intrathoracic pressures and better diastolic systemic pressures but lower CPP as right atrial pressure also (equally) increased.</td>
</tr>
<tr>
<td>1984</td>
<td>Maier</td>
<td>24 chronically instrumented dogs (25-35 kg). Uses lateral and supine positions as well as cradle.</td>
<td>High-impulse CPR. Cinefluoroscopic technique.</td>
<td>High rate and force with brief compressions (cardiac pump). Shows major differences between SVC and IVC.</td>
</tr>
</tbody>
</table>

p Selected: dorsal-ventral and right-left thoracic diameters of 1.6:1.0 with compliant chests.
The what and how are we pumping discussion:
As part of the further investigation of the (large) dog as a model, and in response to the basic principle of 'cardiac pump' theory, Mashiro et al. address the issue of filling of the right and left ventricles during ventricular fibrillation using echocardiography. They demonstrated in a large dog setting, that left ventricular dimensions decreased to end-systolic diameter sizes while the right ventricle diameter increased during up to two minutes after VF, this to 15 mm (i.e., 300%) of pre-VF diameters. This is supported by higher left ventricular pressures during the first 15 seconds of arrest.

Table 16: Overview of the pumping discussion in the fifth era

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
</table>
| 1980 | Niemann | Dogs (19-30 kg) Thiopental model. | Static abdominal binding (SAB-CPR). | • Of the "thoracic" pump school.  
• Investigated determinates in venous return to the right heart under CPR conditions with and without static abdominal binding.  
• Concludes. |

1980 Niemann 151 7 patients and dogs (40-60 kg).  
Replicated "cough CPR".  
• Concludes that coughing during a non-perfusing cardiac rhythm produces an artificial pressure wave with opening of the aortic valve (& mitral valve) and antegrade flow for about 0.2 sec.  
• Uses the 200-300 ml/M² blood volume in pulmonary vessels as priming volume.  
• Concludes that there are clear (negative) reasons against the cardiac and positive ones for the thoracic pump theory.

1981 Criley 152 NA.  
Review.  
• Notes that (1) pressures are equal in all cardiac chambers during compression,  
(2) non-closure of the atrio-ventricular valves,  
(3) cough CPR explains much. Suggests that right hearts flow is in CPR diastole and left heart during both CPR systole and diastole. Principle valve is the pulmonary valve!  
• Concludes that there is only support for the thoracic pump.

Investigate timing of flow in SCV-CPR and static bound abdomen at 100 mm Hg and volume loading.  
• Using radiolabeled spheres, this elegant study looked at the arterial and venous flows.  
• Extrathoracic venous to intrathoracic flow including flow to right heart, and pulmonary vasculature primarily occurs during CPR diastole. Suggests that mitral valve must be open during CPR systole (thoracic pump).  
• Concludes that SCV-CPR should not, as yet, be used clinically.

1982 Yin 154 15 dogs (20-45 kg) in thiopental model.  
Evaluation of the carotid artery (staying open) during CPR.  
• Evaluates the carotid artery to stay open. Citing Rudikoff 21 and of the "thoracic" pump school, found that high intrathoracic pressures did not occlude the carotid artery. Addresses concerns by Babbs 34 about collapse.

1986 Paradis 155 22 humans.  
• Investigates pressure gradients from a "thoracic" pump perspective. With catheters at the JVB, RA and aorta with epinephrine, sodium bicarbonate and mechanical CPR
As part of the further investigation of the (large) dog as a model, and in response to the basic what and how are we pumping discussion:

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1986</td>
<td>Parakos</td>
<td>NA. Review</td>
<td>(60 CPM, 50% C/R ratio, 1:5, 2 inch compression (80-100 lbs pressure). He finds no correlation between Ao-Ra and JVB to Ra pressures. • Supports ‘Thoracic’ pump model.</td>
<td>• Proclaims the “thoracic” pump as the mechanism for cardiac output in CCCR.</td>
</tr>
<tr>
<td>1990</td>
<td>Chandra</td>
<td>Humans.</td>
<td>Interested in intra- and extrathoracic hemodynamics.</td>
<td>• Points out that (1) the pump mechanism is not clear, and (2) that dogs may or may not be a good model for humans (cites Barsan). Concludes that: • If thoracic pump model correct then lateral pleural pressure, intrathoracic arterial and venous pressure should be equal to each other and to the extrathoracic arterial pressure, all of which are higher than the extrathoracic venous pressure (cites Rudikoff, and Niemann). • If cardiac pump then lateral pleural pressure, intrathoracic arterial and venous pressures should be dissimilar while intrathoracic arterial and extrathoracic arterial are similar. (cites Babbs, Chandra, and Maier). • Functional venous valves exist (3–4 cm below the clavicula).</td>
</tr>
</tbody>
</table>

Table 17: Compression frequency in the sixth era

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1992</td>
<td>Kern</td>
<td>25 patients convenience sample in Emergency Department.</td>
<td>Rate effects of CPR using ETCO2 as measure of effect of CPR.</td>
<td>• Concerned about the efficacy of ongoing efforts of CPR. • Notes that while no evidence in humans of better outcome with higher CPM, the consensus felt that this</td>
</tr>
</tbody>
</table>

Brief summary of the fifth era

"Standard" rates in 1980 was 60 CPM for two-caregiver CPR and 80 CPM (1 caregiver) for adults as "regular, smooth and uninterrupted" compressions. Finally, in 1986, the Standards and Guidelines suggested an increase of rate to a minimum of 80 and to 100 CPM if possible. This seems supported by cardiac-pump related data.

3.7 The sixth era: 1991–2004 (formalization and evidence based)

The beginning of the sixth era coincides with the advent of evidence-based medicine (EBM). One of the first aspects of cardiopulmonary resuscitation addressed in the style of EBM was adrenaline, a bastion in the treatment of cardiac arrest and seen as a principle supporter of the effect of chest compressions. This first review by Rainer and Robertson suggested that a "where do we go from here" discussion was strongly needed, and suggested that human studies in controlled situations should become leading. It will be interesting to see if this advice gained support.

Cohen et al. proponents of the thoracic pump theory, moved to active compression-decompression following an anecdotal account of a resuscitation with a plumbers helper being used to convey both impression as active relaxation of the thoracic wall. The interest in this technique developed into a continuation of the pump theory discussion based on adjuncts well into the 90's.
Little new investigation of rate was performed in this period, as emphasis shifts to what the human caregiver can actually perform and more interest in feedback and monitoring of what is actually being performed.

Compression depth:

Table 18: Compression depth and force in the sixth era

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
</table>
| 1991         | Kikuchi[197]         | 3 Excised canine chest cavity. | Investigate elastance and hysteresis.          | • Performed within 3.25 hrs of death but note effects of rigor mortis, with force being applied via a screw in the sternum.  
• Notes that hysteresis similar in humans and dogs, and more or less constant at < 2 Hz.  
• No data given as to forces and displacements in actual units. |
| 1992         | Standards and Guidelines 1992[162] | NA.          |                                                 | • Recommends 80-120 CPM as well as 100 CPM for children, based on Halperin et al. [124] |
| 1993         | Gruben[188]          | 16 Human patients in convenience sample in Emergency Department. | Evaluated force-depth displacement.           | • Cohort failing to respond to pre-hospital and in-hospital resuscitation.  
• Concludes that 245-800 N needed while 5 cm of actual compression not reached.  
• Describes the cumulative effect of the chest during compressions as a fourth order polynomial. Manikins' poor representation. Agrees on this with  

---

6 See Chapter 2 for explanations of levels of evidence

70 Chapter 3: A historical, mechanistic approach to chest compressions: Facts or polite fiction
During this era, as with compression frequency, little new work was done in compression depth.

**Duty cycle:**

**Table 19: Duty cycles in the sixth era**

<table>
<thead>
<tr>
<th>Year</th>
<th>Author Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1990</td>
<td>Babbs 163</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>1992</td>
<td>Standards and Guidelines 1992 162</td>
<td>NA</td>
<td>• Recommends 80-120 CPM as well as 100 CPM for children, based on Halperin et al. (1986). 124</td>
</tr>
</tbody>
</table>

**Brief summary of the sixth era**

The sixth era, working towards the Standards and Guidelines 2005, directed itself towards outcome and the first quality impulses, even more strongly developed in the post-2005 period.

### 3.8 The seventh era: 2005 – (the return to core task)

In 2005 the ILCOR Standards and Guidelines broke with tradition by looking both towards what a (lay) caregiver could perform and re-evaluating which core aspects in CPR had become suppressed by other evidence or non-evidence-based activities. The Guidelines set out to refocus attention on the chain of survival and essential activities in it. Where the Guidelines 2000 had focused on the breathing, the Guidelines 2005 focused on the circulation. No studies had compression frequency as their endpoint, compression depth was only indirectly an endpoint as an increasing number of studies questioned whether 4-5 cm was actually being performed by caregivers. 160

**Compression depth:**

**Table 20: Compression depth and force in the 7th era 2005 - present**

<table>
<thead>
<tr>
<th>Year</th>
<th>Author Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>2007</td>
<td>Odegaard et al 170</td>
<td>Humans patients. Feedback/depth.</td>
<td>• Caregivers are not willing to press as hard as feedback suggests.</td>
</tr>
<tr>
<td>2007</td>
<td>Thomlinson et al 171</td>
<td>Observational in humans. Force-depth relationship (accelerometer).</td>
<td>• Note that 50 kg covers &gt; 90 of cases at 38 mm depth. Strong tendency for shallow compressions. Wide spread in force seen.</td>
</tr>
<tr>
<td>2009</td>
<td>Neurter et al 172</td>
<td>Humans and swine. Validation of animal model.</td>
<td>• Within parts of force swine and human chests are comparable, as well as period of CPR.</td>
</tr>
</tbody>
</table>

Interest continued to develop for compression-only CPR.

Feedback, electronic 173 or mechanical, as well as stand-alone versus incorporated in monitor defibrillators became a major focus during the post 2005 period.
3.9 Adjuvant techniques to generate flow in basic life support

As early as 1962, investigators started to look at "adjuvants" to standard CCCR, based on their insight into poor pressures and (surprisingly) poor outcomes. A number of schools of optimization were developed. These are among further variations (1) the use of airway pressure, (with and without abdominal binding), (2) the use of only abdominal binding, (continuous or intermittent, during either CPR systole or diastole), (3) the active compression-decompression (venous return), (4) high impulse groups and (5) vest or cough CPR. Naturally, combinations of different techniques were also investigated and will be discussed in each section, as needed. Developments in each will be discussed in turn.

Different mechanisms may contribute or detract from the effectiveness of CCCR. Anatomical structures, such as lung tissue or the diaphragm (fixing the heart, changing the intrathoracic volume or becoming a functional valve in the inferior vena cava) may be non-consistent factors in experiments. The following "adjuvant" techniques look into this.

3.9.1 The use of airway pressure:

Airway pressure has been a well-known and easily accessible method of increasing intrathoracic pressure.

Table 21: Airway pressure as effector on cardiac output

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1963</td>
<td>Wilder</td>
<td>45 dogs (10-15 kg)</td>
<td>HAP-CPR.</td>
<td>• Looked at simultaneous, independent, alternating, no ventilation and constant pressure ventilation in relationship to standard compressions. He finds that simultaneous insufflation and compression generates the best femoral artery pressures (in investigations of 15 minutes length).</td>
</tr>
<tr>
<td>1977</td>
<td>Chandra</td>
<td>7 dogs (20-40 kg); in Pentobarbital model</td>
<td>HAP-CPR.</td>
<td>• Proposed simultaneous airway insufflation (HAP-CPR ≅ NEW CPR) with chest compressions to augment output. He suggests a slower rate (40 CPM) with a 0.6 C/R ratio, and simultaneous ventilation with pressures of 60-110 cm H_2O in a human model after failure of classic CCCR. He finds increased carotid artery flow (25%), and increased radial artery pressure (mean) pressure 35.5 – 45.9 mm Hg) without impairment of oxygenation or ventilation (in 383).</td>
</tr>
<tr>
<td>1980</td>
<td>Chandra</td>
<td>4 dogs</td>
<td>SVC-CPR</td>
<td>• Investigated simultaneous compression-ventilations (frequency 40 CPM, insufflation to 60-100 mm Hg during 0.9 sec and bound abdomen. The studies were of short duration with samples take at 30 seconds after initiation. Measurements were taken using a cannulating electromagnetic sine wave flow probe at 6-8 cm above the thoracic inlet, (2) negative diastolic...</td>
</tr>
</tbody>
</table>

72 Chapter 3: A historical, mechanistic approach to chest compressions: Facts or polite fiction
3.9.1 The use of airway pressure:

Airway pressure has been a well-known and easily accessible method of increasing intrathoracic volume or becoming a functional valve in the inferior vena cava) may be non-consistent factors in structures, such as lung tissue or the diaphragm (fixing the heart, changing the intrathoracic thoracic inlet, (2) negative diastolic wave flow probe at 6-8 cm above the cannulating electromagnetic sine model. Measurements were taken using a phase of the left ventricle, which is between 10–25 mm Hg (cites Downey). Very positive about SCV-CPR.

### Table 21

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1981</td>
<td>Chandra</td>
<td>146</td>
<td>Investigated simultaneous compression and ventilation with high airway pressure (SVC-CPR ≅ NEW CPR), working in 4 dog models to evaluate whether the size of the dog and the area of compression (in example large dogs may have a dorso-ventral size larger than humans) may influence the mechanism in blood flow. Some improvement is found. Suppositions on dog size (small for pediatric) medium for human in found here.</td>
<td></td>
</tr>
<tr>
<td>1982</td>
<td>Babbs</td>
<td>34</td>
<td>SVC-CPR ≅ NEW CPR.</td>
<td>• Investigated simultaneous compression and ventilation with high airway pressure (SVC-CPR ≅ NEW CPR), working in 4 dog models to evaluate whether the size of the dog and the area of compression (in example large dogs may have a dorso-ventral size larger than humans) may influence the mechanism in blood flow. Some improvement is found. Suppositions on dog size (small for pediatric) medium for human in found here.</td>
</tr>
<tr>
<td>1986</td>
<td>Martin</td>
<td>175</td>
<td>SVC-CPR.</td>
<td>• After a period of about 16 minutes of BLS. • Investigated one-minute trials of SVC-CPR found a decrease in the effect of intrathoracic pressure with strapping on mitral valve closure. • Difficulty understanding model with respect to intrapulmonary pressure. • citing Rudikoff21</td>
</tr>
<tr>
<td>1988</td>
<td>Swenson</td>
<td>176</td>
<td>SRC-CPR.</td>
<td>• Investigated conventional as opposed to SRC-CPR (among others). In this investigation a vest (250 mm Hg) was used for compression and improved.</td>
</tr>
<tr>
<td>1988</td>
<td>Halperin</td>
<td>176</td>
<td>SRC-CPR.</td>
<td>• Investigated conventional as opposed to SRC-CPR (among others). In this investigation a vest (250 mm Hg) was used for compression and improved.</td>
</tr>
<tr>
<td>1989</td>
<td>Krischer</td>
<td>177</td>
<td>SVC-CPR.</td>
<td>• Prehospital, prospective study evaluated conventional CPR with SVC-CPR (rate of 40 CPM, P aw of 80 mm Hg) with abdominal binders (50 mm Hg). 26.2% survival in the conventional and 19% in the experimental group (initial) and 10.6 versus 5.9 in long term survival. • The suggestion is made that the slow rate may be a factor in the decreased survival. • No invasive data was available.</td>
</tr>
</tbody>
</table>

Chapter 3: A historical, mechanistic approach to chest compressions: Facts or polite fiction 73
The use of abdominal binding:
The abdominal cavity has been the source of interest in resuscitation since the introduction of closed-chest techniques. Since the diaphragm is lax, abdominal content can (a) impact upon the intrathoracic volume (decrease it), (b) act as a flexible inferior wall to the thoracic cavity causing loss of pressure, (c) be used as a systemic vascular resister, and (d) act as a reservoir of recruitable volume.

Table 22: Effects of Abdominal binding

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1967</td>
<td>Harris</td>
<td>Humans and dogs.</td>
<td>Use (static) abdominal binding to augment CPR.</td>
<td>• Ventilation–compression ratio (3:15 good but 6:30 not good).</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• Interposed versus simultaneous inflation (not better).</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• Increased CPM (48 to 120 CPM with linear improvement in carotid flow) basis for the 2:15 and 1:5 ratios.</td>
</tr>
<tr>
<td>1971</td>
<td>Redding</td>
<td></td>
<td></td>
<td>• Described static abdominal binding, a mechanism intended to increase intrathoracic pressure and redistribute blood flow.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• However venous pressure are also (severely) increased, with negative effect of CPP, increased ICP and diminished cerebral oxygen content.</td>
</tr>
<tr>
<td>1982</td>
<td>Ralston</td>
<td>Human. IAC-CPR.</td>
<td>Saw improvement in dynamic abdominal binding (IAC-CPR) as a means to improve aortic diastolic pressure and thus CPP, with improvement of cerebral and myocardial blood flow, but requires a third caregiver.</td>
<td>• In a human study equal outcome was shown.</td>
</tr>
</tbody>
</table>

Table 23: Active compression-decompression:

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1993</td>
<td>Tucker</td>
<td>ACD-CPR.</td>
<td>Investigated the transmitral flow and end-CPR-diastolic volume of the left ventricle with and without ACD-CPR.</td>
<td>Findings demonstrated improved end-diastolic volume, increased stroke volume.</td>
</tr>
</tbody>
</table>

Table 24: High impulse CPR:

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1988</td>
<td>Swenson</td>
<td></td>
<td></td>
<td>Investigating conventional as opposed to high-impulse CPR (among others) at 120 CPM and with a short (0.33 duty cycle).</td>
</tr>
</tbody>
</table>
3.9.3 Vest or cough CPR:

Table 25: The effects of vest CPR

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1976</td>
<td>Criley</td>
<td>8 humans.</td>
<td>• Under coronary angiography had circulatory collapse (VF), of which three remained conscious for up to 39 seconds by coughing. This coughing had been suggested by Stone to alleviate bradycardia and hypotension associated with contrast studies. • States that competent ao-valve and intact peripheral resistance maintains a higher pressure in the aorta.</td>
<td></td>
</tr>
<tr>
<td>1980</td>
<td>Criley</td>
<td>7 humans and dogs (40-60 kg) in a Ketamine-model.</td>
<td>• There is an arterial pulse. • The aortic valve opens in during CPR systole • Generates cerebral circulation. • In addition working dogs finds that cough can generate 100-180 mm Hg against a closed glottis (cites id-838), with negative pressures of −10 to −20 mm Hg at inspiration. • The drawing of the conduits has changed V.A.V. 152, 1981</td>
<td></td>
</tr>
<tr>
<td>1985</td>
<td>Niemann</td>
<td>Cough-CPR.</td>
<td>• Validating a mechanical device to simulate cough-CPR.</td>
<td></td>
</tr>
<tr>
<td>1986</td>
<td>Criley</td>
<td></td>
<td>• Using aortograms. • Demonstrated aortic regurgitation with conventional CCCR but none with SCV-CPR.</td>
<td></td>
</tr>
<tr>
<td>1986</td>
<td>Halperin</td>
<td>29 dogs (21-30 kg) in Pentobarbital model.</td>
<td>• First states that vital organ blood flow is related to peak sternal force. He investigated dogs, with microsphere injection, invasive ICP, inflating vest to 380 mm Hg, with intermittent ventilation. • Stated that vest CPR is optimal at 200-280 mm Hg, with no trauma and with near normal flows.</td>
<td></td>
</tr>
<tr>
<td>1988</td>
<td>Swenson</td>
<td></td>
<td>• Investigating conventional as opposed to vest CPR (among others) at 200 – 250 – 300 mm Hg (5-1 series, 72 CPM, for 2 minutes). • Found improved aortic peak pressure and diastolic pressures similar to conventional CPR. • Increasing vest pressure to 300 mm Hg further increased peak pressure by no rise in aortic diastolic pressure. • With a short (0.33 duty cycle), found improved aortic pressure and increased CPP. • When associated with simultaneous ventilation right atrial pressure increased markedly (decreasing CPP) due to air trapping.</td>
<td></td>
</tr>
<tr>
<td>1989</td>
<td>Ben-Haim</td>
<td>Dogs (16-25 kg) in a Pentobarbital model.</td>
<td>• States that aortic diastolic and CPP pressures are reliable indicators of survival and says that cardiac output</td>
<td></td>
</tr>
</tbody>
</table>
Initially introduced for the treatment of pulmonary hypertension, horizontal, whole body CPR: use is reserved for special cases, and is not generally taught. It has recently received more than anecdotal attention, including a review article and has been described as didactically useful to reduce direct trauma due to the stiffness of the posterior thoracic wall supporting the thoracic pump theory for circulation, causes little tiring, and to avoid mouth-to-mouth ventilation hinderers. It has been documented in a series of case reports, as well as a six-patient (lost case) study with its end point mean systolic blood pressure, as well as occurring in operating room settings. It has not been actively advocated. The study showed improved blood pressure (with the exception of diastolic pressures). This technique has continued to be developed and shows promise as it causes less post-resuscitation cardiac dysfunction. Its use is reserved for special cases, and is not generally taught.

3.9.4 Prone CPR:
Prone CPR is a reintroduction of a classic technique. When resuscitation medicine was initially introduced it was applied to persons apparently drowned. The position chosen was face down (effectively a practical approach to opening the airway).

It has recently received more than anecdotal attention, including a review article and has been described as didactically useful to reduce direct trauma due to the stiffness of the posterior thoracic wall supporting the thoracic pump theory for circulation, causes little tiring, and to avoid mouth-to-mouth ventilation hinderers. It has been documented in a series of case reports, as well as a six-patient (lost case) study with its end point mean systolic blood pressure, as well as occurring in operating room settings. It has not been actively advocated. The study showed improved blood pressure (with the exception of diastolic pressures). This technique has continued to be developed and shows promise as it causes less post-resuscitation cardiac dysfunction. Its use is reserved for special cases, and is not generally taught.

3.9.5 pGz CPR:
Initially introduced for the treatment of pulmonary hypertension, periodic acceleration, as an adaptation of cardioballistography, brings force to bear on the cardiovascular system, and seems to have a direct effect on the vascular resistance. Mediator release (i.e., NO) seems to be a major effector. In an experiment using healthy animals (swine, ± 12 kg intact circulations, with microspheres as flow indicator) showed increases in flow of > 70% to the epicardium and >175% to the central nervous system. This has been generalized to intact circulations, and has shown generally better resuscitation and post resuscitation outcomes as well as biophysical (modeling) conditions than classic closed-chest compressions at 2 Hz, 0.6 Gz. This promising technique will remain experimental or limited in its use to selected institutions due to the technical demands intrinsic to its use.

3.9.6 Studies of different "adjuvants" techniques in one model:

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Model</th>
<th>Purpose</th>
<th>Results / Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1988</td>
<td>Newton</td>
<td>Dogs (22-35 kg) in a chronic instrumentation, morphine-crade model with fluid loading.</td>
<td>Compared five techniques of CCCR.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>HIC-CPR at 150 CPM, 1.5-2 cm/20 mm Hg, SVC-CPR, SCV + SAC-CPR, SC-DAC-CPR, and VEST-CPR.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>The difference between peak left ventricular pressure and pleural pressure defines &quot;cardiac&quot; vs &quot;thoracic&quot; pump model (i.e., HIC-CPR vs VEST).</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Blood flow is calculated.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Suggest HIC best.</td>
</tr>
</tbody>
</table>

Table 26: Multiple tasking
3.9.7 Conclusions in technique in BLS:
The technique of basic life support and perhaps more so chest compressions has been compression rate. A great deal of work has been done investigating the rate of compression which (under CCCR conditions) might improve outcome. These investigations, however, were limited by (a) technical opportunities, (b) the discussion of "total" mechanisms in cardiac versus thoracic pump models, and (c) by the use of animal models and (large) incremental changes during relatively short periods of time. Evidence is available to support a slow rate (i.e., 60 CPM) generates limited output, but a fast rate (i.e., 150 CPM) generates little improvement. This may be due to intrathoracic and cardiac refill times. Compression force may be independent of compression depth but only in mechanical modes.

3.10 Mechanical chest compressions: a resolution?
Since the 2000 Guidelines, a careful beginning has been made with the re-introduction of mechanistic aspects into mainstream resuscitation medicine. The wording supports that this is not a new phenomenon: as early as the 1960s external chest compressions were known to be difficult. However, these devices were never accepted by the resuscitation community, being heavy or clumsy, without the endorsement of guidelines, and had the potential to undermine the 'lay-based' characteristics of a popular technique. However, thinking has changed and practical recognition of a need for improvement has been recognized.

An editorial in JAMA, by Lewis & Niemann in 2006 describing manual versus device-assisted CPR stated "Most individuals who sustain cardiac arrest probably receive suboptimal CPR, especially during extended resuscitation efforts."

Two different lines of thinking have been incorporated in two different devices servicing the same purpose. These two devices, the Autopulse and the Lund University Cardiac Arrest System (LUCAS) have made their appearances in professional resuscitation and have rekindled interest with the professional caregivers to the challenges of getting compressions right. They both 'support' the patient; the underlying surface is unimportant.
The LUCAS, working with a pneumatic piston based device offers rapid compression (< 60 msec), as well as a rapid-active-decompression (< 75 msec) with a force of up to 500 N.\(^{199}\) This has led to concerns about excessive trauma,\(^{200}\) while the Autopulse is slightly more ramped. The LUCAS has active decompression potential (not in use in the USA), and a pad of about 6 cm diameter to transfer force. Multiple laboratory experiments have demonstrated increased flow in the brain\(^{198}\) as well as CPP,\(^{201}\) and selected blood flows.

The Autopulse uses a wide band to apply a patented Lifeband (air-chamber system) to distribute high forces over a large area, decreasing the instantaneous pressure and trauma. The Lifeband allows 125 kg force to be spread over board area (leading to 2 Pa local pressures), causes some limitation in lateral thoracic distension, in favor of central distribution. The Lifeband is shortened after the software has determined the force-distance relationship needed to decrease the anterior-posterior diameter of the chest by 20%, up to a maximum of 5 cm. Due to the form of the band, the importance of individual chest geometry is restricted. The pumping mechanism, while not as yet documented by valve-oriented investigation, is suggested to be both thoracic and cardiac pump (not vest, due to the guideline required depth of impression). High CPP and high flows have been documented, leading to 'pinking up' of the patient and improved survival (i.e., 499 and 284 cases respectively).\(^{202}\) Ramping speed is 100 msec for both compression and decompression. The Autopulse is also capable of timing defibrillation to end of compression as well as giving advice as to whether a counter shock would be useful.

---

\(^{1}\) A battery powered device will be available in September 2009.
The LUCAS, working with a pneumatic piston-based device offers rapid compression (<60 msec), as well as rapid-active-decompression (<75 msec) with a force of up to 500 N. This has led to concerns about excessive trauma, while the Autopulse is slightly more ramped. The LUCAS has active decompression potential (not in use in the USA), and a pad of about 6 cm diameter to transfer force. Multiple laboratory experiments have demonstrated increased flow in the brain as well as CPP, and selected blood flows.

Figure 5: Interest of the Compression-relaxation ratio as well as the effects of 'hold' periods has resurfaced time and again, including discussions about the viscosity in blood and its effects on circulation driven externally.

The Autopulse uses a wide band to apply a patented Lifeband (air-chamber system) to distribute high forces over a large area, decreasing the instantaneous pressure and trauma. The Lifeband allows 125 kg force to be spread over a large area (leading to 2 Pa local pressures), causing some limitation in lateral thoracic distension, in favor of central distribution. The Lifeband is shortened after the software has determined the force-distance relationship needed to decrease the anterior-posterior diameter of the chest by 20%, up to a maximum of 5 cm. Due to the form of the band, the importance of individual chest geometry is restricted. The pumping mechanism, while not yet documented by valve-oriented investigation, is suggested to be both thoracic and cardiac pump (not vest, due to the guideline required depth of impression). High CPP and high flows have been documented, leading to 'pinking up' of the patient and improved survival (i.e., 499 and 284 cases respectively). Ramping speed is 100 msec for both compression and decompression. The Autopulse is also capable of timing defibrillation to end of compression as well as giving advice as to whether a counter shock would be useful.

A battery powered device will be available in September 2009.

Figure 6: Data from the Autopulse device (Zoll), using post-hoc analysis of data from a clinical trial. The question at hand may not be whether this data is valid, but whether the primary endpoint in the evaluation of devices made to create an artificial circulation should not be exactly that: how well is the (basic) immediate artificial circulation (i.e., before ROSC) and other there other confounders impacting this endpoint.

Both devices work within the restrictions set by the Guidelines 2005, while both are able to generate more force than the 500 N suggested be required for adequate compressions. Neither device has been able to produce the definitive RCT, demonstrating important ROSC improvements, but this is expected in the near future. The returning difficulties are that (a) almost no patients will be "fresh" when mechanical CPR is started, that there is a significant (i.e. > 6 minute) delay before mechanical CPR can be started as the device must arrive on scene (once there, typically <30 seconds is needed for application), (b) there are no SMART end terms available. ROSC or hospital discharge criteria involve the complete chain of survival, while the primary and functional goal of mechanical CPR should be to generate a (good), temporary circulation. Measures for this still require development (such as ETCO2, conjunctival or nasal septal O2 measurements, etc. The suggestions that high flows can be generated by these mechanical devices also begs the question of whether oxygenation and ventilation (geared since the guidelines 2000 to a large ventilation-perfusion mismatch) must not also be readjusted.

3.11 Chest compressions: conclusions in facts and polite fiction

This Chapter has presented a selection of the experimental, animal and clinical works focusing on chest compressions. Early work is most extensive, both in sheer volume as well as in intensity. Regrettably, some of the work is suspect since small animal models, and perhaps dog models in general, translate poorly to the human chest. As the Tables show, the last decennium has produced little fundamental work within chest compressions, perhaps not surprisingly since the 4-5 cm compression depth has not been at issue. Interest has shifted to objectification of whether caregivers are actually performing guideline suggestions as objective feedback modalities become available, but has not yet reached PDR as a potential standard of care. This may be since both the possibilities of the lay community is still the leading in basic life support. Shifting the prerogative back to the medical community has not yet gained momentum certainly in part based on the lack of bio-feedback modalities.

The compression depth suggested by Kouwenhoven et al. remains, however, not in evidence. It is not empirically nor practically based in anatomical or physical fact, and no study has been performed to evaluate its effectiveness per se. Performing such a study (i.e., 3-7 cm compression...
depth in a controlled, (blinded) randomized fashion) might be challenging in and of itself. Neither has modeling work been able to address this issue. Other aspects, such as leaning (2.5 kg threshold), the optimal compression to relaxation rate, potentially combining the need for a compression hold with sufficiently slow compression to avoid extraneous trauma, and hand position, have been guided by careful thought and the abilities of the (lay) caregiver more than by studies.

As mechanical devices become accepted, the line of evidence in this Chapter will, to some large degree need to be redone, allowing the possibility that mechanical devices adhere to other parameters than those set down for manual compressions.
Chapter 3: A historical, mechanistic approach to chest compressions: Facts or polite fiction

3.12 References

2. Holy Bible, King James Version.
16. Harvey G. Exercitatio anatomica, de motu cordis et sanguinis in animalibus. 1628. Frankfurt, D.


Chapter 3: A historical, mechanistic approach to chest compressions: Facts or polite fiction


92 Anstadt GL, Rawling CA, Krahkinkel DT, Casey HW, Schiff P. Prolonged circulatory support by direct mechanical ventricular assistance for two or three days of ventricular fibrillation. Trans Amer Soc Artif Intern Organs. 1971; 17: 174-182.


118 Standards and Guidelines for cardiopulmonary resuscitation (CPR) and emergency cardiac care (ECC). JAMA 1986; Jun 6;255(21):2905-2989.
133 Miles S, and Roylance PJ. Teaching first Aid. Balliere Tindall and Casell, London GB.
137 Cardiopulmonary resuscitation: statement of the ad hoc Committee on cardiopulmonary resuscitation of the Division of Medical Sciences, National Academy of Sciences, National research Council. JAMA 1966; 198: 372-379.


Chandra NC, Rudikoff M, Tsitlik J, Weisfeldt ML. Augmentation of carotid flow during cardiopulmonary resuscitation in the dog by simultaneous compression and ventilation with high airway pressure. Am J Cardiol. 1979; 43: 422-422.


Standards and guidelines for cardiopulmonary resuscitation (CPR) and emergency cardiac care (1979 consensus), JAMA 1980; 244: 453-509.


190 Babbs CF. Biophysics of cardiopulmonary resuscitation with periodic z-axis acceleration or abdominal compression at aortic resonant frequencies. Resuscitation. 2006; 69: 455-469.


Chapter 3: A historical, mechanistic approach to chest compressions: Facts or polite fiction
Thoracic CT scans and cardiovascular models: the effect of external force in CPR


Part of this material was read as a preliminary report at the NIH “2nd International Conference on Conference of Cardiovascular Medicine and Science”, July 23-25 2003, Bethesda, MD. USA.
Abstract

The cardiac pump theory holds that cardiac compression occurs between the bony structures of the sternum and the vertebral column if the sternum is depressed 3.8 – 5.1 cm. Clinical observations have confirmed but also refuted this, as have mathematical models. Evaluation from 50 CT scans demonstrate that the human anterior-posterior internal distance at the level of the tricuspid valve (T₂) is 13.0 ± 2.3 cm, with cardiac or vascular structures accounting for only 8.6 ± 1.2 cm. In earlier literature the internal distance has been estimated to be between 8 and 19 cm. In earlier manuscripts force was used as the independent variable in the models.

A potential explanation for the inconsistent observations could be that sternal depression has been variably applied in animal, human and mathematical models. Review of the literature demonstrates that a functional target depth has never been determined in relationship to thoracic and organ volumes. Forces applied vary from 245 to 1831 N (55 to 130 lbs), with chest compliance varying from 0.1 x 10⁻² to 2.9 x 10⁻² cm N⁻¹.

Principally, two groups have used mathematical models to investigate pump mechanisms. These models have used parameters not in evidence. This study suggests that, while models are uniquely suitable for investigating pump mechanisms, fundamental aspects as proposed by Kouwenhoven must be included.
Abstract

The cardiac pump theory holds that cardiac compression occurs between the bony structures of the sternum and the vertebral column if the sternum is depressed 3.8 – 5.1 cm. Clinical observations have confirmed but also refuted this, as have mathematical models. Evaluation from 50 CT scans demonstrate that the human anterior-posterior internal distance at the level of the tricuspid valve (Ti2) is 13.0 ± 2.3 cm, with cardiac or vascular structures accounting for only 8.6 ± 1.2 cm. In earlier literature the internal distance has been estimated to be between 8 and 19 cm. In earlier manuscripts force was used as the independent variable in the models.

A potential explanation for the inconsistent observations could be that sternal depression has been variably applied in animal, human and mathematical models. Review of the literature demonstrates that a functional target depth has never been determined in relationship to thoracic and organ volumes. Forces applied vary from 245 to 1831 N (55 to 130 lbs), with chest compliance varying from 0.1 x 10^-2 to 2.9 x 10^-2 cm N^-1.

Principally, two groups have used mathematical models to investigate pump mechanisms. These models have used parameters not in evidence. This study suggests that, while models are uniquely suitable for investigating pump mechanisms, fundamental aspects as proposed by Kouwenhoven must be included.
4.1 Introduction

In the 1960’s closed chest cardiac resuscitation (CCCR) was made popular by Kouwenhoven et al. as a by-product of clinical research involving external cardiac defibrillation. This team suggested that a cardiac pump mechanism - in analogy to open chest cardiac resuscitation (OCCR) - produced circulation by compressing the heart between sternum and vertebral column. This theory, which became known as the “cardiac pump theory” of cardiopulmonary resuscitation (CPR), has been extensively investigated, but has never been adequately confirmed, modified or rejected. The depth for chest compression, suggested by Kouwenhoven et al. as one-and-a-half to two inches, or in metric terms, 3.8 to 5.1 cm, has remained fundamentally unchanged and is (strenuously) taught to professionals and laymen. In older standards and guidelines (e.g., 1974) the careful reader will encounter suggestions that this impression range is suitable for the “typical” patient and that clinical adaptation may be required, without suggesting an algorithm to apply this advice. European perception (2000) currently advocates 4 to 5 cm impression depth, from a teaching perspective. While there are suggestions that a threshold impression depth must exist before effective cardiac compression is achieved, surprisingly little work has been done about verifying whether impression depth may or may not be a factor in the poor outcome after sudden cardiac death, and whether it may be a variable contributing to an understanding of which patients might survive. Notably, this discussion has been held for models and animal experiments, however, doubts about assumptions in building models have led to the need for validation.

This study reviews information and work done on impression depth and the contribution models have made to insight. Of principal interest would be two aspects which have impact on impression depth: first, if the current recommendations do in fact directly cause cardiac compression (i.e., when the Kouwenhoven recommendation is actually followed); and second, is there consistent use of parameters and variables in the models which are supported by evidence. Both these may impact on impression depth in the clinical setting.

4.2 Materials and Methods

Following permission from the Standing Committee for Medical-Ethics, a retrospective review was performed on a set of 50 spiral Computer Axial Tomography scans (CT scan) (Mx8000 Philips Medical Systems, Eindhoven, NL) of the thoracic cavity, selected at random from those performed in the calendar year. In order to be used in this report the CT scan had to have been recorded using the standard hospital protocol (Table 1), under non-emergency conditions. Patients mechanically ventilated were allowed to participate, as the protocol requires that ventilation be suspended during the CT scanning procedure.

Table 1: Description of the thoracic scan protocol used in all patients.

<table>
<thead>
<tr>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fasting patient (T &gt; 4 hours, if possible)</td>
</tr>
<tr>
<td>Breathing stopped for the duration of the scan</td>
</tr>
<tr>
<td>Slice size every 5 millimeters, from thoracic opening into the liver</td>
</tr>
<tr>
<td>Speed: 34 slices/sec.</td>
</tr>
</tbody>
</table>

Note: Non-ECG triggered scan

Demographic data were acquired from the medical records, as was indication for the CT scan, specifications of any underlying disease, as well as general health and weight status. In cases in which this was unclear, patients were approached by telephone, and the missing data acquired. All scans were evaluated by the same study team, using calibrated measurements accurate to 1 mm, and corrected for each individual scan. Lung and soft tissue settings were used to optimize measurement and identification.
Measurement locations were standardized following a pilot study of ten patients to determine the length of the sternum covered by a massaging hand (circa 9 cm) and how this could be precisely projected on structures which could be identified on the CT scans. As cranial reference (level 1), the lowest border of the pulmonary arch and as midpoint (level 2) the lower border of the tricuspid valve were used. During the pilot study the lower hand edge (level 3, just above the junction of the xyphoid process with the sternal corpus) was shown consistently to have no underlying cardiac structures (the liver) and was not scored further.

Six different distances were noted in the two remaining transverse planes. T_{e1} and T_{e2} represent the shortest distances from the outside (skin) border of the sternum to the outside of the dorsal thorax (cm); T_{i1} and T_{i2} represent the transverse distances from the internal border of the sternum to the most ventral aspect of the curved, underlying, vertebral body (cm). Each structure (cardiac, vascular, esophagus) in these planes was listed, with the diameters of cardiac and vascular cavities being described as C_1 and C_2. In addition, L_2, was measured (cm), as the distance in the frontal plane between the middle of the ventral aspect of the vertebral corpus and the lateral thoracic wall. V_2, described the height of the vertebral aspect of the thoracic wall in a sagittal plane at the edge of the vertebra, with V_{1max}, describing the maximal depth in the sagittal plane (towards the lateral thoracic wall). L_1, V_1 and V_{1max} were not described as clinical significance was unlikely. These lines are illustrated in Figures 1 and 2.

Figure 1. CT scan slice of the cranial border of hand position on the sternum, with lines designating the measurements. T_{i1} and T_{i2} = the transverse distance at the lowest level of the pulmonary arch, from skin to skin and from the dorsal aspect of the sternum to the ventral most aspect of the vertebral corpus, respectively. C_1 describes the cardiac or vascular structure diameters. L_1, V_1 and V_{1max} were not described.
Figure 2: CT scan slice of the “middle” hand position on the sternum, with lines designating the measurements. T_{e1} and T_{i1} = the transverse distance at the lowest level of the pulmonary arch, from skin to skin and from the dorsal aspect of the sternum to the ventral most aspect of the vertebral corpus, respectively; C_{i} describes the cardiac or vascular structure diameters. L_{i} describes the horizontal distance at this level for the radius of the thoracic cavity; V_{i} and V_{i\text{max}} describes the vertical distance from the plan at the top of the vertebral body to the inside of the thoracic wall (i.e., ribs) and the deepest point, respectively.

Note that caudal edge of the hand position was not reviewed as no vascular structures were found there.

A literature search was performed using a Medline search working from MeSH headings Cardiopulmonary resuscitation (CPR), Heart massage (and further with chest compression, force, impression depth), Computer simulation models, theoretical models, biological. Additionally, EMBASE, BIOBASE and BIOSIS were reviewed.

All data was entered into an SPSS v11 database. Descriptive statistics were used to verify the normal distribution of the population, and data is reported as mean ± standard deviation. P < 0.05 was taken to be statistically significant.

4.3 Results

50 CT scans were reviewed of 49 different persons. All scans were reviewed by the same team. One person has two CT scans which were made separated by more than eight months. They were reviewed as two entities. During the second evaluation data from the first was not available. The double inclusion was noted when the data was entered in the central database. No patients on a mechanical ventilator were included. Table 2 shows the demographics of the individuals included. Age, with a mean of 62.5 ± 15 years falls in the peak range for relevance, as does the higher percentage of males included. Weight, with a body mass index of 26 ± 3 for males, and 26 ± 5 for females, reflects the increasing incidence over overweight in the European population.

The distance from skin-to-skin at the level of T_{e1} (i.e., the upper border of hand position on the sternum) was found to be 23.1 ± 3.1 cm, with the distance from the inside border of the sternum to the ventral aspect of the underlying vertebral body 12.6 ± 2.4 cm (T_{i1}). In this transverse plane the following structures were identified: the (aortic and pulmonary) outflow tracts in all patients, in 80% of cases central veins, in 35% of cases the descending thoracic aorta, and incidentally the esophagus. The C_{i}, L_{i}, and V_{i} measurements showed no relevance and are not reported here (Figure 3).
Table 2: Demographics of the included population. Note that 49 individuals were included, with 50 CT scans being reviewed.

<table>
<thead>
<tr>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Included</td>
</tr>
<tr>
<td>49 individuals</td>
</tr>
<tr>
<td>50 CT scans</td>
</tr>
<tr>
<td>Gender</td>
</tr>
<tr>
<td>40% female</td>
</tr>
<tr>
<td>Age (years)</td>
</tr>
<tr>
<td>26 – 80 (median 67)</td>
</tr>
<tr>
<td>(62.5 ± 15)</td>
</tr>
<tr>
<td>Weight (kg)</td>
</tr>
<tr>
<td>52 – 105 (median 78)</td>
</tr>
<tr>
<td>(77.6 ± 12)</td>
</tr>
<tr>
<td>Height (cm)</td>
</tr>
<tr>
<td>156 – 188 (median 173)</td>
</tr>
<tr>
<td>(172 ± 7.4)</td>
</tr>
</tbody>
</table>

Figure 3: Outcome of CT scans. $T_{e1}$, $T_{i1}$ and $T_{e2}$, $T_{i2}$ and $C_2$ presented as mean ± SD, N = 50. For explanation of abbreviations see text and figures 1 and 2. Note the minor increase in chest depth at $T_{e2}$, while this is the area with the underlying cardiac structures.

At the $T_{e2}$ level the skin-to-skin distance increased to 24.1 ± 4 cm (range 17-29 cm), reflecting the conical aspect of the thoracic cavity. The internal distance was 13.4 ± 2.3 cm, with a difference of 11.1 ±1.2 cm between outside and inside diameters. The structures found in the plane between the sternum and the vertebral body were predominately the right atrium, the esophagus, within a majority of cases (part of) the right ventricle, and in some cases the left atrium with an edge of the left ventricle. The descending thoracic aorta lay, in most cases, lateral of the vertebral column. $V_2$ was found to be 5.8 cm on average, with the cardiac structures in this line being 8.6 ± 1.6 cm. The $L_2$ was found to be 10.4 ± 2.5 cm. The ‘non cardiac’ distance available was 4.6 ± 1.6 cm with a range of 1.8 – 7.3 cm). Figures 3 and 4 represent the principal outcomes, relevant for this study.

Figure 4: “Non cardiac” distances between sternum and vertebral column. At $T_1$, while none of the four cardiac chambers were involved, the outflow tracts, etc, were used for reference purposes. The $T_2$
non-cardiac distance of 4.6 cm is in the upper range of impression depth currently taught for clinical CPR purposes.

4.4 Discussion

Common teaching on CPR, in particular to laymen, holds that the heart (i.e., the left ventricle) is compressed between the sternum and the vertebral column, and that this is why the force to displace the sternum must be directed ‘straight down’.

This study reinforces what has been known in a general way for a long time: the heart is a spherical to cone shaped, non-centric organ, suspended by the pericardium in the mediastinum and situated only in part beneath the lower middle of the sternum. The anteroposterior (AP) internal distance (T) in this study ranged from 12.6 ± 2.4 to 13.4 ± 2.3 cm, respectively at T1 and T2, with cardiac or vascular structures accounting for only 8.6 ± 1.6 cm at the T2 level (Figure 3). These results do not contradict Kouwenhoven, in suggesting that part of the heart may not be directly compressed between bony structures when moving the sternum dorsally for up to 5.1 cm to cover the mean value of 4.2 cm between bony and cardiac structures. Instead it highlights simplifications that have been added since Kouwenhoven, who stated that allowance must be made for (larger) chest size, accommodating the need for deeper impression. With part of the cardiac structure subject to direct compression, notably the right atrium and to a lesser degree the right ventricle, makes it difficult to explain significant size reduction of the left ventricle reported in clinical and animal echocardiographic studies.

Johansen and Ruben, working in eight healthy, anesthetized and curarised humans, divided into a younger and an older group, found that internal AP size of the midline thoracic cavity is between 8 and 19 cm and noted that force and impression depth were linearly related in the range of 0.8 – 2 mm kg⁻¹ (0.36 – 0.9 mm lb⁻¹). Johansen also suggested that the difference between internal and external AP distance was in the order of 8 cm. The present study points to ΔT₁,₁ = 10.5 ± 1.6 cm (range 8.1-14.8 cm) and ΔT₁,₂ = 11.0 ± 2.1 cm (range 5.9 – 14.4 cm). In the quoted study, and more particularly in their later study Ruben notes that depression of the sternum was generally less than 5 cm, and no correlation was sought with the effect of this distance on the midline structures. Notably, this group demonstrated that the compliance, involving two areas, a tenfold ratio in size to each other, showed reproducible (static) effects in impression depth. Roberts and Chen, modeling the thoracic cavity for high impact purposes describe the effect of sternal loading at 100 lbs find less than 3 inches posterior displacement. Recently, Steen et al. reiterated the importance of this point, as a byproduct of their validation of LUCAS in a swine model. Their 19.5 cm skin-to-skin AP diameter approaches the median of 24 cm in humans found in the present study. The associated (maximum) compliance of 1.0 x 10⁻² cm /N⁻¹ is at the upper half of earlier studies as demonstrated in Table 3. A thoracic to cardiac diameter ratio cannot be distilled from their work. Table 3, culled from the literature, with units converted to cm N⁻¹ to allow comparison of chest compliance, demonstrates variation in compliance by an order of magnitude. This variation means that different forces will be required to achieve the same impression depth, a challenge difficult to meet without monitoring depth in any given patient.

4.4.1 Models of the circulation for cardiopulmonary resuscitation

Applying distributed models (Table 4) a few groups, notably Babbs et al. (1984 and further) and Beyar et al. (1985 and further) expanded the search for causes that support the circulation by considering both the cardiac pump theory (blood being moved by cardiac compressions only, with mitral valve closure) to the thoracic pump theory (variations in intra- and extra thoracic pressure move blood through the heart, and beyond, with peripheral venous valves contributing to unidirectional flow.) These groups have consistently attempted to convert human and animal experiments into useful models without the clinical confounders which have made interpretation of data difficult.

In these models, organs as well as structures are subjected to external pressures as chosen by the investigators. This permits selective application to a single organ (i.e., the heart or parts of it), or
multiple organs. This and the individual characteristics of the organs and structures may have been overly simplified in the models, when the effects of outside pressure were analyzed. In recent insight, Markstaller and coworkers,\textsuperscript{16} working in a swine model demonstrated that large segments of the lung collapse between ventilations and that this may detract from the conduction of pressure to intrathoracic structures, as supposed in the “thoracic pump” models.

Table 3: Review of human experiments involving sternal impression, force and distances, and including an early ‘Guideline’ (in italics) as reference. This table lists the studies in chronological order with the findings as explained in the manuscript. To simplify comparison, standardized compliance is calculated and presented, assumptions, if made, are noted in parenthesis. The comments column supplies additional information. n.d.a. = no data available.

<table>
<thead>
<tr>
<th>Findings as described (x) = number of cases</th>
<th>Standardised N &amp; compliance C (cm N$^{-1}$)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Harkins et al.\textsuperscript{17} 1961.</td>
<td>Force: 60-75 (95) lb Depth: n.d.a. Source: n.d.a.</td>
<td>$267-334 \frac{N}{1 \times 10^2}$ Depth = 3 cm (assumption).</td>
</tr>
<tr>
<td>Rivkin et al.\textsuperscript{18} 1962.</td>
<td>Force: 70-90 lb Depth: 3-4 cm Source: n.d.a.</td>
<td>$356 \frac{N}{1 \times 10^2}$</td>
</tr>
<tr>
<td>Warttier \textsuperscript{19} 1963.</td>
<td>Force: 30-40 kg Depth: 10 cm Source: clinical (18)</td>
<td>$294-392 \frac{N}{2.9 \times 10^2}$ Used a calibrated mechanical compression device. Effect evaluated as palpable femoral pulsation.</td>
</tr>
<tr>
<td>Johansen \textsuperscript{10} et al. 1964.</td>
<td>Force: 25-50 kg Depth: 5 cm Source: clinical (8) Overall: 0.8-2 mmkg$^{-1}$</td>
<td>$245-490 \frac{N}{0.8-2 \times 10^2}$ Static experiment with different compression area in healthy volunteers. (2 blocks) Linear relationship.</td>
</tr>
<tr>
<td>Ruben \textsuperscript{11} et al. 1965.</td>
<td>Force: up to 60 kg Depth: up to 71 mm Source: cadavers (8) Overall: 0.6-2.0 mmkg$^{-1}$</td>
<td>$390 \frac{N}{1.2 \times 10^2}$ Static experiment 2-10 hours postmortem. Auditory and visual validation of fractures. 2 blocks. Sternal &amp; sternal + rib compression between blocks. Linear relationship.</td>
</tr>
<tr>
<td>Nachlas \textsuperscript{20} et al. 1965.</td>
<td>Force: 120-130 lb Depth: n.d.a. Source: clinical (7)</td>
<td>$356 \frac{N}{0.5 \times 10^2}$ Depth = 3 cm (assumption) Used a calibrated mechanical compression device. Effect evaluated as palpable arterial pulsation. No correlation found between output and depth.</td>
</tr>
<tr>
<td>\textsuperscript{1966.\textsuperscript{14}}</td>
<td>Force: 80-120 lb Depth: 1.5 – 2 inches Source: n.d.a.</td>
<td>$356-534 \frac{N}{1 \times 10^2}$ Ad hoc committee on CPR, National Institute of Health.</td>
</tr>
<tr>
<td>Tsilik \textsuperscript{12} et al. 1983.</td>
<td>Force: 0-540 N Depth: formula Source: clinical (11) $F = \beta D_s + \gamma D_r^2$</td>
<td>$769 \frac{N}{0.2 \times 10^4}$ for $D_s$ = 3 cm $1831 \frac{N}{0.1 \times 10^2}$ for $D_r$ = 5 cm Patients in arrest. $\beta = 91.7 \pm 31.2$ N cm$^{-1}$ $\gamma = 54.9 \pm 29.4$ N cm$^{-2}$ Nonlinear relationship.</td>
</tr>
<tr>
<td>Bankman \textsuperscript{3} et al. 1990.</td>
<td>Force: ~350 N Depth: ~3 cm Source: clinical (1)</td>
<td>$350 \frac{N}{0.9 \times 10^4}$ First visco-elastic model, never deeper than 3 cm.</td>
</tr>
<tr>
<td>Grubin \textsuperscript{18} et al. 1993.</td>
<td>Force: $431 \pm 30$ N Depth: $3.83 \pm 0.56$ cm Source: clinical (16) Formula in manuscript.</td>
<td>$431 \frac{N}{0.9 \times 10^4}$ Patients at end-stage failed resuscitation (57 – &gt; 120kg) 5 cm depth not reached in any patient. Confirmed visco-elastic effects.</td>
</tr>
</tbody>
</table>
While the model allows quantitative assessment of the pressures and flow throughout the range included in the model, Table 4 provides an overview, listing pressures applied and equivalent forces (in italics) if the pressure were applied to a flat surface like the palm of the hand with an area of 90 cm² to facilitate comparison between Tables 3 and 4. For an impression of 5 cm, the forces required in Table 3 are force = (1 / compliance) x (compression depth) and tend to exceed the forces listed in italics in Table 4, by a significant amount.

The early standard, suggested by Kouwenhoven as (at least) 1.5 to 2 inches, has remained the basis for work done, even though Babbs and coworkers suggested a clinically relevant minimal impression depth of 1.5 to 3 cm while working in the highly compliant dog model.27, 24 Conversely, Gruben et al.25 questioned the feasibility of reaching this depth.

Opinions, centering on supporters of the thoracic pump theory, and its pure form in vest CPR26 have, however, laid a basis for re-evaluation of the purpose for sternal impression, as reaching the heart is not an issue in this model.

The use of models has found little appreciation within clinical medicine, although they have reported in-depth insights and positive results in a large percentage of the types of work attempted. Reasons for this may be that modeling studies have addressed behavior of the circulation emphasizing more complex material or CPR modalities such as adjuvant techniques not as yet broadly accepted. While the models are uniquely suitable for the determination of the effect of depth (a teachable entity) and force (a more readily measured entity), choices are made based on facts not in evidence. Complex model studies evaluate the potential gain between orphaned innovations and more conservative techniques, without first validating the model for optimal aspects in the standard technique. Nowhere in the models are explicit investigations into the effect of impression depth on a specific thoracic to cardiac ratio to be found.

4.4.2 Motivation for further model making

If impression depth is insufficient to act directly on the cardiac structures, and most particularly on the left ventricle, as Kouwenhoven suggested, what is the effect that is keeping up to 30% of victims of cardiac death alive. The CT scan suggests that if cardiac structures are compressed these structures should be the right atrium and the in- and outflow tracts, within some cases the right ventricle, and the thoracic aorta. This is compatible with available evidence for pressures in the right atrium equal or higher than in the aorta during CPR systole.

Chest compression causes a general increase in intrathoracic pressure which will operate differently on the central systemic veins and the right atrium, the right ventricle, the pulmonary vasculature and the left heart. That there is a difference in how the pressure acts on the intrathoracic structures may be illustrated by the following specific case. If blood is contained in a perfectly rigid envelope, an increase in external pressure affects neither blood pressure nor dimensions. If blood is contained in an easily deformed envelope, the blood pressure inside will simply follow the change outside and alterations in dimensions may occur secondarily via modification of local blood flow as made possible by valves and compliance. Formulated mathematically, with \( p_i \) and \( p_e \) being internal and external pressure respectively, the pressure difference across the wall will be \( \Delta p = p_i - p_e \) which is equal to \( V/C \), with \( V \) (volume) and \( C \) (compliance) of the envelope, assumed to be constant in the operating range. Restated \( V = C \Delta p \). For a constant volume \( V \), in a very distensible chamber, \( C \) is very large making \( \Delta p \) very small, and internal pressure will tend to approximate external pressure. If \( C \) is small, however, this transmission of pressure will tend towards zero.

Applied to the models of CPR, an increase of intrathoracic pressure, secondary to movement of the sternum, will be readily transferred to the central veins and the thin-walled right atrium, to some lesser degree to the right ventricle, and so on. There is evidence that the pericardium will not protect against this mechanism.

Difficulty also remained in clinical practice, where impression was practiced on manikins and reflected in depth, force not being a “teachable” entity to either professionals or laymen, and requiring apparatus for use. Training manikin makers have chosen 343-392 N (88 lbs) as being acceptable for 5
cm of impression depth (39-59 lb inch\(^{-1}\)), but later reduced this to at least 294 N for 4-5 cm impression depth.\(^{27, 28}\) Baubin demonstrated that some manikins have been constructed to simulate non-linearity after 3 cm, improving modeling of the human chest.\(^{29}\) That a relationship between the quality of a model and clinically relevant outcome is likely was pointed out by Wik and coworkers,\(^{30}\) who reflect on the poor skills in reaching adequate compressions during actual out-of-hospital CPR. These aspects in manikins, as optimal model for the practice of psychomotor skills, cannot be ignored.

Babbs et al.\(^{14}\) Beyar et al.\(^{15}\) and others have shown that models can promote understanding of physiological mechanisms, but that the use of the model may be limited by available insight. Recognition of the cardiac pump mechanism in a thorax with a deeper anterior-posterior depth than animal data has allowed for, and further specification of pressure effects, will contribute to future understanding.

### 4.5 Conclusions

Compression of the sternum by 1.5 to 2 inches, achieving cardiac compression and generating cardiac output, has been suggested to be due to left ventricular compression with closure of the mitral valve. Animal models, findings in humans and mathematical models have, however, used forces (25-50 kg), pressures (0-120 mm Hg) and distances (0 – 7 cm) that are different in static and dynamic settings. This study demonstrates that past assumptions as to the internal size of the chest may be conservative (8 cm versus measured \(\Delta T_{e1} = 10.5 \pm 1.6\) cm (range 8.1-14.8 cm) and \(\Delta T_{e2} = 11.0 \pm 2.1\) cm (range 5.9 – 14.4 cm), and that cardiac structures amount to only 8.6 ± 1.2 cm in the transverse plane at the lower border of the tricuspid valve.

Mathematical models are uniquely suited to investigate effects of pressure and compression depth, without clinical confounders, but should be more consistent, be directed more toward fundamentals as opposed to demonstrating benefits of orphaned innovations. The groups led by Babbs and Beyar have laid a foundation readily available for further work.
<table>
<thead>
<tr>
<th>Table 4: Force and depth chosen to drive the circulation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brief description of model</td>
</tr>
<tr>
<td>Babbs et al. 1984</td>
</tr>
<tr>
<td>Beyar et al. 1987</td>
</tr>
<tr>
<td>Beyar et al. 1985</td>
</tr>
<tr>
<td>Hulce et al. 1987</td>
</tr>
<tr>
<td>Toma et al. 1987</td>
</tr>
<tr>
<td>Babbs et al. 1999</td>
</tr>
<tr>
<td>Beyar et al. 2000</td>
</tr>
</tbody>
</table>

Abbreviations: ACDC = active compression decompression; AO abd AO th = abdominal and thoracic pump.

For the calculation of force, the area of contact was assumed to be = 90 cm² unless otherwise stated in Table 4.
Table 4: Summary of models involving CPR with reference to sternal impression, force and distances. Abbreviations: ACDC = active compression decompression; AO_{ab} AO_{th} = abdominal and thoracic aorta respectively; Eq = equations; HIC CPR: High impulse compression CPR; IAC = interposed abdominal compressions; IVC = inferior vena cava; CPR = cardiopulmonary resuscitation; mm Hg = millimeters of mercury; N = Newton; n.a. = not applicable; OCCR = open chest cardiac resuscitation; SPICE (Simulation program for integrated circuit evaluation, see ref in 1995; 458; SVC = superior vena cava; T_p = thoracic pump factor.

For the calculation of force, the area of contact was assumed to be \( A = 90 \text{ cm}^2 \) unless otherwise stated in the ‘comments’ column.
4.6 References:

Models, model making, simulations and their potential for circulatory research

Chapter 5: Modeling, and its potential for circulatory research.

5. MODELS, MODEL MAKING, SIMULATIONS AND THEIR POTENTIAL FOR CIRCULATORY RESEARCH......................................................... 105

5.1 Introduction ............................................................................................................................. 107
5.2 Modeling and simulation......................................................................................................... 107
5.3 Simple terms in modeling........................................................................................................ 108
5.4 Early models in cardiovascular research ................................................................................. 109
5.4.1 Fluid-mechanical models.................................................................................................... 109
5.4.2 Electrical models................................................................................................................ 109
5.4.3 Mathematical models.......................................................................................................... 110
5.5 The development of cardiovascular system models based on sophistication ....................... 110
5.6 Experiments on animal models of different kinds................................................................. 112
5.7 Models in CPR: under appreciation of a potential source of insight ..................................... 113
5.8 Summary ................................................................................................................................. 114
5.9 References ................................................................................................................................ 115

Table of contents

5. MODELS, MODEL MAKING, SIMULATIONS AND THEIR POTENTIAL FOR CIRCULATORY RESEARCH......................................................... 105

5.1 Introduction ............................................................................................................................. 107
5.2 Modeling and simulation......................................................................................................... 107
5.3 Simple terms in modeling........................................................................................................ 108
5.4 Early models in cardiovascular research ................................................................................. 109
5.4.1 Fluid-mechanical models.................................................................................................... 109
5.4.2 Electrical models................................................................................................................ 109
5.4.3 Mathematical models.......................................................................................................... 110
5.5 The development of cardiovascular system models based on sophistication ....................... 110
5.6 Experiments on animal models of different kinds................................................................. 112
5.7 Models in CPR: under appreciation of a potential source of insight ..................................... 113
5.8 Summary ................................................................................................................................. 114
5.9 References ................................................................................................................................ 115
5.1 Introduction

The term ‘model’ is frequently used. In clinical medicine, it is often applied to animals who take the position of humans, allowing experimental protocols to be performed based on the assumption that the animal is physiologically or pathologically similar to the human. It may also apply to isolated organs. Manikins, used for teaching or experimental work have also been described as models. All too often this issue is further confused by the use of model, simulation and experiment as if they are interchangeable concepts.

In this chapter the scientific continuum involved in modeling will be reviewed with specific focus on the circulation and where feasible, on cardiopulmonary resuscitation.

5.2 Modeling and simulation

Aristoteles\(^1\) (384-322 BC) argued that the study of nature requires investigators to follow a specific Method. He complained that though there had been a true interest in this approach during the time in which Socrates lived, (c. 470-399 BC), men had since then given up enquiring into the works of nature and had diverted their attention to political science and to such virtues as seemed to benefit mankind.

Over the centuries, interest in fundamental investigation continued to wax and wane, until eventually the Western Renaissance inspired a long and sustained era of study, which emphasized experimental work. This allowed Descartes (1596-1650), in his discussion on the Method, to expand upon Aristoteles’ view by reasoning that to him, mathematics should provide the starting point for an explanation of what is observed in nature.\(^2\) Adherence to his own teaching proved difficult, however. In part five of the Discourse, while discussing the movement of blood, he agrees with Harvey that blood is in perpetual motion around the circuit, while disagreeing with Harvey about the cause of this motion. Descartes imposes older views by claiming that it is the heat from the heart which is the cause for the circulation. Aristoteles’ original Method, as well as Descartes’ Method have been expanded further.\(^3\)

Interest again shifted subtly in the Renaissance, up through the Second World War, to focus on interpretation of observations in nature. When possible, the observations would lead to physical (i.e. apparatus oriented) or animal models as a form of quantitative analysis. The domination of the physical experiment, prior to the second World War, became more balanced with the creation and growth of biochemistry, subsequently followed by biophysics, biomathematics, bioengineering, and similar process-oriented courses of study and work. In these young fields the emphasis was based on models, i.e. mathematical formulation of observed natural phenomena, based on natural laws.\(^4\)

Making a model bears some similarity to a confession of faith: the investigator makes a strong effort to identify the mechanisms involved in what is observed.\(^5\) What the model suggests must be interpreted, has the status of new observations in nature and may be questioned leading to either reduction or expansion of that model or to completely new conceptual interpretations.\(^6\) This is fundamentally different from simulations where the goal is to reproduce or imitate observed phenomena, and the mechanisms involved in this reproduction of the observations are essentially irrelevant.\(^7\) Simulation has been proven to have important didactic uses though, as seen in flight simulations, and anesthesia oriented patient simulations.
The purpose of modeling is to gain an understanding of the process and the laws as they apply, while the principal purpose of simulation is the directed interaction of potentially complex processes to a predefined goal.

Both modeling and experimentation have strong and weak points. They are often considered at opposite extremes of attempts to achieve progress. Experiments, with their appealing aura of extracting information from reality, may not allow determination of boundary conditions or the value of critical parameters, leading sometimes to opposite conclusions derived from allegedly equivalent or even identical experiments. The literature furnishes many examples witnessing this, as does CPR research in particular, where positive outcomes in experiments in the lab cannot be duplicated in the field. Modeling entails the search for a relationship between cause and effect, based on established laws. In other words the search for a mechanism able to interpret observations. Frequently, this approach requires dexterity in mathematics, which commonly, is not part of medical school curricula. Models may pursue relevant concepts or incorrect ideas or focus on details while ignoring related major effects. Ideally, the interaction between experiment, clinical medicine, and modeling should serve to weed out such errors and deficiencies.

5.3 Simple terms in modeling

Historically, the terms used in modeling have been given electrical equivalents in order to visualize their functions (Table 1). In this system flow (Q) and current (i) may be equated. Care should be taken in the definitions involved in these representations as some electrical symbols may have their own definitions. Note that valves may be drawn as diodes, but that electrical diodes have a structure different from biomedical valves. Note also that while an electrical charge may become negative, volume, i.e. intravascular volume, its equivalent, can not.

In the Donders model (Appendix II), while electrical networks are used for succinct presentation purposes, the model is mathematical.

Table 1: Equivalent electrical and physical symbols used in models of the human circulation.

<table>
<thead>
<tr>
<th>ELECTRICAL</th>
<th>PHYSIOLOGICAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quantity</td>
<td>Symbol (units)</td>
</tr>
<tr>
<td>Variable</td>
<td>Symbol (units)</td>
</tr>
<tr>
<td>Charge</td>
<td>q (Coul.)</td>
</tr>
<tr>
<td>Current</td>
<td>i (Amp)</td>
</tr>
<tr>
<td>Voltage</td>
<td>V (Volt)</td>
</tr>
<tr>
<td>Parameter</td>
<td>Symbol (units)</td>
</tr>
<tr>
<td>Resistance</td>
<td>R (Ohm)</td>
</tr>
<tr>
<td>Inductance</td>
<td>L (Henry)</td>
</tr>
<tr>
<td>Compliance</td>
<td>C (Farad)</td>
</tr>
</tbody>
</table>

As far as possible, and allowing for typical usage in clinical medicine, the g-cm-sec, system of international units is used, although the popular mm Hg does not belong in this system. For limitations, see text in section 5.3.
5.4 Early models in cardiovascular research

5.4.1 Fluid-mechanical models
The interdependence of events in the circulatory system has long enchanted investigators. To visualize the situation better, some built hydraulic models of the circulation. Such models, originally designed primarily for teaching purposes, were published by Weber and by Marry. These models rely on mechanical parts, such as elastic tubes filled with a fluid (e.g. water, which naturally introduced a small variance in density of circa 6%). These models could be used to clarify the motion of the fluid under given boundary conditions, for example the value of the pulse wave velocity. In such a model, compliances are joined via resistors and inductors. This sort of system permits pressure and flow to depend on location, i.e. by the differences in compliance and size of the vessels. Practical problems, such as blood viscosity, the adjustability of parameters, the lack of availability of mechanical parts with prescribed properties (e.g. tubes that display prescribed changes in diameter and in elastic properties along their lengths) and leakage, encountered in hydraulic models discouraged their further development.

5.4.2 Electrical models
Over the years, as described above, researchers learned that the flexibility of fluid-mechanical models were rather restricted. Progress in electrical engineering gradually made it more attractive to translate the fluid-mechanical problem into ‘equivalent’ electrical terminology, solve the electrical problem and translate the solution back into the fluid-mechanical model. Starting with a hydrodynamic model, then moving to a mathematical and later an electrical model, Jochim, in 1948, studied the effect of changes in peripheral resistance on arterial pulse pressure using heart rate and stroke volume as independent variables. Landes demonstrated that the hemodynamic pulsations in the circulation were similar in character to electrical impulses propagated through cables. In this way, simple models could be built to further understanding of the circulation.

More flexible versions made their appearance starting in the late 1950’s, laying the ground work for features in the Donders model. In the 1960’s, De Pater et al., were the first to attempt to apply the electrical analog to the entire circulatory loop. Attinger, continuing this line of investigation, recognized the difficulty in finding good experimental (clinical) data to verify observations made in models. Indeed, the oversimplification in many models reduced their potential for advancing insight. The more complex versions modeled the left side of the heart, the aorta, the peripheral resistance and the veins as a closed loop, with valves at a few appropriate sites. A limited set of parameters, such as peripheral resistance, arterial and venous compliances and the frequency of ventricular contraction, could be adjusted allowing qualitative analysis of the resulting effects.

As early as 1984, Babbs et al. applied an electrical model to the circulation under CPR conditions. The purpose of this study was the exploration of possible advantages of abdominal compressions together with chest compressions in cardiopulmonary resuscitation by the use of an electrical model. No electrically oriented work had been done on CPR, which could serve as a basis for this specific technique.

An actual electrical model was used, allowing isolated variance of the compressions on the heart and abdominal compression pressure. However, even though questions were raised about the quality of the parameter values used, this procedure was extended to animal and even human use. This concept of modeling to generate baseline data was extended further when Babbs, in 1984, attempted to compare cardiac, thoracic and abdominal pump mechanisms. Here, an extended electrical model was used, allowing isolated variance of pressure effects on the heart (i.e. cardiac compression), the entire thorax (i.e. the thoracic pump), the abdominal aorta or the abdominal veins (i.e. the abdominal pump). In this study, some known patho-physiological aspects (i.e.
decreased compliance as a factor of distention) were ignored. Indeed, conversion of fluidic to
electrical units required a number of assumptions. Interestingly, clinical science refused to accept
this data as a basis for mention in the guidelines for resuscitation.23

5.4.3 Mathematical models
With the advent of computers and their rapid development, new possibilities became available.
These include the solution of nonlinear problems, which were difficult to achieve with fluid-
mechanical and electrical models (e.g. relating to collapse of vessels, effects of valve closure and
opening, ventricular contraction).

Beyar, using a mathematical model with parallel capacitance elements and series inductive and
resistive elements within a “lumped-parameter” model explored the variation of intrathoracic
opening, ventricular contraction).

Modeling may also serve as a tool to obtain unknown, not directly measurable, properties of
mechanical structures (e.g. the stiffness of ribs and the flexibility of joints). To this goal,
modeling has been done on the dynamic characteristics of the chest, as this is of direct influence
on the mechanisms by which blood should flow when the chest is compressed during CPR.

Babbs used a mathematical model, in 1999, to describe and compare interposed abdominal compressions
(IAC), active compression-decompression (ACD), and Lifestick™ CPR techniques.25

Bankman et al.26 describe such a viscoelastic system incorporating a force operating on a spring
with damping and mass. They suggest that there are severe limitations in the training manikins
available, based on the linearity in their spring systems. Others have supported the notion that the
human chest has distinct non-linear characteristics when cyclically compressed and relaxed.27,28

5.5 The development of cardiovascular system models based on sophistication

The variety of models that have been designed to study of the circulation can be conveniently
divided into four classes according to levels of their sophistication.29 In its simplest form, the
circulation has been represented by a resistive model. Hill et al.30 designed a model of this nature
to study the changes in the circulation at birth, while Vadot proposed one to predict the changes
to be expected from major surgical intervention.31

The resistive-capacitive model32 was aimed at a description of average values of pressures, flows
and volumes and introduced the behavior of the heart based on Starling’s concept33 in the form:

\[ W_s = SV_{ed} \] (eq. 5.1)

where \( W_s \) denotes the work performed by the ventricle and \( V_{ed} \) its end diastolic volume (EDV),
while S is a proportionality constant between the two, denoted the contractile strength of the
ventricle. The incorporation of Starling’s concept in the model popularized formulations of
ventricular performance.

Pulsatile phenomena were first included in Warner’s resistive-inductive-capacitive (RLC) model
which also set the structural pattern for many subsequent studies.34 To illustrate the growing
complexity and the development of the systems of equations, this model is described in more
detail.

The circulatory loop was subdivided into a number of sections with the condition that outflow of
any section equals inflow into the next. For each section, “i”, a set of at least three equations must
be written. In Warner’s model these are: i) an equation of motion, which is commonly a
simplified version of the Navier-Stokes equation:

\[ p_i(t) - p_i(t) = L_{i-1} \frac{d}{dt} Q(t) + R_{i-1} Q(t) \]  
(eq. 5.2)

in which \( p \) defines the operating pressure, \( Q \) describes the flow, \( L \) accounts for the inertial, and \( R \)
for viscous properties of blood; ii) an equation of continuity which relates change in blood
volume \( V \) contained by a section to its inflow and outflow, such that:

\[ V_i(t) = V_i(t=0) + \int [Q_i(t) - Q_{i+1}(t)] dt \]  
(eq. 5.3)

and iii) an equation of state, which relates pressure in a section to its volume through the
compliant properties of the wall. For vessels he defined \( m = 1 \) for arteries and \( m > 1 \) for veins,
such that:

\[ p_i = \left( \frac{1}{C_{id}} \right) V_i^m \]  
(eq. 5.4)

For the ventricles, Warner introduced the concept of the time varying compliance \( C \), with \( C_{id} \) and
\( C_{is} \) as constants for diastolic and systolic segments of a cycle, respectively, such that:

\[ p_i = \left[ \frac{1}{(C_{id} \text{ or } C_{is})} \right] V_i^m \]  
(eq. 5.5)

Subdivision of the circulation into six sections required the solution of 18 simultaneous equations,
with the cardiac valves opening and closing with the aid of a multiple diode function generator.

Simple resistive-capacitive as well as the more complex resistive-inductive-capacitive models
were able to exhibit some of the striking features that are recognized in the mammalian
circulation, (Figure 1) such as modulation of stroke volume and effect of volume loading.
However, the conduct of contractile chambers remained a notable exception.

The success of resistive-capacitive, but more so of RLC models strongly stimulated their repeated
application and rapid expansion, e.g. by Defares et al., Beneken, and Dick et al., as well as
inspiring inclusion of control phenomena in which the model becomes active. As an example of
this fourth group, Beneken and De Wit\(^{39} \) incorporated direct baroreceptor control of heart rate and
of the systemic peripheral resistance.

Karreman and Weygandt expanded carotid sinus control of the peripheral vascular bed resistances
by modeling the physiological feedback mechanism through a set of equations. They related
carotid sinus pressure, via its wall deformation to the value of baroreceptor nerve firing rate, then
to sympathetic nerve firing rate, and finally sympathetic firing rate to the peripheral bed
resistances as well as to negative feedback in the elastic modulus of the carotid arterial wall itself.
Shifting emphasis to longer-range control, Guyton et al., developed a larger set with over 350 equations. In essence, their model contains the blood conducting pathway, vascular stress and relaxation as it affects circulatory pressure, membrane dynamics of the capillaries, tissue fluid volume and pressure, electrolyte shift, angiotensin, aldosterone, and antidiuretic hormone control, kidney dynamics, control of blood flow in muscle, autoregulation, autonomic control as well as a number of other control facets.

After these and many similar expressions of enthusiasm, the tide turned and critical levels began to prevail. As a result, the biomedical basis for the Korotkoff sounds was discovered with the aid of a mathematical model, and the interpretation of wave transmissions through arteries was finally found by the use of an extensive mathematical model.

**5.6 Experiments on animal models of different kinds**

As stated earlier, if a study object cannot be completely defined and its variation controlled it may lead researchers to draw misleading conclusions in animal and even human models. Lafollette and Shanks suggest that animal models are, in fact, weak except when they are identical in all causally relevant aspects.

Very different animal models have been used for research in CPR over the decennia, ranging from nonhuman primates, rats, cats, canines and most recently, swine.

The popularity of the pig as an acceptable model for many investigative lines can be demonstrated by the decline of dogs registered for research purposes from 201,000 in 2001 to...
68,000 in 2002, while the number of pigs registered grew to 86,000 in 2002. Central to the acceptance of animal models in a complex process should be careful investigation and validation of their physiology and pathology in relation to humans. This should include aspects of gross anatomy, such as the position and form of the heart and its internal anatomy as well as electromechanical aspects of, for example, the myocardium or more detailed coronary artery effects. Early CPR research focused on the use of cat and dog oriented animal models, some of which were highly developed, which was later differentiated to include a small dog (suitable as a highly compliant, potential cardiac pump) model for pediatric analysis. A large dog, generally described to weigh more than 20 kg was deemed suitable for adult CPR investigations. The use of a supine or a “V” board for support was accepted as interchangeable, while current thinking recognizes the importance of the effect of thoracic lateral motion during compression.

Details of the coronary circulation have been described in dogs as early as 1987, suggesting that this model was less suitable for ischemic models due to the wide range of networks. This was, perhaps, a factor in suggesting that the swine would be a better model, due to its greater similarity to humans in this area. In addition, the heart size to body weight ratio of (0.005 ml gr^-1) for a 30kg swine is identical to that of the human, the typical structure of the coronary arteries, including its right-dominant system and its cardiac conduction system were deemed similar though other aspects, such as the morphology of the heart itself, differ.

As pointed out above, animal models incorporate a number of intrinsic limitations, including, but not limited to, anatomical differences, physiological, as well as morphological and biochemical differences from the human. While swine models have become the standard in CPR research, well known limitations of this model remain the non-human anatomy of the chest wall and the (thoracic) visceral organs.

5.7 Models in CPR: under appreciation of a potential source of insight

Babbs et al. initiated work in modeling as it applies to CPR. In a consecutive series of papers they described and analyzed a number of features both inside and outside main-stream CPR research.

Many theoretical scientists have been frustrated by the value placed by clinicians on model-makers using animals and their outcomes. Babbs, in his extensive review of experimentation on interposed-abdominal compression CPR comments on this.

However, success has also been achieved using mathematical modeling of the circulation. Even under controllable conditions, fluid-mechanical models may be limited by the complex fluid properties and unknown mechanical properties, causing the researcher to encounter difficulties defining the object of the study. Electrical models, if carefully organized, allow the object of study to be more carefully defined and controlled as the parameter values and variables can be controlled. For experiments in mathematical models with computer support, the greatest available flexibility is allowed, provided the researchers know any possible distortion introduced by the computer, such as negative volumes. As illustrated in Table 5.1 quantitative translation between models can often be achieved.

In principle, an investigator can choose from (fluid) – mechanical, electrical, or mathematical models in the search for the interpretation of a physiological or clinical observation.

The choice is often biased by the training and the preference of the investigator as well as by the practical limitations of the different models.

---

* http://www.aphis.usda.gov/ac/publications
5.8 Summary

Clinical medicine has, to date, made surprisingly little use of inanimate models for fundamental research and has maintained that the - admittedly imperfect- options offered by animals and large series of experiments in humans set the optimal standard for making choices. This is reflected in the level of evidence (LOE-7) accorded to model makers (see Chapter 2). Much of the research done in CPR, up through the 1990’s, must be carefully analyzed as it was done in cat and dog models. More recent investigations have used swine, which while also imperfect, have superior fidelity as a model for the human. Careful steps in changing this are, however, visible, with CPR research implementing ventilation to compressions ratio’s suggested by a mathematical model.
Chapter 5: Modeling, and its potential for circulatory research

5.8 Summary
Clinical medicine has, to date, made surprisingly little use of inanimate models for fundamental research and has maintained that the - admittedly imperfect- options offered by animals and large series of experiments in humans set the optimal standard for making choices. This is reflected in the level of evidence (LOE-7) accorded to model makers (see Chapter 2). Much of the research done in CPR, up through the 1990’s, must be carefully analyzed as it was done in cat and dog models. More recent investigations have used swine, which while also imperfect, have superior fidelity as a model for the human. Careful steps in changing this are, however, visible, with CPR research implementing ventilation to compressions ratio’s suggested by a mathematical model.

5.9 References
6. Arnoczky SP. Selecting a valid animal model; scientific, veterinary and practical consideration. 53rd Annual Meeting of the orthopaedic research society. San Diego, CA, Feb. 2007


33 Patterson SW, Starling EH. On the mechanical factors which determine the output of the ventricles. J. Physiology 1914; 48: 357-379.


46 Bourne PR, Kitney RI. Investigation of circulatory model design for clinical application. J Physiol. 1979; 293: 6P-7P.


Chapter 5: Modeling, and its potential for circulatory research

Layout and description of the Donders model and its progression
Chapter 6: Layout and description of the Donders model and its progression

6.1 Introduction

The term 'model' and the purpose of 'modeling' in science were introduced, illustrated and discussed in the preceding chapter. In the current chapter, our model, named after the late Dutch medical scientist Franciscus C. Donders (1818 – 1889) will be presented, as well as brief descriptions of its successive expansions. The reasons to use Donders name are somewhat diverse: he is well known as the most important scientist born in Tilburg, where a large part of this dissertation was prepared, due to his little known early work in cardiovascular-pulmonary interrelationships, as well as his work in the field of neuro-cognitive analysis, for which an international institute has been founded in Nijmegen.

The primary purpose of this tool is to help understand pathological aspects of the circulatory system, especially those occurring in failure of the heart and the respiration. In this chapter, we will present the chronological developments in the Donders model, and illustrate technical solutions which have been incorporated into the model over time.

Models of the circulation have been under development since Euler (1775), who attempted to predict the pulse wave velocity in an artery, but was unable to solve the two underlying differential equations. A century later, Weber (1850), who became interested in the role of the volume of circulating blood, actually built a model. This activity reached a zenith in 1972 with the publication of Guyton et al.'s model of circulatory regulation and control, containing over 350 mathematical equations. The size of this model triggered a period of introspection.

Since the closed loop circulation contains blood conducting pathways as well as blood pumps, a number of physical quantities play key roles. These include inertial and viscous properties of blood, the distensible properties of blood vessels and the time-varying contractile and relaxing properties of pumps. These parameters are related in equations which result in pressure, flow and volume phenomena, all of which vary with time. Preceded by years of research into the conduct of the arterial system, initial modern descriptions included no less that 200 simultaneous differential equations, requiring computer assistance to solve. This could, with the introduction of Zin be reduced to three element modeling 'units' with the characteristic impedance Zo, the total compliance of the system C, and the peripheral resistance Rs. Expansion include the 'ejection effect' kappa, which reduces ventricular pressure during ejection and magnifies it during ventricular relaxation, as well in inclusion of the shapes of the ventricular ejection curve and pressure was, based on work by Mulier et al. early in 1998. The equations establish the Donders model. The solutions to these equations furnish the material intended to help understand pathological issues.

Table of contents

6  LAYOUT AND DESCRIPTION OF THE DONDERS MODEL AND ITS PROGRESSION 119

6.1 Introduction ................................................................. 121
6.2 General anatomic and symbolic layout of the Donders model (DII) ........................................... 123
6.3 The cardiac chambers .......................................................... 124
6.3.1 Negative volume in the specific case of cardiac chambers ................................................. 127
6.4 Valvular opening and closure .............................................................. 128
6.5 Arterial systems .................................................................................. 128
6.6 Venous systems .................................................................................. 128
6.7 Controls .......................................................................................... 129
6.8 Adaptations to CPR ............................................................................. 129
6.9 Initial conditions .................................................................................. 130
6.10 Summary .......................................................................................... 130
6.11 References ....................................................................................... 131

120 Chapter 6: Layout and description of the Donders model and its progression
6.1 Introduction

The term ‘model’ and the purpose of ‘modeling’ in science were introduced, illustrated and discussed in the preceding chapter. In the current chapter, our model, named after the late Dutch medical scientist Franciscus C. Donders (1818 – 1889) will be presented, as well as brief descriptions of its successive expansions. The reasons to use Donders name are somewhat diverse: he is well known as the most important scientist born in Tilburg, where a large part of this dissertation was prepared, due to his little known early work in cardiovascular-pulmonary interrelationships, as well as his work in the field of neuro-cognitive analysis, for which an international institute has been founded in Nijmegen.

The primary purpose of this tool is to help understand pathological aspects of the circulatory system, especially those occurring in failure of the heart and the respiration. In this chapter, we will present the chronological developments in the Donders model, and illustrate technical solutions which have been incorporated into the model over time.

Models of the circulation have been under development since Euler (1775), who attempted to predict the pulse wave velocity in an artery, but was unable to solve the two underlying differential equations. A century later, Weber (1850), who became interested in the role of the volume of circulating blood, actually built a model. This activity reached a zenith in 1972 with the publication of Guyton et al.’s model of circulatory regulation and control, containing over 350 mathematical equations. The size of this model triggered a period of introspection.

Since the closed loop circulation contains blood conducting pathways as well as blood pumps, a number of physical quantities play key roles. These include inertial and viscous properties of blood, the distensible properties of blood vessels and the time-varying contractile and relaxing properties of pumps. These parameters are related in equations which result in pressure, flow and volume phenomena, all of which vary with time. Preceded by years of research into the conduct of the arterial system, initial modern descriptions included no less that 200 simultaneous differential equations, requiring computer assistance to solve. This could, with the introduction of Zo, be reduced to three element modeling ‘units’ with the characteristic impedance Z0, the total compliance of the system C, and the peripheral resistance Rs. This reduction could also be performed for the pulmonary circulation, and the three-element approach could be retained as the two circulations became a closed loop. Expansion include the ‘ejection effect’ kappa, which reduces ventricular pressure during ejection and magnifies it during ventricular relaxation, as well in inclusion of the shapes of the ventricular ejection curve and pressure was, based on work by Mulier et al. early in 1998. The equations establish the Donders model. The solutions to these equations furnish the material intended to help understand pathological issues.
The Donders model, emerging from its arterial origin, has evolved to its fourth version. The first version (DI), written in Mathematica, was restricted to the systemic part of the circulation. (Figure 2) It encountered the problem of negative volumes, also found by other investigators, who did not remedy it, and introduced the basics in the concept of sloshing when the asystolic left ventricle was massaged in accordance with cardiac pump theory (Chapters 3, 8, 9).8 The cause of the negative volume issue was identified, and for the first time, explicitly addressed but without reconciling this with cardiovascular or anatomical reality.

The second version (DII) comprised the closed loop of the human circulatory system, which could function physiologically under normal conditions. (Figure 3) Addition of a number of physiologic controls allowed for exercise conditions and enlarged cardiac output, as well as validation of the model as a physiological reality. Impedance-defined flow, ejection effects, as well as respiration as an outside agency operating on the circulation (per) were added as new features. Limitations to cardiopulmonary resuscitation were investigated, with specific attention to those caused by collapse and sloshing (Chapter 9).9

The third version, DIII, converted to Matlab, refined its predecessor significantly, by updating programming adding compliance of the more extensively modeled veins (C) to the parameters and allowing a decreased manipulation of the values.10 The third version includes a potential solution for negative volumes, elimination of some anatomical uncertainty, elimination of “time compression” with the ventricular ejection effect and more elaborate modeling of chamber dynamics, which suppressed resonances of short duration. In its current form, the model has returned conservation of volume to a physiological approach, allowing analysis of the conduct of different central veins to become a focus. (see for diagram the Appendix)

In summary, the Donders model, in its evolutionary states (Appendices I, II, III representing D-I, D-II and D-III, respectively) embodies aspects of a resistive-inductive-capacitive model with control features into a system of equations, describing basic functions of the human circulation. In this chapter the developments incorporated into the major segments of the circulation and their associated equations are described, including a number of its novel features. Where possible, validation is carried out. The equations are listed in each Appendix with default parameter values. Focus is placed on Donder DII (Appendix II) since this represents the work presented in Chapter 8.

6.2 General anatomic and symbolic layout of the Donders model (DII)

In the central part of Figure 6.1, the cardiac chambers can be recognized easily, with the pulmonary circulation between the right ventricle and the left atrium. The coronary circulation, modeled as an arterial system and a compressible venous system separated by a variable resistance, representing the coronary microvasculature, can also be identified. The systemic arterial system begins on the far right, leading to the abdominal vasculature, the legs and to the vasculature above the thorax as arm and cerebral circulations. The principal arteries appear separately. The vasculatures supplied by the systemic system lead eventually into the venae cavae.
Chapter 6: Layout and description of the Donders model and its progression

122

Figure 1:

Brief overview of major vascular structures useful in resuscitation medicine.

The Donders model, emerging from its arterial origin, has evolved to its fourth version. The first version (DI), written in Mathematica, was restricted to the systemic part of the circulation. (Figure 2) It encountered the problem of negative volumes, also found by other investigators, who did not remedy it, and introduced the basics in the concept of sloshing when the asystolic left ventricle was massaged in accordance with cardiac pump theory (Chapters 3, 8, 9). The cause of the negative volume issue was identified, and for the first time, explicitly addressed but without reconciling this with cardiovascular or anatomical reality.

Figure 2:

DI as an prototype for the essential aspects in chest compressions based on the Cardiac pump theory.

The second version (DII) comprised the closed loop of the human circulatory system, which could function physiologically under normal conditions. (Figure 3) Addition of a number of physiologic controls allowed for exercise conditions and enlarged cardiac output, as well as validation of the model as a physiological reality. Impedance-defined flow, ejection effects, as well as respiration as an outside agency operating on the circulation (per) were added as new features. Limitations to cardiopulmonary resuscitation were investigated, with specific attention to those caused by collapse and sloshing (Chapter 9).

Its third version, DIII, converted to Matlab, refined its predecessor significantly, by updating programming adding compliance of the more extensively modeled veins (C) to the parameters and allowing a decreased manipulation of the values. The third version includes a potential solution for negative volumes, elimination of some anatomical uncertainty, elimination of “time compression” with the ventricular ejection effect and more elaborate modeling of chamber dynamics, which suppressed resonances of short duration. In its current form, the model has returned conservation of volume to a physiological approach, allowing analysis of the conduct of different central veins to become a focus. (see for diagram the Appendix)

In summary, the Donders model, in its evolutionary states (Appendices I, II, III representing D-I, D-II and D-III, respectively) embodies aspects of a resistive-inductive-capacitive model with control features into a system of equations, describing basic functions of the human circulation. In this chapter the developments incorporated into the major segments of the circulation and their associated equations are described, including a number of its novel features. Where possible, validation is carried out. The equations are listed in each Appendix with default parameter values. Focus is placed on Donder DII (Appendix II) since this represents the work presented in Chapter 8.

6.2 General anatomic and symbolic layout of the Donders model (DII)

In the central part of Figure 6.1, the cardiac chambers can be recognized easily, with the pulmonary circulation between the right ventricle and the left atrium. The coronary circulation, modeled as an arterial system and a compressible venous system separated by a variable resistance, representing the coronary microvasculature, can also be identified. The systemic arterial system begins on the far right, leading to the abdominal vasculature, the legs and to the vasculature above the thorax as arm and cerebral circulations. The principal arteries appear separately. The vasculatures supplied by the systemic system lead eventually into the venae cavae.
superior and inferior. In version DIII (anno 2008), the venous system has been more extensively modeled such that sloshing effects and volumes shifts can be followed. The description is based on DII.

Ambient pressure around most vessels is at the barometric level, set here at zero and furnished with the “ground” symbol. This applies also to the extra cardiac connections. Where other conditions prevail or deviation is mandated, a facility, indicated by an open circle in Figure 6.1c is available to introduce an external pressure. Local internal pressures are indicated by p(t), each with an index to denote its locale, and flows by Q(t) also with an index, along an arrow indicating the direction defined as positive flow. ‘Vertical’ flows (QV) relate to the filling or emptying of a compliance. ‘Horizontal’ flows (QH) refer to blood moving from one compartment of the circuit to another. The volume in a particular compartment may be thought of as the sum of two volumes: the “dead” or filling volume, Vd, causing pd = 0, and a time dependent volume V(t). Their sum equals zero when the compartment is collapsed. V can never go negative. Volumes in the different parts of the circulation include 60 ml in the right and left atria (V4 and V8), and circa 150 ml in the right and left ventricles (V5 and V9), as determined at end diastole, by:

Solving for V(t):

\[
p(t) = a [V(t) - b]^2 \quad \text{(eq. 6.1)}
\]

\[
V(t) = b + \sqrt{p(t)/a} \quad \text{(eq. 6.2)}
\]

where \( p \) is 15 mm Hg, ‘a’ the coefficient related to the compliance, as specified per chamber, and \( b \) is the volume at which the transmural pressure is zero, clinically described as the ‘dead volume’). \( V_d i \) (i = 1 through 13) describes the dead volumes in the compliances spread throughout the circuit. The total blood volume is 5.3 L. Of this (\( C_2 \) and \( C_6 \)) 120 ml of volume fill the systemic and pulmonary arterial beds (see Chapter 2) Total blood volume in the extra cardiac compliances is computed from:

\[
V = \sum [C_i p_i + V_{ai}] \quad \text{(eq. 6.3)}
\]

Note that the pressure-volume relationships in passive cardiac chambers are non-linear (quadratic, eq 6.2) and therefore must be calculated separately. The arterial compliances were chosen from available literature. Those for the venous compliances were assembled from the general literature, as no one source presents this data.

### 6.3 The cardiac chambers

The equations for the cardiac chambers recognize both diastolic and systolic phases by using separate terms. For practical, experimental, and clinical practice, systole is defined as the period during which force and pressure are built up within the pump. It continues up through the moment of maximum energy contained by the pump, or in CPR, by its replacement \( p_e(t) \). Diastole fills the rest of the cycle, including some residual crossbridges built during the active ventricle, but without pressure build up, and also includes a period of inactivity. This is a more specific definition then the commonly used measure at the dicrotic notch (flow = zero), which is later in time, and an external measure. (Figure 4: normalized \( f(t) \), with extra symbols for systole and diastole) These definitions are also applicable to CPR, (Figure 5) where the form of the curves (i.e. sinusoidal compressions with more of less continuous motion, and more square waveform with both a rapid increase and release of pressure as well as hold cycle during compression and release influence both potential trauma and blood flow.

\* i.e. the transmural pressure associated with a given dead volume
The diastolic portions are described in the first term on the right in eqs (6-4) to (6-7). These are followed by a second term which describes contraction of the chamber by a product of two functions: the first is a normalized function \( f(t) \) covering the time aspect (Appendix II,b) where \( t_d \) is the end of the build-up of energy and the period \( t_d \) to \( t_h \) defines the period to the beginning of the next beat or compression. The second part is a function of the form

\[
\frac{cV(t) - d}{a}
\]

which accounts for volume aspects. The products appear in eqs. (6.6) and (6.7).

In eq. (6.5), \( c \) and \( d \) are parameters and \( V(t) \) is the time dependent volume variable. These formulas apply to both atria, the indices distinguishing the individual characteristics. The \( f(t) \) function describes the making and breaking of cross-bridge bonds by exponential functions, the build up with a time constant of \( \tau_c \), the relaxation with a time constant \( \tau_r \). The delay between the commencement of the two processes is denoted \( \tau_d \). The description of the properties of a contracting/relaxing cardiac chamber is based on experimental work in isolated animal hearts.\(^{12,13}\)

Ventricular contraction follows atrial contraction after a time delay. In addition, for the ventricles, a so-called ejection effect is embodied, which modifies \( f(t) \) to \( F(t) \) [eqs. (6.5) and (6.7)].\(^{14}\) The ejection effect itself consists of the sum of two terms: a pressure reducing term during the major part of the ejection phase \([ -k_1Q_{in}(t) \text{ for the right ventricle} \] and \([ -k_1'Q_{in}(t) \text{ for the left ventricle} \]) followed by a delayed (by a time interval \( \tau \)) pressure augmenting term during late systole \([ +k_2Q_{in}(t-\tau) \text{ and } +k_2'Q_{in}(t-\tau) \text{ for the same ventricles} \]). All \( k \)'s are parameters. The first terms (with \( k_1 \) and \( k_1' \)) are analogous to the Hill effect: shortening muscle becomes weaker and are called deactivation.\(^9\) The \( k_2 \) terms compensate for the pressure loss by hyperactivation.
Furthermore, just like the chambers’ performances are sensitive to their internal (blood) pressures, they are sensitive to their external pressures, denoted $p_e(t)$. This is implemented on the left side of eqs. (6.5) to (6.8). Pressure $p_e$ shows a minus sign since it operates on the outside of all cardiac chambers, while the quantity itself may be positive or negative. Consequently, the cardiac function curves became sensitive to Frank-Starling effects (preload and arterial load) augmented with a pressure-autoregulation [so-called in analogy to homeometric and heterometric autoregulation, but imposed by $p_e(t)$].

It is convenient to distinguish $p_e(t)$ into two cases, one caused by respiration, denoted $p_{d}(t)$, which itself is negative most of the time, the other, denoted $p_{b}(t)$ by chest compression as applied in CPR, which itself, is zero or positive.

\[
\begin{align*}
\text{RA:} & \quad p_1 - p_3(t) = \pm a_{RA}(V_4 - b_{RA})^2 + (c_{RA}V_4 - d_{RA}) f_{RA}(t) & (\text{eq. 6.5/ I.77}) \\
\text{RV:} & \quad p_2 - p_4(t) = \pm a_{RV}(V_5 - b_{RV})^2 + (c_{RV}V_5 - d_{RV}) f_{RV}(t) & (\text{eq. 6.6/ I.78}) \\
\text{LA:} & \quad p_{501} - p_6(t) = \pm a_{LA}(V_8 - b_{LA})^2 + (c_{LA}V_8 - d_{LA}) f_{LA}(t) & (\text{eq. 6.7/ I.80}) \\
\text{LV:} & \quad p_8 - p_6(t) = \pm a_{LV}(V_9 - b_{LV})^2 + (c_{LV}V_9 - d_{LV}) f_{LV}(t) & (\text{eq. 6.8/ I.81})
\end{align*}
\]

It should be noted also that the first terms on the right of the same equations are preceded by a ± symbol. This arises from the nonlinear properties of the atria, shown by the exponent. For $V > b$ in any of these four equations, there is no difficulty and the + sign applies. However, for $V < b$, atrial compliance would become negative, an unrealistic phenomenon, unless the minus sign is used instead.

This is just one example of physical impossibilities against which the mathematics does not guard. Another one of relevance here is that for positive external pressures ($p_e$) exceeding internal pressure for any compartment, the volume $V$ of such a compartment may become negative according to its governing equation. Since this is also physically impossible, the computer was instructed to take measures to avoid such effects.

**The atria:**

The systolic functions represented in the equations defining the contraction of the atria as well as the ventricles, recognize that two phases exist. The building of the bridges as the initial phase of systole (eq. I.85) and the reduction of bridges (eq. I-86) a process towards diastole; diastole being defined as total inactivity of actin-myosine bridge movement. The transition between eq.I.85 and I.86 is determined by $t_d$ which is defined in eq. I.87. The duration of the complete beat is defined as $t_0$.

The exponential function $(1 - e)^{-t/(t_0)}$ describes the upstroke of contraction, with $e^{-t/(t_0)}$ describing the downstroke. This description of the properties of a contracting cardiac chamber is based on experimental work in isolated animal hearts. This paper is first in the further demonstration of its characteristics.

The symbols $t_0$ designates the time constant for the progression of actin-myosin bridge building, $t_d$ designating the relaxation phase, $t_0$ is the time that the number of actin-myosin bridges is maximal, and $t_d$ is the moment that bridge release is initiated, as specified in eq. I.87. $\alpha$ contributes to the speed of the actin-myosin bridge building and release, in order to allow for more rapidity.

**The ventricles:**

The effects described above all carry over to the ventricles, with an added factor. The equations for the ventricles are eqs. I.77 and I.81, with eqs. I.79 and I.82 describing the $F$ functions in the
first two equations. The addition is the ejection effect, incorporated in the latter two equations. In the model, during the first two stages, the ejection effect, operating on the ventricular ejection flow \(Q_{ch}\), consists of two expressions with \(k_1\) and \(k_2\) as positive coefficients. The first term \(k_1\) (preceded by a negative sign) is analogous to the Hill effect (shortening a muscle causes it to be weaker) as applied for cardiac muscle fibers, describing the loss of cross bridges due to muscle shortening in contraction as results at the physical movement. The loss of energy is corrected by the \(k_2\) term.\(^{16}\) This effect only operates during ejection. The second term, \(k_2\), introduces the reestablishment of the stored energy into the contractile force during a later phase of the contraction, as influenced by \(\kappa\). This kappa is the time delay involved in the reestablishment of these bridges, based on experimental evidence.\(^8\)

Visually, if a suspended ventricle were allowed to fill and then be stimulated, pumping blood into the circulation including its peripheral resistance, the eqs. I.79 and I.82, would calculate the actual pressure in the ventricular cavity. During the initial phase \(k_1\) corrects for the divergence of the curves in which the pressure in the ventricle is actually lower than that projected by the equations I.78 and I.81 without the ejection effect. During the later part of ejection the pressure in the cavity decreases less than the equation projects, reflected by reestablishment of high energy bridges, leading to the incorporation of the \(k_2\) term.

On the left hand side in the four chambers, \(p_e\) appears. The pressure \(p_e(t)\) is imposed by either the respiratory system (and is primarily negative) or by the CPR (which is zero or positive).

For negative \(p_e(0)\) the curve shifts to the left, for positive to the right. This is referred to as horizontal shift (along the volume axis). When the heart receives sympathetic stimulation the factor “c” increases and the cardiac function curves become steeper and taller (vertical shift). These shifts modify filling of the chambers and hence their performance as pumps. The final terms in eqs. I.79 and I.82 reflect the mathematical symbolic approach to repetition of beats.

### 6.3.1 Negative volume in the specific case of cardiac chambers

An exceptional phenomenon, reflecting on the difference in electrical and/or mathematical reality and clinical, medical, reality, appears in the form of negative volume.

In the equations I.77, I.78, I.80 and I.81 (Appendix II), the left hand term reflects the potential impact of \(p_e\). The equations permit \(p_e\) to become higher than cavity pressure, even though the compliance of the cavity wall is incorporated in the equations (coefficient “a”). This is reflecting in that while the pressure difference may be negative, initially volumes are not. When volume become negative (to the left of the vertical axis), a clinical impossibility is introduced, caused by \(p_e > p_c\) the cavity pressure. If this is allowed volume preservation within the model is no longer maintained. Requiring the model to ‘set’ the volume to zero introduces a discontinuity, as well as an instantaneous numeric invalid solution. Iteration magnifies even small inconstancies allowing the model to ‘overfill’ or ‘bleed to death’.

While this phenomenon is mathematically and electrically understandable, clinically, it can also be recognized. The different cardiac chambers and more in general the venous vascular, set different boundary conditions for \(p_{ei}\). The application of one \(p_{ei}\) during external chest compressions on the thoracic cavity and its enclosed organs may inadvertently cause local “negative” or “locked” volumes. The effect is used during transthoracic echocardiographic investigations to evaluate the function of the right heart (i.e. does the inferior vena cava close during inspiration at the level of the diaphragm). Note that spontaneous respiration is unlikely to cause this effect on the (typically a negative \(p_e\) as long as the ‘airway’ in open, while mechanical ventilation is more likely to have this effect (completely positive \(p_e\)).

Equation I.88 addresses the issue of negative volumes. Originally, in order to prevent negative volumes, specific resistors (R) were introduced at the inlet and outlet of the cardiac chambers.
Subscripts have been added to indicate positioning. These resistors are negligibly small when cavity volumes are positive and becomes infinitely large when negative volume threatens. This is defined by the right hand term in eq. I.88. Since the defining term in eq. I.88 is cavity volume, reopening of the cavity is operated on by flows, defined by other equations.\textsuperscript{7} However, this remedy proved cumbersome as the program running time increased. Switching from Mathematica (v.5.2, Wolfram Research Ltd, Oxfordshire, UK) to Matlab (The Mathworks, Inc, Natick, MA), allowed the requirement to be simply stated as volumes cannot go negative, a mathematically convenient, though physiologically unrealistic solution.

### 6.4 Valvular opening and closure

The valves have been modeled to open when the pressure gradient across them becomes positive (for example eq. I.32). The valves will close when flow through the ostium becomes negative, with 5% of the forward flow being allowed as retrograde flow as part of valvular closure, after which eq. I.33 applies.

Nieman’s valve (N), suggesting a functional valve at the superior thoracic outlet, is incorporated in the model, in addition to a representative valve in the peripheral vasculature. Early work (Gray’s Anatomy, Anatomy of the Human body, p. 167, 1918) suggest that their may be more distal valves in the jugular sinus. Their opening is also pressure driven, and closure is flow driven.

### 6.5 Arterial systems

The arterial system is a reduced system with a distributed peripheral resistance, lowering the number of equations to less than 10 from the original several hundred. The validity of the reduction has been demonstrated previously.\textsuperscript{11} Unique is the facility to apply p\textsubscript{ec}. The reduction applies to the input impedance and comprises the characteristic impedances (Z\textsubscript{os}), the compliance (C\textsubscript{10}) and distributed peripheral resistances (R, Appendix II). The characteristic impedance of the systemic circulation is divided to allow inclusion of the coronary circulation. The resistors for the coronary circulation (R\textsubscript{coa} and R\textsubscript{cov(t)}) were set to approach normal, documented, flows.

The pulmonary input impedance displays a characteristic Z\textsubscript{op} and more detail than its systemic counterpart in a successful effort to prevent disturbing resonance phenomena. The values for the different descriptors were extracted from the literature and validated for humans.

In eqs I.19 through I.31 the volumes (V\textsubscript{x}) in the capacitors are related to the in- and outflows (Q\textsubscript{i}), with any of those capacitors.

### 6.6 Venous systems

The elaborate venous system is represented as distributed over the various regions and lumped within a region. The latter is represented by standard resistive-inductive-capacitive (RLC) circuits. The critical component is the compliance. Since the venous vessels are wider than their companion arteries, while their walls are thin and easily distensible, the most striking features of the venous systems are the large fraction of total blood volume they usually store, in the order of 60%, and their collapsibility. A characteristic quantity to describe the filling situation at any point in time is the transmural pressure p\textsubscript{t}(t), defined as the internal (blood) pressure minus the external pressure. The volume veins contain at p\textsubscript{t} = 0 is called the filling volume, or dead volume V\textsubscript{d}. Above this volume level, the additional volume, now a function of venous pressure, may be calculated from

\[ V(t) = Cp(t) \]  

(eq. 6.9)
where $C$ denotes the compliance of the veins, frequently treated as a constant. The volume-pressure relation is then linearized. In reality, venous compliance displays two different types of nonlinearity. The concept of transmural pressure equal to zero also serves to distinguish the two: for $p > p_c$, the vessel wall’s nonlinear elastic properties emerge, for $p < p_c$, the collapse phenomenon appears.

For the former condition, the compliance per unit length, $C'$ equals

$$C' = 2\pi K \left[r(0)^3 + 3Kp_c\right]^{1/3} \quad \text{(eq. 6.11)}$$

With

$$K = [3r(0)^4]/[4h(0)E(\tau(0))] \quad \text{(eq. 6.12)}$$

In which $h$ = wall thickness, $E$ is Young’s modulus of the wall material, and $(0)$ at $p_c$ is zero.

For the latter condition, nonlinearity due to (partial) collapse is far more pronounced. Normalized compliances are published by Kresch et al. They display a maximum in compliance close to zero transmural pressure. These nonlinearities are now incorporated in the Donders model (D III/IV).

Currently, the (constant) values used were, as far as possible, extracted from the literature, with further developments made internal to the model.

### 6.7 Controls

Five controls, some manifesting more than one facet have been identified that influence cardiac output. These controls are sympathetic stimulation, which operates on the contractile properties of the cardiac chambers. It modifies the cardiac function curves by rotating them counterclockwise and stretching them along the output axis. These are embodied by the coefficients ‘c’ in the chamber equations (eqs. I.77, 78 and I.80, 81). The control mechanism of neural stimulation of heart rate is embodied by an adjustable ‘$\beta$’. The respiratory influences (depth and frequency), an impedance-defined flow, on the cardiac chambers, modifying the cardiac function curves by shifting them back and forth along the preload (filling) axis. This is included in the chamber equations where $p_c(t)$ appears (eqs. I.77, 78, 80 and 81). Finally, baroreceptor control of the systemic (‘$R_s$’) and pulmonary (‘$R_p$’) sides of the circulation. If cardiac output would, for example, triple as a result of exercise, arterial pressure would also if the peripheral resistance remained unchanged. In the circulation, baroreceptors, embedded in some of the systemic arteries, report arterial blood pressure levels to the central nervous system, its neural response adjusting the level of the peripheral resistance to undo most of such effects. In such instances transfer of blood from the arteries into the veins is facilitated.8

An additional control, not included in the model but recognized in the human circulation is that of venomotion, another impedance-defined flow phenomenon. This incorporates pumping executed by the small muscular venules in the peripheral vasculature, most likely due to local metabolic control.59 Venomotion, has not been incorporated in the Donders model yet, owing to its provisional character.

### 6.8 Adaptations to CPR

The cardiac chambers can be made asystolic by setting $F(t) = 0$ for the ventricles and $f(t) = 0$ for the atria. In selected, or on all open circles of Fig. 1 chest compression, $p_{ec}(t)$, can be introduced. The actual value of $p_{ec}(t)$ is the externally applied pressure, corrected for the attenuation imposed by the rib cage. The characteristics of the chambers, are, under these circumstances, defined by
their diastolic properties. These properties \((a\ and\ b,\ eq.\ 6.1)\) can be altered to allow for the effects of changing myocardial stiffness as clinically observed.\(^{20}\)

An unexpected effect, puzzling for some time, was the appearance of negative volumes when CPR was administered. This imposed large variations in the total blood volume contained by the Donders model. This artifact is caused by keeping compliance values constant, while in reality they can vary over a wide range, and most importantly to zero, when the vein is completely flattened. The underlying clinical effect, of more than sufficient intramural pressure to completely seal, or lock, volume into a vascular (venous) segment, seems to be an important, underlying, but as yet underexposed, effect in CPR.

### 6.9 Initial conditions

The model is ‘filled’ with blood, generating equal pressures in all parts of the circulation. The pressure for initial conditions is a parameter, the value of which can be adjusted. The default value is 15 mm Hg. For initial conditions the status of the cardiac valves is indifferent: there is neither reason for them to be open nor to be closed as there are no pressure gradients and no flows. The function \(p_e(t)\), generated by the respiration or by CPR, is turned off, \(f(t)\) and \(F(t)\) are set equal to zero, making all cardiac parts of the circuit to be defined by their diastolic properties.

### 6.10 Summary

The equations for the cardiac chambers recognize diastolic and systolic phases. In the atrial equations (I-77 and I-79) the right hand side’s second term contains a function \(f(t)\) which covers contraction and relaxation. Likewise the ventricular equations (I-78 and I-80) display second terms on the right side that contain a function \(F(t)\), which describes contraction, relaxation, and ejection effects.

For CPR studies, the atrial and ventricular equations are obtained by setting the functions \(f\) and \(F\) equal to zero, which deletes the contractile phenomena and only the first terms on the right remain.
6.11 References

12 Mirsky I, Scott Rankin J. The effects of geometry, elasticity, and external pressures on the diastolic pressure-volume and stiffness-stress relations, How important is the pericardium. Circulation Research. 1979 May; 601-611;44(5)
Chapter 6: Layout and description of the Donders model and its progression
Modeling in Cardiopulmonary Resuscitation: Pumping the heart
Abstract

Physiology offers insights into fundamental aspects of the circulation, even when the circulation is non-physiological, as during cardiac arrest and cardiopulmonary resuscitation (CPR). Human and animal experiments offer only limited insights as they are susceptible to uncontrollable variables. Mathematical models offer quantitative results for flows, pressures and volumes under clearly defined conditions, chosen by the experimenters.

This report describes the left ventricle and its immediate environment as the core of a large mathematical model. The model works normally under physiological conditions, and is specifically designed to allow understanding of flow, pressure, and volume phenomena under CPR as an extreme pathophysiological scenario. Using impedance defined flow the importance of valves for both the contracting as well as the asystolic ventricle is quantified. It demonstrates the role of venous pressure, sloshing of blood and flow around the cardiovascular circuit. The flow of 8 ml/sec, in a pathological situation is demonstrated to be due not to cardiac compression, but to venous pressure.

The principal conclusions are that the model functions in the physiological situation and that in an asystolic left ventricle with competent valves, intrathoracic pressure can effectively replace contractile properties. In an asystolic ventricle without competent valves, intrathoracic pressure variations accomplish little. The cardiac pump theory has limited applicability in CPR.
Abstract

Physiology offers insights into fundamental aspects of the circulation, even when the circulation is non-physiological, as during cardiac arrest and cardiopulmonary resuscitation (CPR). Human and animal experiments offer only limited insights as they are susceptible to uncontrollable variables. Mathematical models offer quantitative results for flows, pressures and volumes under clearly defined conditions, chosen by the experimenters.

This report describes the left ventricle and its immediate environment as the core of a large mathematical model. The model works normally under physiological conditions, and is specifically designed to allow understanding of flow, pressure, and volume phenomena under CPR as an extreme pathophysiological scenario. Using impedance defined flow the importance of valves for both the contracting as well as the asystolic ventricle is quantified. It demonstrates the role of venous pressure, sloshing of blood and flow around the cardiovascular circuit. The flow of 8 ml/sec, in a pathological situation is demonstrated to be due not to cardiac compression, but to venous pressure.

The principal conclusions are that the model functions in the physiological situation and that in an asystolic left ventricle with competent valves, intrathoracic pressure can effectively replace contractile properties. In an asystolic ventricle without competent valves, intrathoracic pressure variations accomplish little. The cardiac pump theory has limited applicability in CPR.

Table of contents

7 MODELING IN CARDIOPULMONARY RESUSCITATION: PUMPING THE HEART ... 133

7.1 Introduction ............................................................................................................................. 136

7.1.1 Models in the circulation ................................................................................................... 136

7.2 Methods and procedures.......................................................................................................... 137

7.3 Results ..................................................................................................................................... 139

7.4 Discussion ............................................................................................................................... 148

7.5 Conclusion ............................................................................................................................... 152

7.6 References ............................................................................................................................... 153
7.1 Introduction

Modeling of the circulation has a long history, dating back to 1776; its application to cardiopulmonary resuscitation has enjoyed a much shorter life. This limitation is due to the still prevailing partial understanding of the circulatory system under the best of conditions. Under extreme situations, the challenge to describe the system quantitatively, i.e., perform modeling, remains difficult. Modeling can be divided into three major approaches: fluid-mechanical, electrical, and mathematical modeling. Each will be discussed briefly.

The purpose of this study is twofold: (a) to determine whether the steady or the oscillatory terms of the respiratory system are capable of influencing cardiac output. In a preliminary study it was found that for the case of small sinusoidal modulation, to avoid venous collapse, and an integer ratio of heart and respiratory rates, the difference between the inspiratory effect and the expiratory one cancelled one another so that no net effect on cardiac output accrued (b) to determine the magnitude of cardiac output alteration as a function of the depth of respiration. To define conditions accurately, this will be a model-based study, which features a closed circulatory system. The respiratory system will interact with the circulatory system primarily at the level of the vena cava superior and inferior, the pulmonary veins, and both atria, all in the thoracic cavity.

The application of external cardiopulmonary resuscitation (CPR), as suggested by Kouwenhoven et al., has for the most part remained unchanged, despite an impressive - cardiac resuscitation (OCCR) to closed-chest cardiac resuscitation (CCCR), a non-invasive technique, coincided with a seemingly significant downturn in the survival rate. This raised a warning sign for other investigators with respect to Kouwenhoven’s assumption that CCCR was a straight imitation of OCCR. The steadfast nature of the CCCR technique has, for the most part, been caused by difficulty in achieving reproducible results in research though it includes 25 years of discussion into the fundamental distinctions between the ‘cardiac’ pump theory, and its younger competitor the ‘thoracic’ pump theory. In this theory, intrathoracic pressure changes are thought to cause forward blood flow due to collapse of thin-walled vessels at the thoracic outlets, with little concern about continued blood supply. Another aspect contributing to the dearth of fundamental change could be the use of less suitable animal models for physiological analysis. In the mean time, the survival rate remains at an unchanged 5-10%.

Kouwenhoven suggested that compression on the sternum would decrease the anteroposterior diameter of the chest, and force the heart (e.g., the left ventricle) against the spine. This would, in the presence of a competent mitral valve, cause forward blood flow, allowing myocardial and cerebral oxygenation. Release, as artificial diastole, would allow for ventricular filling. Currently, the chest is compressed 80-100 times min⁻¹, with a force of up to 70 kg yielding an extrapolated sternal displacement of 4-5 cm. In the 1980s, in the face of an ongoing fundamental discussion of ‘cardiac’ versus ‘thoracic’ pump, the development of adjuncts, such as active compression-decompression cardiopulmonary resuscitation (ACD-CPR) techniques generated interest in the use of analytical procedures (models). However, as Babbs et al. noted in their review, these models never achieved the status of serious original contributions, and played only a nominal role. Retrospectively, an important opportunity for gaining fundamental physiological and pathophysiological insights into CPR may have been overlooked.

7.1.1 Models in the circulation

The design of models of the closed cardiovascular loop is rooted in 1850, when Weber published his fluid dynamic model, primarily for teaching purposes. Electrical, then mathematical models followed in due course, sometimes reaching high levels of complexity.
Model making in CPR was initiated in the 1980s and follows, in large part, the same line: fluid-mechanical models for illustrative purposes, and distributed electrical and mathematical models for analytical purposes. These models cover the vasculature at different levels of detail, but, unlike their predecessors have not been reported with validation under physiological conditions, a traditional prerequisite. The models rarely featured a heart that can contract and relax. Instead, modeling was utilized predominantly to investigate or support the effectiveness of adjuncts to standard CPR. Landmarks are the addition of abdominal counterpulsation and the effect of variation of intrathoracic pressure accompanied by phased chest and abdominal compressions or fixation. Halperin, evaluating the vest as a viable option to chest compressions, changed compression frequency, force and duration as major criteria to elucidate the pump mechanism. Using Babbs’s model, the effects of arterial and venous volume loading were re-evaluated by Tomaszeski et al.

All of these interventions resulted in pressure, volume and flow alterations at multiple sites in the closed loop. For clinical, physiological and more specifically CPR-related practitioners, these models were difficult to comprehend. A number of factors may have contributed to this, such as the use of clinically unfamiliar electrical analogs, the introduction of parameter values which for the most part have not been defined in the clinical setting, and their use to support techniques superimposed on standard CPR as opposed to their use for fundamental work.

In the present study, a model has been developed and analyzed for the express purpose of promoting understanding of pressure and flow phenomena during CPR as observed in a wide range of experimental and clinical reports. We concentrate on the core of a larger model. This report will focus on computer experiments on the left ventricle provided with a preload and an arterial load. Pressures, flows and volumes will be displayed and analyzed. Negative volumes, a modeling artifact that has anecdotally appeared for at least half a decade will also be discussed with presentation of a method to guard against this flaw. Current clinical concerns and developments will be touched upon.

### 7.2 Methods and procedures

In this report the focus is on the left ventricle, equipped with adjustable preload and arterial load, a small but vital part of the circulatory system. It will be modeled quantitatively, first for control conditions, then under various pathological conditions related to CPR, including cardiac arrest status in which the myocardial compliance changes, as seems to occur during progression of CPR. The motive for performing a model study is to have access to experiments not executable in the human, and serve to gain insight into classic as well as adjunct theories for CPR.

Under control conditions, a number of routine responses of the ventricle are demonstrated as validation of its physiological properties. Three pathological circumstances, a non-contracting ventricle with competent valves under application of external compression, a normally contracting ventricle, operating as an open conduit, and a non-contracting ventricle operating as an open conduit with external compression are reported.

The model is a reduced, lumped parameter model, and is represented as a mixed system of differential-algebraic equations (DAE). This system of DAEs has been solved with Mathematica. The core model consists of 12 variables, including seven flows and four pressures. Figure 1A shows the model utilizing fluid mechanical symbols, Figure 1B using the more specific electrical symbols. Similarity in vascular layout is evident among the models listed above and the core of the model presented here.
Particular attention will be devoted to the role played by the recently discovered principle of impedance defined flow. This principle holds that blood flow around the cardiovascular circuit can be generated by any of its compressible or distensible parts. The mechanism can be contraction and relaxation, compression and release (e.g., CPR), shaking the body, or changing the gravitational field. The theory holds for conditions with and without functional valves, as well as making a distinction between sloshing of blood and actual effective forward flow, allowing the quantification of antegrade and retrograde stroke volumes over any valve or valve ostium. Stroke volume can thereby be defined in the classic sense, while antegrade volume is defined as the area under the positive part of the ejection curve, retrograde flow being the area under the negative part of the injection curve. These distinctions led to appreciation of the difference between sloshing of blood and flow around the cardiovascular circuit.

The left ventricle (Figure 1A) is identified in conjunction with its preload in the form of a constant pressure source, $p_{\text{ven}}$, implying a large venous reservoir, such as occurs in a normo- to hypervolemic, supine human, and an arterial load represented by the three element modified windkessel to the far right of the ventricle. The mitral and aortic valves, modeled as free-flowing two-dimensional leaflets are marked, as is the facility to apply periodic external compression and relaxation, $p_{e}(t)$. The valves are not modeled as electrical diodes in order to allow internal definition of movement. For simplicity, the external pressure is interpreted as acting directly and selectively on the ventricle.

The same features appear in Figure 1B, with $R_{LV}$ denoting a (small) inflow resistance, $Z_{oa}$ the characteristic impedance of the aorta, $R_s$ the systemic peripheral resistance and $C_{10}$ the arterial compliance. Pressures are marked $p$. Flows, $Q$, with their positive directions, are defined in each

**Figure 1A:** Fluid mechanical representation of the left ventricle with preload ($p_{\text{ven}}$ at the far left), and arterial load (the three elements on the far right), plus a facility to compress the ventricle ($p_{e}(t)$). $V_{LV}(t)$ denotes left ventricular volume at time $t$, and $R_{LV}$ a ventricular inflow resistance. For the function of the two variable resistors marked $R_{LV}$, consult the caption of Figure 9.

**Figure 1B:** Electrical representation of the fluid mechanical model in Figure 1A with the valves identified by name. Local flows $Q$ are marked with the arrows indicating their positive directions.

**Figure 2:** Ventricular pressure ($p_6(t)$, solid) and outflow through the aortic valve ($Q_{H15}(t)$, dashed), with the ejection effect. Stroke volume $V_sLV$ is 64 ml, ejection fraction is 52%, venous pressure is 10 mm Hg, and $R_s$ is 1.2 mm Hg sec ml$^{-1}$. The current model continues where the former is restricted to the introduction of the ejection effect into the outflow curve, incorporating this into the theory of impedance defined flow. The ejection effect embodies the influence of outflow on ventricular pressure. Its inclusion tends to make both pressure and flow downstrokes more convex. The stroke volume is 64 ml, the ejection fraction 52%. The summaries of a preload ranging from 4-20 mm Hg and of peripheral resistance changes from 0.6 to 2.2 mm Hg sec ml$^{-1}$ are shown in Figures. 3A and 3B.
Particular attention will be devoted to the role played by the recently discovered principle of impedance defined flow. This principle holds that blood flow around the cardiovascular circuit can be generated by any of its compressible or distensible parts. The mechanism can be contraction and relaxation, compression and release (e.g., CPR), shaking the body, or changing the gravitational field. The theory holds for conditions with and without functional valves, as well as making a distinction between sloshing of blood and actual effective forward flow, allowing the quantification of antegrade and retrograde stroke volumes over any valve or valve ostium. Stroke volume can thereby be defined in the classic sense, while antegrade volume is defined as the area under the positive part of the ejection curve, retrograde flow being the area under the negative part of the injection curve. These distinctions led to appreciation of the difference between sloshing of blood and flow around the cardiovascular circuit.

The left ventricle (Figure 1A) is identified in conjunction with its preload in the form of a constant pressure source, \( p_{\text{ven}} \), implying a large venous reservoir, such as occurs in a normo- to hypervolemic, supine human, and an arterial load represented by the three element modified windkessel to the far right of the ventricle. The mitral and aortic valves, modeled as free-flowing two-dimensional leaflets are marked, as is the facility to apply periodic external compression and relaxation, \( p_{\text{e}}(t) \). The valves are not modeled as electrical diodes in order to allow internal definition of movement. For simplicity, the external pressure is interpreted as acting directly and selectively on the ventricle.

The same features appear in Figure 1B, with \( R_{\text{LV}} \) denoting a (small) inflow resistance, \( Z_{\text{os}} \) the characteristic impedance of the aorta, \( R_{\text{s}} \) the systemic peripheral resistance and \( C_{10} \) the arterial compliance. Pressures are marked \( p \). Flows, \( Q \), with their positive directions, are defined in each Figure 1A:

\[ V_{\text{LV}}(t) \text{ denotes left ventricular volume at time } t, \text{ and } R_{\text{LV}} \text{ a ventricular inflow resistance. For the function of the two variable resistors marked } R_{\text{lv}}, \text{ consult the caption of Figure 9.} \]

7.3 Results

In the model, under controlled conditions, with definition of preload, normal myocardial contractility (\( F_{\text{LV}} \)) and the load impedance by the aorta, physiological output curves for flow and pressure are demonstrated in Figure 2.

This figure displays the left ventricular pressure and ejection flow including the so-called ejection effect\(^2\) for a heart rate of 60 min\(^{-1}\), a venous pressure (\( p_{\text{ven}} \)) of 10 mm Hg and \( R_{\text{s}} \) at 1.2 mm Hg sec ml\(^{-1}\). The current model continues where the former is restricted to the introduction of the ejection effect into the outflow curve, incorporating this into the theory of impedance defined flow. The ejection effect embodies the influence of outflow on ventricular pressure. Its inclusion tends to make both pressure and flow downstrokes more convex. The stroke volume is 64 ml, the ejection fraction 52 %. The summaries of a preload ranging from 4-20 mm Hg and of peripheral resistance changes from 0.6 to 2.2 mm Hg sec ml\(^{-1}\) are shown in Figures. 3A and 3B,

\[ \text{Figure 2: Ventricular pressure (} p_{\text{v}}(t), \text{ solid) and outflow through the aortic valve (} Q_{\text{lv}}(t), \text{ dashed), with the ejection effect. Stroke volume } V_{\text{lv}} \text{ is 64 ml, ejection fraction is 52 %, venous pressure is 10 mm Hg, and } R_{\text{s}} \text{ is 1.2 mm Hg sec ml}^{-1}. \]

This figure displays the left ventricular pressure and ejection flow including the so-called ejection effect\(^2\) for a heart rate of 60 min\(^{-1}\), a venous pressure (\( p_{\text{ven}} \)) of 10 mm Hg and \( R_{\text{s}} \) at 1.2 mm Hg sec ml\(^{-1}\). The current model continues where the former is restricted to the introduction of the ejection effect into the outflow curve, incorporating this into the theory of impedance defined flow. The ejection effect embodies the influence of outflow on ventricular pressure. Its inclusion tends to make both pressure and flow downstrokes more convex. The stroke volume is 64 ml, the ejection fraction 52 %. The summaries of a preload ranging from 4-20 mm Hg and of peripheral resistance changes from 0.6 to 2.2 mm Hg sec ml\(^{-1}\) are shown in Figures. 3A and 3B,
respectively, with the other parameters held as above. The stroke volume (solid line), defined as net output, is shown with the antegrade stroke volume, ($Q_{H15}$, dashed line), and reflects upon the competence of the aortic valve during relaxation. The end-diastolic volume (dash-dot line) is also shown.

**Figure 3A:** Stroke volume (solid), antegrade stroke volume (dashed), and end-diastolic volume (dash-dot), versus preload ($p_{ven}$). Heart rate is 60 beats per minute.

**Figure 3B:** Stroke volume (solid), antegrade stroke volume (dashed) and end-diastolic volume (dash-dot), versus systemic peripheral resistance ($R_s$). Note: the horizontal axis runs from 0.6 to 2.2 in steps of 0.4. Heart rate 60 bpm.
Chapter 7: CVe1, pumping the heart

respectively, with the other parameters held as above. The stroke volume (solid line), defined as net output, is shown with the antegrade stroke volume, (Q_H15, dashed line), and reflects upon the competence of the aortic valve during relaxation. The end-diastolic volume (dash-dot line) is also shown.

Figure 3A: Stroke volume (solid), antegrade stroke volume (dashed), and end-diastolic volume (dash-dot), versus preload (p_{ven}). Heart rate is 60 beats per minute.

By setting F_{LV} (Appendix 1.1-1.3) equal to zero, the ventricle becomes asystolic. Its myocardial properties become dependent on its diastolic function \(a_{LV} \) and \(b_{LV}\) and, as CPR is started, on the external pressure \(p_e(t)\) applied to the outside of the ventricle. In order to allow direct comparison with the physiological validation, the compression frequency was chosen at 60 min\(^{-1}\) as opposed to the 80-100 compressions min\(^{-1}\) in current clinical practice. The duty cycle, the time relationship between compression and relaxation as a continuous sine wave, is 50%, as clinically appropriate. The \(p_e(t)\) curve is shown in the top of Figure 5A for reference purposes, with \(p_{emin}\) being equal to zero. Figure 4 depicts stroke volume as a function of the maximal external pressure during its systolic phase, while in Figure 5A-C the \(p_{emax}(t)\) increases from 20 to 70 to 140 mm Hg, respectively, while ventricular pressure (p_{ve}) is plotted synchronously with mitral inflow and aortic outflow.

Figure 3B: Stroke volume (solid), antegrade stroke volume (dashed) and end-diastolic volume (dash-dot), versus systemic peripheral resistance (R_s). Note: the horizontal axis runs from 0.6 to 2.2 in steps of 0.4. Heart rate 60 bpm.
Figure 4: Stroke volume (solid), antegrade stroke volume (dashed), and end-diastolic volume (dash-dot), versus peak compression pressure ($p_{\text{emax}}$). Compression rate is 60 cpm.

Figure 5A: Left ventricular pressure ($p_6(t)$, solid), inflow through the mitral valve ($Q_{H13}(t)$, dash-dot) and outflow through the aortic valve ($Q_{H15}(t)$, dashed) for a non-contracting ventricle exposed to a compression pressure $p_{\text{emax}}$ of 20 mm Hg. The time average values over one period of $Q_{H13}$ and $Q_{H15}$ are 17 ml/sec. The time course of $p_6(t)$ is displayed at the top. Venous pressure is 10 mm Hg in all three displays. In other Figures $p_{\text{emax}}$ may be different as defined in the pertinent captions.
Chapter 7: CVe1, pumping the heart

Figure 4: Stroke volume (solid), antegrade stroke volume (dashed), and end-diastolic volume (dash-dot), versus peak compression pressure (p_{\text{emax}}). Compression rate is 60 cpm.

Figure 5A: Left ventricular pressure (p_6(t), solid), inflow through the mitral valve (Q_{H13}(t), dash-dot) and outflow through the aortic valve (Q_{H15}(t), dashed) for a non-contracting ventricle exposed to a compression pressure p_{\text{emax}} of 20 mm Hg. The time average values over one period of Q_{H13} and Q_{H15} are 17 ml/sec. The time course of p_e(t) is displayed at the top. Venous pressure is 10 mm Hg in all three displays. In other Figures p_{\text{emax}} may be different as defined in the pertinent captions.

Figure 5B: Left ventricular pressure (p_6(t), solid), inflow through the mitral valve (Q_{H13}(t), dash-dot) and outflow through the aortic valve (Q_{H15}(t), dashed) for a non-contracting ventricle exposed to a compression pressure p_{\text{emax}} of 70 mm Hg. The time average values over one period of Q_{H13} and Q_{H15} are 46 ml/sec.

Figure 5C: Left ventricular pressure (p_6(t), solid), inflow through the mitral valve (Q_{H13}(t), dash-dot) and outflow through the aortic valve (Q_{H15}(t), dashed) for a non-contracting ventricle exposed to a compression pressure p_{\text{emax}} of 140 mm Hg. The time average values over one period of Q_{H13} and Q_{H15} are 88 ml/sec.
These same pressure and flow characteristics are displayed in Figure 6 for a less compliant (stiffened) ventricle or so-called stone heart as may happen after the first minutes of asystole. The mitral and aortic valves are allowed to move freely and are seen to be competent in all cases above.

Figure 6: As Figure 5 for a stiffer relaxed ventricle ($a_{LV}=40 \times 10^{-7}$ instead of $7 \times 10^{-7}$ mm Hg/ml$^2$). Left ventricular pressure ($p_6(t)$, solid), inflow through the mitral valve ($Q_{H13}(t)$, dash-dot) and outflow through the aortic valve ($Q_{H15}(t)$, dashed) with a compression pressure $p_{ven}$ of 70 mm Hg. The time average values of $Q_{H13}$ and $Q_{H15}$ are 45 ml/sec over one period.

With respect to the controversy in whether the (left) heart is a conduit during CPR, further experiments can be performed by defining incompetent valves within the model. Figure 7 illustrates the dependence of stroke volume ($Q_{H13}$), antegrade stroke volume, and end-diastolic volume on preload, $p_{ven}$, with a normally contracting ventricle. The percentage difference between antegrade stroke volume and stroke volume increases as $p_{ven}$ increases. Comparison of Figure 3A with Figure 7 demonstrates a reduction of stroke volume by 7/8.
These same pressure and flow characteristics are displayed in Figure 6 for a less compliant (stiffened) ventricle or so-called stone heart as may happen after the first minutes of asystole. The mitral and aortic valves are allowed to move freely and are seen to be competent in all cases above.

With respect to the controversy in whether the (left) heart is a conduit during CPR, further experiments can be performed by defining incompetent valves within the model. Figure 7 illustrates the dependence of stroke volume ($Q_{H15}$), antegrade stroke volume, and end-diastolic volume on preload, $p_{ven}$ with a normally contracting ventricle. The percentage difference between antegrade stroke volume and stroke volume increases as $p_{ven}$ increases. Comparison of Figure 3A with Figure 7 demonstrates a reduction of stroke volume by $\frac{7}{8}$.

The dependence of left ventricular volume, ventricular pressure, flow through the mitral ostium and flow through the aortic root in a normally contracting valveless ventricle with $p_{ven}$ at 10 mm Hg is graphed in Figure 8A. Time averaged values for the two flows are 7.8 ml/sec. Figure 8B shows the time courses of the same quantities when the venous pressure is increased to 20 mm Hg. Time averaged values for the two flows increases to 15 ml/sec. After removal of the ‘functional’ valves, the mitral and aortic ostia retain their defined area. The inflow resistance, $R_{LV}$, and the characteristic impedance in the aorta remain unaltered.

The figure shows a graph with the x-axis labeled as "Preload ($p_{ven}$, mmHg)" and the y-axis labeled as "Volume (ml)". The graph illustrates stroke volume (solid), antegrade stroke volume (dashed), and end-diastolic volume (dash-dot), versus preload ($p_{ven}$) in a normally contracting ventricle after removal of both valves.

**Figure 7:** Stroke volume (solid), antegrade stroke volume (dashed), and end-diastolic volume (dash-dot), versus preload ($p_{ven}$) in a normally contracting ventricle after removal of both valves.

The dependence of left ventricular volume, ventricular pressure, flow through the mitral ostium and flow through the aortic root in a normally contracting valveless ventricle with $p_{ven}$ at 10 mm Hg is graphed in Figure 8A. Time averaged values for the two flows are 7.8 ml/sec. Figure 8B shows the time courses of the same quantities when the venous pressure is increased to 20 mm Hg. Time averaged values for the two flows increases to 15 ml/sec. After removal of the ‘functional’ valves, the mitral and aortic ostia retain their defined area. The inflow resistance, $R_{LV}$, and the characteristic impedance in the aorta remain unaltered.

**Figure 8A:** Left ventricular volume ($V_{LV}(t)$, solid), ventricular pressure ($p_{v}(t)$, solid), flow through the mitral valve ostium ($Q_{H13}(t)$, dash-dot) and flow through the aortic root ($Q_{H15}(t)$, dashed), in a normally contracting valveless ventricle with $p_{ven}$ at 10 mm Hg. The time average values over one period of $Q_{H13}$ and $Q_{H15}$ are 7.8 ml/sec.
The removal of valves may also be directly applied in the CPR situation. Stroke volume, antegrade stroke volume, and retrograde stroke volume (the area under one cycle of the negative part of the \( Q_{H13} \) curve) as a representation of regurgitant flow into the atrium, as well as end-diastolic volume are displayed as a function of venous pressure in Figure 9.

Figure 9: A detailed explanation and a remedy may be found in the discussion. Figures 9, 10A and 10B are computer solutions obtained after the remedy was instituted. Stroke volume (solid), antegrade stroke volume (dashed), retrograde stroke volume, the area under the negative \( Q_{H13} \) curve (dotted), and end-diastolic volume (dash-dot) versus preload (\( p_{ven} \)) for a non-contractile valveless ventricle.
At a venous pressure of 10 mm Hg, Figure 10A displays the left ventricular volume, ventricular pressure, flow through the mitral ostium and through the aortic root for a peak compression pressure of 60 mm Hg. Time averaged values for both flows are 7.5 ml/sec. Antegrade and retrograde stroke volumes are 12 and 97 ml, respectively, denoting prominent sloshing on the venous side.

Figure 10B displays the same time functions for the same conditions, but for a venous pressure of 20 mm Hg. Time averaged values for both flows increased to 15 ml/sec, the antegrade and retrograde stroke volumes to 21 and 149 ml, respectively. The venous sloshing increases proportionately.
In the original computer experiments for Figure 9 and 10, ventricular volumes were sometimes found to become negative. Other investigators have mentioned, but left unpublished, similar observations for several years. Since negative ventricular volume is a physical impossibility, its origin was sought and remedied. The remedy is incorporated in the figures.

7.4 Discussion

Modeling procedures are utilized in view of the fact that they permit experiments to be performed under carefully controlled conditions and allow manipulations not acceptable for application to the animal or human studies. It permits the use of selective pathology: the totally healthy being with an asystolic heart but also the normal heart without functional valves. The small model depicted in Figure 1A comprises the left ventricle in fluid mechanical representation. The preload is defined by an adjustable, constant venous pressure; the arterial load is cast in the form of a three-element modified windkessel. The ventricle is capable of normal contraction and relaxation with an adjustable frequency, and can be made passive. Inlet and outlet valves (representing mitral and aortic) may be included or excluded. The time dependence of variable pressures is presented in the Appendix, as are the parameter values. Steady state solutions to the set of equations are obtained through a numerical solution method.

Experiments, for the validation of the model, shown in Figures 2 and 3 demonstrate the standard patterns of behavior in terms of pressure and ejection flow, and sensitivity to preload and to peripheral resistance of stroke volume, and end-diastolic volume. This evidence is deemed to indicate that the ventricle behaves normally.
When the ventricle loses its capability to contract, modeled by setting $F$ in Appendix 1 to zero, intrathoracic pressure, $p_e(t)$ is applied as an external pressure to the ventricle with a 50% duty cycle, as displayed at the top of Figure 5A.

The model allows an understanding of Kouwenhoven’s theory, in conjunction with increasing clinical, but still mostly empirical concerns about ventricular filling in the asystolic heart. It confirms recent work in swine by Klouche et al.\textsuperscript{23} who suggest that current techniques may not be suitable after only eight minutes of CPR. In addition, it allows hemodynamic insights into contradictory echocardiographic studies which have been performed in patients undergoing CPR.

While 77 patients have been described, the compression force has yet to be objectified in conjunction with valve motion and output.\textsuperscript{5} These studies tend to show that subtotal closure of the mitral or aortic valve is associated with minimal forward flow, as the model shows.

This model suggests that the ‘cardiac’ pump theory, in its fundamental form, can be applied, but that this offers limited forward flow. The left ventricle is compressed by an outside force without interfering with the cardiac valves, and without influencing filling pressures, as described by Kouwenhoven et al.\textsuperscript{2} It ignores the viscoelastic properties of the chest when applying $p_e$ and describes the ventricle as if it supported on a firm surface. Clinically, whether the support actually exists is still controversial, although reports have been describing the heart to be fixed within the mediastinum by the pericardium.\textsuperscript{23,24} The improved effect of increasing $p_e(t)$ is in line with experimental findings.\textsuperscript{22} A limitation is the compression frequency of 1 Hz, while current clinical practice is 100 compressions min$^{-1}$ and the higher frequency may improve cardiac output.\textsuperscript{26}

As Figure 4 shows, stroke volume improves by increasing the level of external pressure (Figures 5A-C) when both valves operate normally. Stroke volume suffers slightly when the ventricle’s passive elastic properties become less compliant (stone heart; Figure 6).

Analysis of the conduct of the model requires an understanding of early work by Harvey and Liebau. William Harvey, in his 1628 treatise entitled ‘De Motu Cordis et Sanguinis’ assigned the movement of blood around the cardiovascular circuit to the valve-equipped pumping heart alone.\textsuperscript{27} During the 1950’s, the physician Liebau\textsuperscript{28} challenged this position by demonstrating that valveless, fluid dynamic, closed-loop models of his own construction could generate average flow around the closed loop as a result of periodic local compression and release. Despite support by fluid dynamicists, Liebau was unable to offer an explanation and interest waned until a quantitative interpretation was published in 1998.\textsuperscript{29} The non-uniform distribution of impedance around the cardiovascular circuit proved to be the secret and the phenomenon was called impedance defined flow. Harvey’s observations of the heart and its valves may be seen as a special case, and consequently, both concepts could be unified within impedance defined flow.\textsuperscript{29} Impedance defined flow also applies to open systems such as in Figure 1.

In classic models of the cardiovascular system, flow was generated by a pressure difference between the arteries and veins, such as has been described during the first minutes of circulatory collapse. Parameter values were adapted to match experimentally derived information. If a ventricle contracts and relaxes, its blood volume can, in principle, escape in two directions during contraction and return from two directions during relaxation. If it were the only mechanism operating to move blood, then flow in either direction would be equal to the pressure difference between a selected point and the ventricle, divided by the resistance (impedance) between these two points.
The situation in Figure 1 is slightly more complicated by the presence of a venous pressure source, $p_{ven}$, which may be responsible for a part of the observed flow. These two flows may be identified separately, as illustrated in Figure 8 (a valveless ventricle). Here, $p_{ven}$ generates a flow of $(10-0)/1.2 \approx 8 \text{ ml/sec}$, which is equal to the average calculated flow through the model. This same analysis for a venous contribution applies in Figure 10. In both cases the superimposed oscillations are due to direct ventricular cyclic application of $p_e(t)$, as would be done during clinical CPR. This means that a heart contracting without valves, or during CPR applied to a ventricle without competent valves, forces an insignificant amount of blood through the peripheral resistance. Essentially, the model demonstrates that all flow passing $R_s$ is due to the venous pressure. In Figures 8 and 10 sloshing of blood is a second spectacular event: large inflow and outflow signals follow each other on the venous side during a single heart beat. As a result, the large flow pulses fail to contribute to transportation.

This contrasts strongly with the results in Figures 2 and 5, where the valves are competent. In these illustrations the ventricles generate all the flow through the peripheral resistance, without a contribution by venous pressure since at least one valve is closed at any one time. The role of $p_{ven}$ is limited to filling the ventricle.

Figure 11 shows a basic sketch of measured pressure-volume relations for the left ventricle. The relation may be formulated mathematically by:

$$p_6 = a_{LV}(V_{LV}-b_{LV})^2$$  \hspace{1cm} (eq. 7.1)

**Figure 11:** Pressure-Volume relationships in the left ventricle. Description of the conduct of the left ventricle as a pressure-volume relationship, when the ventricle is passive. Demonstrates the principle suggested by Galenus, with ‘suction’ of the ventricle when volumes are very low. $p_6$ = pressure in the left ventricle, $a_{LV}$ = a measure of elastance of the left ventricle, $b_{LV}$ = volume of the left ventricle, with $b$ is the point at which the pressure is zero, $V_{LV}$ = the volume of the left ventricle.
Chapter 7: CVe1, pumping the heart

The situation in Figure 1 is slightly more complicated by the presence of a venous pressure source, \( p_{ven} \), which may be responsible for a part of the observed flow. These two flows may be identified separately, as illustrated in Figure 8 (a valveless ventricle). Here, \( p_{ven} \) generates a flow of \( (10-0)/1.2 \approx 8 \) ml/sec, which is equal to the average calculated flow through the model. This same analysis for a venous contribution applies in Figure 10. In both cases the superimposed oscillations are due to direct ventricular cyclic application of \( p_{e}(t) \), as would be done during clinical CPR. This means that a heart contracting without valves, or during CPR applied to a ventricle without competent valves, forces an insignificant amount of blood through the peripheral resistance. Essentially, the model demonstrates that all flow passing \( R_s \) is due to the venous pressure. In Figures 8 and 10 sloshing of blood is a second spectacular event: large inflow and outflow signals follow each other on the venous side during a single heartbeat. As a result, the large flow pulses fail to contribute to transportation.

This contrasts strongly with the results in Figures 2 and 5, where the valves are competent. In these illustrations the ventricles generate all the flow through the peripheral resistance, without a contribution by venous pressure since at least one valve is closed at any one time. The role of \( p_{ven} \) is limited to filling the ventricle.

Figure 11 shows a basic sketch of measured pressure-volume relations for the left ventricle. The relation may be formulated mathematically by:

\[
p_{6} = a_{LV}(V_{LV}-b_{LV})^{2} \tag{eq. 7.1}
\]

For a relaxed ventricle (\( F_{LV}=0 \), Appendix 1). The function is nonlinear, and becomes zero for \( V_{LV}=b_{LV} \). For larger values of \( V_{LV} \) ventricular pressure is positive, for smaller ones negative (broken line, indicating suction). Therefore, for \( V_{LV}>b_{LV} \), \( a_{LV} \) must be positive, for \( V_{LV}<b_{LV} \), \( a_{LV} \) must be negative. This is consistent with the elastance (\( = 1/\text{compliance} \)) of the ventricle:

\[
dp_{6}/dV_{LV} = 2a_{LV}(V_{LV}-b_{LV}) \tag{eq. 7.2}
\]

which is then positive over the range of the sketch. If the change in sign of \( a_{LV} \) is ignored, the broken part of the sketch changes to its mirror image, \( p_{6} \) is always positive, while the elastance turns negative for \( V_{LV}<b_{LV} \), all unrealistic features.

Figure 1B suggests the solution immediately: the discharge (outflow) from \( C_{b} \), the ventricular compliance, can continue beyond the empty state and the computer will designate such volume to be negative. The artifact of negative volume creation can be avoided by allowing the resistors \( R_{lv} \) to go to infinity as ventricular volume approaches zero, which is their sole function. For positive ventricular volumes \( R_{lv} \) values are negligibly small.

Figure 1A, the model in fluid mechanical symbols, is not sensitive to this flaw, for the simple reason that flattening of the left ventricle eliminates communication between the ventricle and the venous and the arterial sides. \( R_{lv} \)'s are not required, though they are drawn in to show the need for them, and their location in Figure 1B where the venous and arterial parts of the model continue to communicate.

In mathematical terms, while including external pressure \( p_{e}(t) \), the ventricular pressure-volume relation reads:

\[
p_{6}(t) - p_{o}(t) = a_{LV}(V_{LV}(t)-b_{LV})^{2} \tag{eq. 7.3}
\]

for an asystolic ventricle (\( F_{LV}=0 \)). If \( p_{e}(t) > p_{6}(t) \), the left hand side of the equation becomes negative, hence \( a_{LV} \) must be negative, which it is in the range where \( V_{LV} < b_{LV} \). Solving for \( V_{LV} \) under these conditions yields

\[
V_{LV} = b_{LV} - [(p_{e}-p_{o})/(-a_{LV})]^{1/2} \tag{eq. 7.4}
\]

or \( V_{LV} \) will become negative when

\[
p_{e}(t)-p_{6}(t) > -a_{LV}b_{LV}^{2} \tag{eq. 7.5}
\]

which is a small positive pressure (Appendix 1).
7.5 Conclusion

The primary conclusion is that the model ventricle performs physiologically in terms of flow, ejection curves and pressures. Under asystolic conditions, the model demonstrates potential for the cardiac pump mechanism as proposed by Kouwenhoven et al.² The study also demonstrates that it allows further insights into the actual source of flow under incompetent valve conditions, both for a contracting heart and a heart under CPR conditions. The absence of contraction can, in principle, be replaced by external compression, provided the valves are competent (Figure 8), increasing flow from 17 to 88 ml/sec with increasing $p_e(t)$. If the valves are not competent the 8 ml/sec output is due to venous filling pressure and insensitive to external compression (Figure 10). Impedance defined flow demonstrates that sloshing, on the venous side, becomes dominant, negating effective movement.
The primary conclusion is that the model ventricle performs physiologically in terms of flow, ejection curves and pressures. Under asystolic conditions, the model demonstrates potential for the cardiac pump mechanism as proposed by Kouwenhoven et al. The study also demonstrates that it allows further insights into the actual source of flow under incompetent valve conditions, both for a contracting heart and a heart under CPR conditions. The absence of contraction can, in principle, be replaced by external compression, provided the valves are competent (Figure 8), increasing flow from 17 to 88 ml/sec with increasing $p_e(t)$. If the valves are not competent the 8 ml/sec output is due to venous filling pressure and insensitive to external compression (Figure 10). Impedance defined flow demonstrates that sloshing, on the venous side, becomes dominant, negating effective movement.

### References

The Donders model of the circulation in normo- and pathophysiology

Abstract

A model of the closed human cardiovascular loop is developed. This model, using one set of 88 equations, allows variations from normal resting conditions to exercise, as well as to the extreme condition of a circulation following cardiac arrest. The principal purpose of the model is to evaluate the continuum of physiological conditions to cardiopulmonary resuscitation effects within the circulation.

Within the model, Harvey’s view of the circulation has been broadened to include impedance defined flow as a unifying concept. The cardiac function curve, the relation between ventricular filling and output, changes during exercise. First, it rotates counterclockwise and stretches along the output axis, second, it shifts along the filling axis. The first is induced by sympathetic control, the second by respiratory control. The model shows that depth of respiration, sympathetic stimulation of cardiac contractile properties and baroreceptor activity can exert powerful influences on the increase in cardiac output, while heart and respiratory rate increases tend to exert an inhibiting influence. Impedance defined flow encompasses both positive and negative effects.

The model demonstrates the limitations to cardiopulmonary resuscitation caused by external force applied to intrathoracic structures, with effective cardiac output being limited by collapse and sloshing. It demonstrates that the clinical inclination to apply high pressures may be unjustified.
Chapter 8: CVe2, the circulation in normo- and pathophysiology

Abstract

A model of the closed human cardiovascular loop is developed. This model, using one set of 88 equations, allows variations from normal resting conditions to exercise, as well as to the extreme condition of a circulation following cardiac arrest. The principal purpose of the model is to evaluate the continuum of physiological conditions to cardiopulmonary resuscitation effects within the circulation.

Within the model, Harvey's view of the circulation has been broadened to include impedance defined flow as a unifying concept. The cardiac function curve, the relation between ventricular filling and output, changes during exercise. First, it rotates counterclockwise and stretches along the output axis, second, it shifts along the filling axis. The first is induced by sympathetic control, the second by respiratory control. The model shows that depth of respiration, sympathetic stimulation of cardiac contractile properties and baroreceptor activity can exert powerful influences on the increase in cardiac output, while heart and respiratory rate increases tend to exert an inhibiting influence. Impedance defined flow encompasses both positive and negative effects.

The model demonstrates the limitations to cardiopulmonary resuscitation caused by external force applied to intrathoracic structures, with effective cardiac output being limited by collapse and sloshing. It demonstrates that the clinical inclination to apply high pressures may be unjustified.
8.1 Introduction and motivation

William Harvey is generally credited with the first functional description, or conceptual model, of the circulation, despite a preceding presentation by Andreas Caesalpinus in 1593. As early as the 19th century, a number of prominent scientists expressed serious doubts about the validity of Harvey’s conclusion that the heart was the only pump supporting the circulation. The heart was felt to be too weak. Franciscus Donders, one of the critics, proposed in his 1856 textbook that the respiratory system could aid venous return. While resulting in a vigorous debate, no consensus was reached, leaving the basic question of perceived cardiac weakness unanswered.

Guyton, sidestepping the issue raised by Donders, addressed the impressive rise in cardiac output during heavy exercise. Selecting right atrial pressure as the independent variable, he evaluated venous return and cardiac output as they depend on the strength of sympathetic stimulation and the value of mean circulatory filling pressure.

During the 20th century, Liebau added complexity to the discussion by showing, in hydraulic models free of valves, that a steady flow around a closed circuit could be generated by periodic local compression and release. This led him to suggest that cardiac valves are superfluous. Moser et al. developed a mathematical basis for Liebau’s experimental observations, while both Donders and Guyton had limited themselves to generalization of clinical observations. Moser et al. identified general conditions for its applicability while also pointing out specific restrictions for its utilization in the cardiovascular system. The concept was named impedance defined flow, reflecting the importance of flow as opposed to pressure.

In this same period closed-chest cardiac resuscitation was developed, presenting a new circulatory condition under which the same issues applied. Cardiopulmonary resuscitation (CPR), with its persistence of poor outcome, has raised a spectrum of problems that require clarification, all of which involve the central issue of the role of pumps, the differences between flows and pressures, and the function of valves. Clarification of these issues has continued to be elusive, despite intensive research efforts.

We introduce a newly developed model, called the Donders model. This mathematical model will be applied to the circulation under the broad spectrum of conditions ranging from rest, to physiological exercise, and finally to circulatory collapse, with the application of CPR. It is unique in that it contains a physiological heart, control features, readily adjustable parameter values, and allows a biophysical approach to clinical problems, as was its predecessor in solving arterial wave transmissions issues [Westerhof et al., 1969]. Mechanisms supporting venous return during this broad range of conditions will be demonstrated including the role played by impedance defined flow. Validation of model performance will be provided.

The model supplies solutions to long-standing problems as well as to current ones by a single mathematical system. Its clinical relevance lies in the presentation of solutions that could not be obtained in the past since they required experiments difficult or ethically unacceptable to perform.
8.2 The development of cardiovascular system modeling

The interdependence of events in the circulatory system has long enchanted investigators. To visualize the situation better, some have built hydraulic models of the circulation. Such models, originally designed primarily for teaching purposes, were published by Weber\textsuperscript{11} and by Marey.\textsuperscript{12} Practical problems, such as blood viscosity, the adjustability of parameters, and leakage encountered in hydrodynamic models discouraged their further development. Starting with a hydrodynamic model, and moving to a mathematical and later an electrical model, Jochim\textsuperscript{13} studied the effect of changes in peripheral resistance on arterial pulse pressure using heart rate and stroke volume as parameters. More flexible versions made their appearance starting in the 1960’s.\textsuperscript{14,15} The more complex ones modeled the left side of the heart, the aorta, the peripheral resistance and the veins as a closed loop, with valves at a few appropriate sites. Parameters, such as peripheral resistance, arterial and venous compliances and the frequency of ventricular contraction, could be adjusted allowing qualitative analysis of the resulting effects.

The variety of models that have subsequently been designed to study the circulation can be conveniently divided into four classes according to levels of sophistication.\textsuperscript{16} In its simplest form, the circulation has been represented by a resistive model. Hill et al.\textsuperscript{17} designed a model of this nature to study the changes in the circulation at birth, while Vadot,\textsuperscript{18} proposed one to predict the changes to be expected from major surgical intervention. The resistive-capacitive model\textsuperscript{19} was aimed at a description of average values of pressures, flows and volumes and introduced the behavior of the heart based on Starling’s concept in the form:\textsuperscript{20}

\[ W_t = SV_d \] (eq. 9.1)

where \( W_t \) denotes the work performed by the ventricle and \( V_d \) its end-diastolic volume (EDV), while \( S \) is a proportionality constant between the two, denoted the contractile strength of the ventricle. The incorporation of Starling’s concept in the model popularized formulations of ventricular performance.

Pulsatile phenomena were first included in Warner’s resistive-inductive-capacitive (RLC) model\textsuperscript{21} which also set the structural pattern for many subsequent studies. This circulatory loop was subdivided into a number of sections with the condition that outflow of any section equals inflow into the next. For each section, one of the following three equations must be written. In Warner’s model these are: (a) an equation of motion, which is commonly a simplified version of the Navier-Stokes equation:

\[ p_{i-1}(t) - p_i(t) = L \frac{d}{dt} Q_i(t) + R_{i-1} Q_i(t) \] (eq. 9.2)

in which \( p \) defines the operating pressure, \( Q \) describes the flow, \( L \) accounts for the inertial, and \( R \) for viscous properties of blood; (b) an equation of continuity which relates change in blood volume \( V \) contained by a section to its inflow and outflow, such that:

\[ V_i(t) = V_i(t=0) + \int [Q_i(t) - Q_{i+1}(t)] dt \] (eq. 9.3)

and (c) an equation of state, which relates pressure in a section to its volume, through the compliant properties of the wall. For vessels he defined \( m = 1 \) for arteries and \( m > 1 \) for veins, such that:

\[ p_i = \frac{1}{C_i} V_i^m \] (eq. 9.4a)

Chapter 8: CVe2, the circulation in normo- and pathophysiology
For the ventricles, Warner introduced the concept of the time varying compliance $C$, with $C_d$ and $C_u$ as constants for diastolic and systolic segments of a cycle, respectively, such that:

$$p_i = \frac{1}{[1/(C_d + C_u)]} V_i^{m}$$

(eq. 9.4b)

Subdivision of the circulation into six sections requires the solution of 18 simultaneous equations, with the cardiac valves opening and closing with the aid of a multiple diode function generator.

Simple resistive-capacitive as well as the resistive-inductive-capacitive models exhibited some of the striking features that are recognized in the mammalian circulation, such as modulation of stroke volume and volume loading, but with the notable exception of the conduct of contractile chambers. The success stimulated their repeated application and expansion e.g., by Defares et al.\textsuperscript{22} Beneken,\textsuperscript{23} and Dick et al.,\textsuperscript{24} as well as inspired inclusion of control phenomena in which the model becomes active. As an example of this fourth group, Beneken and DeWit\textsuperscript{25} incorporated direct baroreceptor control of the heart rate and of the systemic peripheral resistance. Karreman and Weygandt\textsuperscript{26} expanded carotid sinus control of the peripheral vascular bed resistances by modeling the physiological feedback mechanism through a set of equations. They related carotid sinus pressure, via its wall deformation to baroreceptor nerve firing rate, then to sympathetic nerve firing rate, and finally sympathetic firing rate to the peripheral bed resistances as well as to negative feedback in the elastic modulus of the carotid arterial wall itself.

Shifting emphasis to longer-range control, Guyton et al.\textsuperscript{27} developed a larger set with over 350 equations. In essence, their model contains the blood conducting pathway, vascular stress and relaxation as it affects circulatory pressure, membrane dynamics of the capillaries, tissue fluid volume and pressure, electrolyte shift, angiotensin, aldosterone, and antidiuretic hormone control, kidney dynamics, control of blood flow in muscle, autoregulation, autonomic control as well as a number of other control facets.

All of these models, as well as their manifold variations, were conceived and built prior to the conceptualization of impedance defined flow. The right and left heart, in harmony with Harvey’s teaching, were assigned the responsibility to pump blood around the circuit. In the next section, we will argue that the operation of Harvey’s pumps relied on the principle of impedance defined flow, though he was unaware of this.

### 8.3 Impedance-defined (Z) flow

The key to impedance defined flow (Z-flow) is the recognition of two aspects of the circulatory loop as well as their implications. These are the insight that flow may be a bidirectional phenomenon and that the closed circulatory system may be subjected to external pumping at a multitude of locations. Potential locations for pumping are in the pulmonary circulation in response to differing airway pressures, or in the peripheral vessels due to venous tone or muscle activity.

In Z-flow considerations, flow is allowed to be bidirectional, which is easy to conceptualize in the vena cava. When a vessel segment is compressed, part or all of the blood displaced can, in principle, escape in two directions. When the compression is terminated, refilling of the segment may occur from two directions as well.

The origin of the forces inducing Z-flow may be sorted into four classes. Organs which display direct muscular contraction on blood, due to contraction and relaxation of their walls, may be...
grouped as class one. This class includes the atrial and ventricular pumps, and the pumps incorporated in the lymphatic and venous vessels walls. Brief focus on the circulatory system in a far peripheral bed, can demonstrate this. Vessel walls contain smooth muscle, though its fractional wall volume varies greatly. The arteriolar wall muscle contraction is viewed as controlling peripheral resistance, and is typically taken as a constant, without recognition for the oscillatory activity of the sphincters, named vasomotion. More recent views distinguish an additional effect, venomotion, concerning the venules, which also exhibit alternating contraction and relaxation at a variable, and independent, frequency. Venomotion operates locally as a tiny pump, moving blood centrally. The serial combination of the two obviously does not have the dimension of a simple resistance.

Class two deals with vessels that are passively compressed and released by an external agent. This class includes the effect of respiration on the intrathoracic blood volume, including that in the central veins. Other examples are the muscular effects on veins embedded in their tissue and the influence of arterial pulsation on the blood flow through their companion veins. The third class, which is less obvious, involves the effects on the body relative to a gravitational field. It recognizes the flow caused by changing position from standing to supine. Finally, the fourth class includes devices, such as human inventions, which may operate as an outside agency on the body and directly or indirectly promote arterial flow and/or venous return. An example of this would be cardiopulmonary resuscitation. The Z-flow concept permits quantitative analysis of systems with and without valves. Analysis of systems with explicit bidirectional flow, or flow in a system without valves, was not envisioned in classical wave transmission theory.

An example, demonstrated in a small section of the Donders model (Appendix 2), of a phenomenon belonging to the first class can be seen in Figure 1 and 2.

**Figure 1:** Illustration of the principle of impedance defined flow generation. Diagram of selected network in electrical symbols. The symbol p denotes blood pressures at the marked locations. Q’s denote blood flows. Rs represents arteriolar resistance, Cvs and Rvs the time varying compliance and resistance of contracting and relaxing muscular venules, Rs*/Cvs* and Rvs*/4 are fixed value downstream elements. Input and output pressures, p_p and p_{100}* are constant.
As a consequence of contraction of venules (decrease in $C_{VS}(t)$), the pressure $p_{ps}$ is increased, forcing outflow, $Q_{HI}$. The augmentation of $p_{ps}$ also generates flow in the upstream direction ($-Q_{HI}$, not shown). In addition, the contraction increases the value of the series resistance $R_{vs}$. The venules relax promptly, restoring the original resistance value. Net outflow is increased by 6% when this contraction is repeated at 0.5 Hz, in the direction of the central veins (16 ml s$^{-1}$ to 17 ml s$^{-1}$) in this pilot model.

This result may be contrasted with the effect of compression of the coronary veins during ventricular ejection (class two). This compression lasts long enough to reduce average flow through the coronary bed. These examples illustrate where intricacies occur in impedance defined flow. (A more complete account may be found in Noordergraaf A.)

Research inspired by Harvey, though carried out long after his life time, made it abundantly clear that pressure-flow relations within the circulatory system are governed by impedances, originally treated as constants (i.e., as resistances). More recently, they have been allowed to be frequency dependent and were called impedances. Eventually, with the availability of the computer, they were permitted to become time-varying as well. In describing a relation between a pressure difference and flow containing time-varying impedances, differential equations may require modification for their proper description. This can be exemplified by valves as time-varying impedances: their impedance negligible when open, large when closed.

If Harvey’s term ‘solus’ is dropped, the two concepts merge and unidirectional flow becomes a special case of bidirectional flow. Ultimately, his view resulted in a multitude of studies of the cardiovascular system in which the subject lies quietly in the horizontal position in an attempt to avoid observed, but not recognized, redistribution of blood (flow) attendant upon muscular activity, body motion or upon changes in gravitational effects.
Chapter 8: CVe2, the circulation in normo- and pathophysiology

8.4 The Donders model

The model developed incorporates a closed circulatory loop, representative of the physiological state and is specifically suitable for cardiopulmonary resuscitation which focuses on intrathoracic structures (Appendix 2). Cerebral, thoracic, extrathoracic (arms), abdominal and extra-abdominal (legs) subdivisions are clearly identifiable. The heart, complete with coronary circulation, lies within the pericardium through which the venae cavae pass. It has two atria, two ventricles, and all four are capable of cycling through contraction and relaxation at variable frequencies. The four cardiac valves are free moving with regulation following from pressure and flow phenomena. The diode symbol is used to mark simple valves. This applies to intracardiac as well as intravascular valves (T = tricuspid, P = pulmonary, M = mitral, A = aortic). Additional valves such as Niemann’s valve (N) at the thoracic outlets and peripheral venous valves (Lp as shown in Appendix 2.2 and p, as listed in Table 1) follow these same effects. The high pressure systemic and low pressure pulmonary systems each have their arterial and venous out- and inflow tracts.

Different compartments may interact depending on conditions set. The model includes a facility to have the respiratory system act on intrathoracic pressure at a physiological frequency (i.e., one well below that of the heart rate). The open circles in sections comprising the thorax and the abdomen (Appendix 2.2) allow for the insertion of respiratory or other external, pressure, \( p_e(t) \), (Figure 3) as if it were an intrapleural effect.

![Figure 3: A normal pressure curve, \( p_e(t) \), for intrathoracic (intrapleural) pressure caused by respiration at rest. The horizontal line defines the time averaged value of the function. The inspiration to expiration ratio is 1:2.](image)
The four chambers of the heart, located within the pericardial sac, are marked by black squares, each with an S-shaped curve superimposed, making the further distinction between linear and nonlinear functions.

Five controls, some manifesting more than one facet have been identified that influence cardiac output (Figure 4).

1.) Sympathetic stimulation operating on the contractile properties of the cardiac chambers, modifying the cardiac function curves by rotating them counterclockwise and stretching them along the output axis. These are embodied by the coefficients ‘c’ in the chamber equations (eqs. I.77, 78 and I.80, 81).

2.) Neural stimulation of heart rate is embodied by an adjustable ‘f_h’.

3.) The respiratory influences (depth and frequency) on the cardiac chambers, modifying the cardiac function curves by shifting them back and forth along the preload (filling) axis. This is included in the chamber equations where p_e(t) appears (eqs. I.77, 78, 80 and 81), (Figure 6).

4.) Baroreceptor control of the systemic (‘R_s’) and pulmonary (‘R_p’) sides of the circulation. If cardiac output would, for example, triple as a result of exercise, arterial pressure would also if the peripheral resistance remained unchanged. In the circulation, baroreceptors, embedded in some of the systemic arteries, report arterial blood pressure levels to the central nervous system, its neural response adjusting the level of the peripheral resistance to undo most of such effects. In such instances transfer of blood from the arteries into the veins is facilitated.

5.) Motion (pumping) executed by the small muscular venules in the peripheral vasculature, most likely due to local metabolic control. 29 Venomotion, briefly discussed in section 3, above is not incorporated in the Donders model yet, owing to its provisional character.

They may be listed as:

1.) Sympathetic stimulation operating on the contractile properties of the cardiac chambers, modifying the cardiac function curves by rotating them counterclockwise and stretching them along the output axis. These are embodied by the coefficients ‘c’ in the chamber equations (eqs. I.77, 78 and I.80, 81).

2.) Neural stimulation of heart rate is embodied by an adjustable ‘f_h’.

3.) The respiratory influences (depth and frequency) on the cardiac chambers, modifying the cardiac function curves by shifting them back and forth along the preload (filling) axis. This is included in the chamber equations where p_e(t) appears (eqs. I.77, 78, 80 and 81), (Figure 6).
4.) Baroreceptor control of the systemic (‘R_s’) and pulmonary (‘R_p’) sides of the circulation. If cardiac output would, for example, triple as a result of exercise, arterial pressure would also if the peripheral resistance remained unchanged. In the circulation, baroreceptors, embedded in some of the systemic arteries, report arterial blood pressure levels to the central nervous system, its neural response adjusting the level of the peripheral resistance to undo most of such effects. In such instances transfer of blood from the arteries into the veins is facilitated.

5.) Motion (pumping) executed by the small muscular venules in the peripheral vasculature, most likely due to local metabolic control. Motion, briefly discussed in section 3, above is not incorporated in the Donders model yet, owing to its provisional character.

Figure 4: Scheme of the major players in the adjustment of cardiac output. The intrathoracic part of the closed loop contains the four cardiac chambers. Their pumping performance is traditionally characterized by cardiac function curves, an input-output relation with the input (filling) displayed along the horizontal axis. Input depends on preload that shifts along the filling axis under the influence of intrathoracic pressure variations imposed by the respiratory system. Output (stroke volume or cardiac output) is sensitive to the arterial load that can be modified by the baroreceptors via adjustment of the peripheral resistances ‘R_s’ and ‘R_p’. The contractile properties of the cardiac musculature are subject to sympathetic stimulation which rotates the cardiac function curves counterclockwise and stretches them along the output axis.

Figure 5: Illustration of the shifting of the cardiac function curve under the influence of intrathoracic pressure ‘p_e(t)’. For the purposes of this illustration, ‘p_e’ was made an independent variable. Its value was set by the investigator at a constant value for each measurement point. For p_e = 0, Starling’s experiment was essentially repeated. The magnitude of the shift, which includes negative and positive filling pressures, equals ‘p_e’ at zero output.

Each part of the closed loop retains its own properties and flexibility. Specifically, the arteries feature higher pulse wave velocity and faster blood transmission than veins. Veins contain more volume than arteries, exceeding 60% of the total blood volume. The contractile properties of the vessel walls, as well as the peripheral resistance at the arterioles, are reflected by their individual characteristics. The ventricles and to a lesser degree, the atria are sensitive to preload (filling pressure), arterial load, as well as to other factors such as contractile properties.
During cardiac arrest, the arterial system will lose volume to the higher compliant and lower pressure venous system, lowering arterial load. Chest compressions cause positive intrathoracic pressures which are transferred to the underlying organs. The pericardial sac allows equal distribution, while the intrinsic (diastolic) properties of the chambers determine their response to this outside pressure [Scharf et al., 1989].

Pressures are marked ‘p’, flows ‘Q’ with their arrows indicating the direction in which flow is positive. Volumes are denoted ‘V’. These quantities are all variables (Appendix 2). Parameters comprise compliances, marked ‘C’, viscous resistances ‘R’, and inertial properties of blood, ‘L’. The nonlinear, time-varying pressure-volume relations of the cardiac atrial and ventricular chambers are marked ‘C4’ and ‘C5’ (right heart), ‘C8’ and ‘C9’, for the left heart.

To initialize the model, it is “filled” with blood volume until the desired, mean or static, circulatory filling pressure is reached. Under initial conditions all pressures are equal and positive (e.g., 15 mm Hg) and all flows are zero. When the contractile properties are activated the blood distribution is modified, pressures change, flows develop, and steady state is achieved. The set of equations employed is listed in Appendix 2 and comprises 31 first order differential equations plus 57 algebraic equations.

The four cardiac chambers cycle through contraction and relaxation phases (Figure 5). The contraction phase is defined by a normalized function f(t) (eqs. I.85-87, Appendix 2). All four chambers observe the same function f(t), though their parameter values are different. For the purpose of identifying a particular chamber, f(t) is assigned the subscript LA (left atrium), LV (left ventricle), RA, or RV. This function is zero during the relaxation phase. It may be noted that atrial contraction commences at t = 0 in Figure 5. If a different time frame is utilized, appropriate time transformation should be made. As an example, if t = 0 is defined as the onset of atrial systole, and ventricular systole is set to start 0.15 sec later, the onset of ventricular contraction should be delayed by that amount while retaining the expressions (eqs. I.85-87, appendix 2). Valves open and close on the basis of pressure gradients and flows, respectively. The cardiac valvular ostia are designed as limiting resistors in conjunction with their normal size (Appendix 2).
During cardiac arrest, the arterial system will lose volume to the higher compliant and lower pressure venous system, lowering arterial load. Chest compressions cause positive intrathoracic pressures which are transferred to the underlying organs. The pericardial sac allows equal distribution, while the intrinsic (diastolic) properties of the chambers determine their response to this outside pressure [Scharf et al., 1989].

Pressures are marked 'p', flows 'Q' with their arrows indicating the direction in which flow is positive. Volumes are denoted 'V'. These quantities are all variables (Appendix 2). Parameters comprise compliances, marked 'C', viscous resistances 'R', and inertial properties of blood, 'L'. The nonlinear, time-varying pressure-volume relations of the cardiac atrial and ventricular chambers are marked 'C4' and 'C5' (right heart), 'C8' and 'C9', for the left heart.

To initialize the model, it is "filled" with blood volume until the desired, mean or static, circulatory filling pressure is reached. Under initial conditions all pressures are equal and positive (e.g., 15 mm Hg) and all flows are zero. When the contractile properties are activated the blood distribution is modified, pressures change, flows develop, and steady state is achieved. The set of equations employed is listed in Appendix 2 and comprises 31 first order differential equations plus 57 algebraic equations.

The four cardiac chambers cycle through contraction and relaxation phases (Figure 5). The contraction phase is defined by a normalized function \( f(t) \) (eqs. I.85-87, Appendix 2). All four chambers observe the same function \( f(t) \), though their parameter values are different. For the purpose of identifying a particular chamber, \( f(t) \) is assigned the subscript LA (left atrium), LV (left ventricle), RA, or RV. This function is zero during the relaxation phase. It may be noted that atrial contraction commences at \( t = 0 \) in Figure 5. If a different time frame is utilized, appropriate time transformation should be made. As an example, if \( t = 0 \) is defined as the onset of atrial systole, and ventricular systole is set to start 0.15 sec later, the onset of ventricular contraction should be delayed by that amount while retaining the expressions (eqs. I.85-87, appendix 2). Valves open and close on the basis of pressure gradients and flows, respectively. The cardiac valvular ostia are designed as limiting resistors in conjunction with their normal size (Appendix 2).

The function \( F(t) \) in equations I.79 and I.82 is equal to \( f(t) \) during diastole and during systole while the chamber is isovolumetric. As the pressure increases to allow ejection, chamber pressure drops as a result of volume loss. This is reflected by the volume containing terms in equations I.77, 78, 80, 81, and of weakening induced by muscle shortening, represented by the second terms in equations I.79 and I.82. Towards the end of systole, muscle is strengthened again, represented by the third terms in these equations. This combination suggests a shift in contractile energy down the time axis.

The difference between \( F(t) \) and \( f(t) \) is approximated mathematically by the sum of two terms:

\[-k_1 \text{ ejection flow} (t) + k_2 \text{ ejection flow}^2 (t-t) \quad \text{(eq. 9.5)}\]

which express weakening and strengthening, respectively, with ‘t’ signifying a time delay. The weighting factors, \( k_1 \) and \( k_2 \) are positive. This concept is applied to both ventricles.

Figure 6: Displays for \( f_{LA}(t) \) and \( f_{LV}(t) \) (eqs. I.85-87) under physiological conditions. As their formulas indicate, the amplitude of the \( f \) functions are normalized. Their counterparts, \( f_{LA}(t) \) and \( f_{LV}(t) \) respectively, are identical.

The function \( F(t) \) in equations I.79 and I.82 is equal to \( f(t) \) during diastole and during systole while the chamber is isovolumetric. As the pressure increases to allow ejection, chamber pressure drops as a result of volume loss. This is reflected by the volume containing terms in equations I.77, 78, 80, 81, and of weakening induced by muscle shortening, represented by the second terms in equations I.79 and I.82. Towards the end of systole, muscle is strengthened again, represented by the third terms in these equations. This combination suggests a shift in contractile energy down the time axis.

The difference between \( F(t) \) and \( f(t) \) is approximated mathematically by the sum of two terms:

\[-k_1 \text{ ejection flow} (t) + k_2 \text{ ejection flow}^2 (t-t) \quad \text{(eq. 9.5)}\]

which express weakening and strengthening, respectively, with ‘t’ signifying a time delay. The weighting factors, \( k_1 \) and \( k_2 \) are positive. This concept is applied to both ventricles.
The two arterial systems are approximated by three element models, the venous system by traditional LRC models, lumped separately for each of the major parts of the body. It may be noted that, for the traditional equation for veins, e.g., in (eq. I.6) of Appendix 2,

$$Q_{v10} = C_{10} \frac{d}{dt}(p_p - p_e)$$  \hspace{1cm} (eq. 9.6)

i.e., only the pulsatile components of the pressures influence the computation of flow.

In equation I.81, which describes the transmural pressure-volume relationship of the left ventricle, a ‘±’ sign has been added to the diastolic term. The ‘minus’ sign prevents ventricular compliance from going negative for small ventricular volumes. These values lie outside the normal physiological range, but may, potentially, be required for CPR situations. Such measurements do not appear to be available for the right ventricle.

Cardiopulmonary resuscitation is performed by applying ‘p_e(t)’ to all or selected structures in the model. This can be done with or without respiration being applied as another source of intrathoracic pressure. The frequency of compression can be varied.

8.5 Results

In this section the Donders model is used to describe well and lesser known entities such as resting, exercise and CPR conditions.

8.5.1 Validation of the model in resting conditions

Resting conditions, with the (70 kg, male) ‘patient’ in the supine position, without the effect of respiration (p_e(t)=0) is shown in Figure 7, A through D, under steady state conditions. Three consecutive beats are shown at a resting heart rate of 60 beats min⁻¹. Initial conditions include 6600 ml of blood volume. The systemic peripheral resistance, composed of six resistors at various points in the circulation, all in parallel (Appendix 2) is 1.2 mm Hg sec ml⁻¹. Table II lists the other parameter values.

Figure 7A focuses on the area in and around the left ventricle. The heart is contracting at 60 beats min⁻¹. The sharp negative excursions signify the instants of valve closure, in this case of the aortic valve. Depicted are left ventricular pressure (p₆ in Figure 2), root aortic pressure (p₆*) and ejection flow (Q_{H14}). End-diastolic volume is 90 ml, with an ejection fraction of 0.56.
Chapter 8: CVe2, the circulation in normo- and pathophysiology

The two arterial systems are approximated by three element models, \(^{34}\) the venous system by traditional LRC models, lumped separately for each of the major parts of the body. It may be noted that, for the traditional equation for veins, e.g., in (eq. I.6) of Appendix 2,

\[
Q_{v10} = C_{10} \frac{d}{dt}(p_{p} - p_{e}) \quad \text{(eq. 9.6)}
\]

i.e., only the pulsatile components of the pressures influence the computation of flow.

In equation I.81, which describes the transmural pressure-volume relationship of the left ventricle, a ‘±’ sign has been added to the diastolic term. The ‘minus’ sign prevents ventricular compliance from going negative for small ventricular volumes. These values lie outside the normal physiological range, but may, potentially, be required for CPR situations.\(^{35}\) Such measurements do not appear to be available for the right ventricle.

Cardiopulmonary resuscitation is performed by applying ‘p e(t)’ to all or selected structures in the model. This can be done with or without respiration being applied as another source of intrathoracic pressure. The frequency of compression can be varied.

8.5 Results

In this section the Donders model is used to describe well and lesser known entities such as resting, exercise and CPR conditions.

8.5.1 Validation of the model in resting conditions

Resting conditions, with the (70 kg, male) ‘patient’ in the supine position, without the effect of respiration (p e(t)=0) is shown in Figure 7, A through D, under steady state conditions. Three consecutive beats are shown at a resting heart rate of 60 beats min\(^{-1}\). Initial conditions include 6600 ml of blood volume. The systemic peripheral resistance, composed of six resistors at various points in the circulation, all in parallel (Appendix 2) is 1.2 mm Hg sec ml\(^{-1}\). Table II lists the other parameter values.

Figure 7A focuses on the area in and around the left ventricle. The heart is contracting at 60 beats min\(^{-1}\). The sharp negative excursions signify the instants of valve closure, in this case of the aortic valve. Depicted are left ventricular pressure (p\(_6\)) and root aortic pressure (p\(_{6^*}\)) and ejection flow (Q\(_{H14}\)). End-diastolic volume = 90, stroke volume = 51, cardiac output (CO) is 3.0 L min\(^{-1}\). The ejection fraction, EF = 0.56.

Figure 7B shows pressure in the left atrium (p\(_{501}\)) together with flow exiting the pulmonary veins (Q\(_{H11}\)), flow through the mitral valve (Q\(_{H12}\)), and left ventricular volume (V\(_9\)). Venous pressures oscillate between 13 and 16 mm Hg, with rapid initial ventricular filling. The mitral valve is closed by the small negative flow.

Figure 7B: No respiration included (p\(_e\) = 0). Left atrial pressure (p\(_{501}\)), pulmonary vein outflow (Q\(_{H11}\)), mitral valve flow (Q\(_{H12}\)), and left ventricular volume (V\(_9\)).
In Figure 7C the focus shifts to the right heart. It illustrates right ventricular outflow ($Q_{H8}$) together with right ventricular ($p_2$) and root pulmonary artery ($p_3$) pressures. End-diastolic volume is 92 ml, with a stroke volume ($V_s$) of 51 ml.

![Figure 7C: No respiration included ($p_e = 0$). Right ventricular pressure ($p_2$), root pulmonary artery pressure ($p_3$), right ventricular outflow ($Q_{H8}$), EF = 0.55.](image)

In parallel with Figure 7B, the curves in Figure 7D shows right atrial pressure ($p_1$), with flow through the tricuspid valve ($Q_{H6}$), the flow through the pulmonary valve ($Q_{H8}$), as well as the right ventricular volume ($V_5$). The atrial ‘kick’ can be recognized at the end of passive filling, with the effect of $k_1$ and $k_2$ as the ventricular ejection effect somewhat overstated.
In Figure 7C the focus shifts to the right heart. It illustrates right ventricular outflow (Q_{H8}) together with right ventricular (p_2) and root pulmonary artery (p_3) pressures. End-diastolic volume is 92 ml, with a stroke volume (V_s) of 51 ml.

In parallel with Figure 7B, the curves in Figure 7D shows right atrial pressure (p_1), with flow through the tricuspid valve (Q_{H6}), the flow through the pulmonary valve (Q_{H8}), as well as the right ventricular volume (V_5). The atrial 'kick' can be recognized at the end of passive filling, with the effect of k_1 and k_2 as the ventricular ejection effect somewhat overstated.

**Figure 7C:** No respiration included (p_e = 0). Right ventricular pressure (p_2), root pulmonary artery pressure (p_3), right ventricular outflow (Q_{H8}), EF = 0.55.

The addition of quiet respiration, in a ratio of one respiratory cycle (Figure 3) to three cardiac cycles is shown in Figure 8 using the same sequence of curves as in Figure 7. The function ‘p_e(t)’, depicted in Figure 3 is applied to all open circles in Appendix 2, with the exception of the abdominal veins (C_2). The time axis was chosen such that zero time in Figure 3 is analogous to ‘96’ in Figure 8, as the initial point in both cardiac and respiratory cycles. On this same basis, the beat with the smallest amplitude pressure excursion in Figure 8A and the beat with lowest value of left atrial pressure in Figure 8B coincide with the inspiratory phase in Figure 3. In Figure 8D inspiration is shown to make right atrial pressure slightly negative. Refilling of the right ventricle (Figure 8C) commences in the negative range as well, as an example of horizontal shift of the cardiac curve.

The final beat in Figure 8 occurs after expiration was complete. Cardiac output, computed from the three beats shown, equals 3.9 L min^{-1}, 28% above the level without respiration. Shifting the curve for p_e(t) along the time axis in Figure 3 by 0.5 sec such that inspiration takes place earlier, has a measurable effect on cardiac output, increasing it to 4.0 L min^{-1}, 35% higher than without respiration.
Figure 8:
As Figure 7, but with quiet respiration, applying the pressure \((p(t))\) in Figure 3 to all open circles in Figure 2, with the exception of the abdominal veins \((C_2)\), locations. The same quantities are displayed in the same order as in Figure 7, showing the effects of quiet respiration. Stroke volumes are for the left ventricle 66.0, 67.5 and 67.3 ml in chronological order of the beats shown. For the right ventricle, the stroke volumes are 63.7, 64.2 and 65.0 ml, making the overall average 65.7 ml. \(CO = 3.9\) L min\(^{-1}\) amounting to an increase of 28% over the value without respiration (Figure 7).
Figure 8: As Figure 7, but with quiet respiration, applying the pressure \( p_r(t) \) in Figure 3 to all open circles in Figure 2, with the exception of the abdominal veins \( C_2 \), [10 locations]. The same quantities are displayed in the same order as in Figure 7, showing the effects of quiet respiration. Stroke volumes are for the left ventricle 66.0, 67.5 and 67.3 ml in chronological order of the beats shown. For the right ventricle, the stroke volumes are 63.7, 64.2 and 65.0 ml, making the overall average 65.7 ml. CO = 3.9 L min\(^{-1}\) amounting to an increase of 28% over the value without respiration (Figure 7).
Coronary blood flow \( Q_{co} \), during light respiration is reproduced together with left ventricular pressure \( p_v(t) \) in Figure 9 with the dominant flow occurring during ventricular diastole. Coronary flow is enhanced while left ventricular pressure drops to the negative level.

8.5.2 Control influence on cardiac output and venous return

A series of 16 experiments was carried out on the model to expose the effects of the controls in the model under various combinations. They are summarized in Table I. In all cases the mean circulatory filling pressure in the closed loop, under no-flow conditions is 14 mm Hg.
Chapter 8: CVe2, the circulation in normo- and pathophysiology

Coronary blood flow (Q co), during light respiration is reproduced together with left ventricular pressure (p 6(t)) in Figure 9 with the dominant flow occurring during ventricular diastole. Coronary flow is enhanced while left ventricular pressure drops to the negative level.

8.5.2 Control influence on cardiac output and venous return

A series of 16 experiments was carried out on the model to expose the effects of the controls in the model under various combinations. They are summarized in Table I. In all cases the mean circulatory filling pressure in the closed loop, under no-flow conditions is 14 mm Hg.

Figure 9: Left ventricular pressure (p 6(t)) in synchrony with coronary flow (Q CO(t)) during quiet respiration. The value of the resistor R COV (Figure 2) changes with ventricular pressure to represent compression and release of the coronary veins. The average value of the depicted coronary flow falls below that of the uncompressed coronary vasculature.

Table I: Steady state cardiac output (CO) under 16 control conditions (values are marked with an asterisk above columns numbered 1-16) and specified in the bottom five rows. These are neural control of cardiac contractile properties (defined as atrial and ventricular c-values); control by respiratory rate (marked as resp. rate in cycles second\(^{-1}\)); control of respiratory depth (marked as the average value of p e); and the baroreceptor control, (marked as barorep and listed as percentage of the default values of the systemic and pulmonary peripheral resistances). In columns 7–16, the average pressures in the right atrium (RA) the left atrium (LA), in the aortic root and in the pulmonary trunk are also listed (in mm Hg). Where applicable, averages are taken over a complete respiratory cycle.

Each column in the table represents a different set of conditions, which are briefly characterized. The cardiac output (CO) belonging to each column is plotted in the upper half of the table as points to allow visualization of the effects. Column 1 refers to resting conditions without respiration and shows a CO of 3.0 L min\(^{-1}\), as demonstrated in more detail in Figure 7. The addition of quiet respiration, (column 2), raises that to 3.9 L min\(^{-1}\), which undergoes a further slight increase, (column 3), to 4.0 L min\(^{-1}\) when inspiration is aligned with ventricular contraction. In column 4, the heart rate is increased from 1.0 to 1.3 Hz (60 to 80 beats min\(^{-1}\)), which leads to a reduction of CO to 3.6 L min\(^{-1}\). Increase of respiratory rate from 0.33 to 0.44 Hz (one respiratory cycle in 3.0 sec becomes one in 2.25 sec) leaves CO unchanged (column 5).

Chapter 8: CVe2, the circulation in normo- and pathophysiology

175
Continuing under these conditions, the addition of weak neural stimulation (column 6), acting
on the contractile properties of both atrial and ventricular ‘c’ raises CO to 4.2 L min⁻¹. Stronger
stimulation, and adding a 50% reduction in pulmonary and systemic peripheral resistances,
(column 7) to the increased cardiac rate, augments CO to 5.5 L min⁻¹. Further reduction of
peripheral resistances to 25% of normal increases CO further to 5.7 L min⁻¹ (column 8).
In column 9 the respiration was stopped and the cardiac rate was returned to resting (60 min⁻¹)
with the peripheral resistances returned to 50% values. CO dropped to 4.6 L min⁻¹. Reinstitution
of quiet respiration returned CO to 5.3 L min⁻¹ (column 10), while deep respiration raised it to
6.4 L min⁻¹ (column 11). Turning off respiration, while reducing peripheral resistance value to
25% drops CO to 4.9 L min⁻¹ (column 12). In column 13, quiet respiration is restored, boosting
CO to 5.5 L min⁻¹, under a low peripheral resistance. Heart rate is increased in column 14, as is
respiratory rate and depth, resulting in a CO of 6.7 L min⁻¹. In column 15, stronger stimulation
of ventricular contraction does not significantly alter CO, while return to quiet heart and quiet
respiratory rates with deep breathing, escalates CO to 8.4 L min⁻¹.

8.5.3 Resuscitation after circulatory collapse

The Donders model can also serve to elucidate conditions in circulatory collapse by relating the
two popular theories on mechanisms responsible for restarting the circulation: the cardiac pump
theory, originally proposed by Kouwenhoven et al.,⁹ or the thoracic pump theory, as proposed
by Criley et al.³⁶ Both theories share the procedure of compressing the thoracic cavity.

- Chest compression and intrathoracic pressure. The external pressure applied on the
chest is referred to as pₑ*(t), which is a function of time. The pressure actually
experienced by the tissue is less owing to an attenuation factor, A, in the transition from
the bony structure to this tissue, hence:

  \[ pₑ(t) = A pₑ*(t), \text{ with } A < 1 \]

  (e.q. 9.7)

Kouwenhoven’s original conceptualization of CPR required the application of pₑ*(t) to
the left ventricle, by squeezing the heart between the sternum and the vertebral column.
Recent studies reiterated reasons why this concept might be weak. First, the left
ventricle is not positioned in the line of compression and second, the shape and depth of
the chest are often such that the degree of compression suggested is too small to actually
achieve left ventricular compression.³⁸ The position of the right ventricle makes it far
more susceptible to direct compression. Nonetheless, the cardiac pump theory remains
the theory taught in most CPR courses.

Reinterpretation of the cardiac pump theory in this spirit requires isolated application of
pₑ*(t) to the left ventricle. The original theory also allowed for atrial refilling during
compression systole, requiring a competent mitral value. Application of pₑ*(t) to the
relaxed, blood-containing left ventricle is expected to drive some, or all, of the blood out
through the aortic valve, the mitral valve not permitting retrograde flow. Early work
suggested that cardiac output should be at least 30% of normal if the ventricle was
compressed ‘correctly’ between sternum and vertebral column.³⁸⁹ Although current
clinical practice is 100 compressions min⁻¹ (cpm), we have used the conditions common
at that time (60 cpm). The reason for this choice will become clear below.

- The magnitude of intrathoracic and left ventricular pressures (pₑ and pₑ). Inspection of
equation I.81 reveals that the difference between pₑ and pₑ for the relaxed, or asystolic,
ventricle (Fₑᵥ = 0) is sensitive to the blood volume contained by this chamber. For small
volumes within the ventricle these pressures become indistinguishable. This is
illustrated in Figure 10, where computer solutions are reproduced in which the peak value of $p_c$ was carefully selected such that ejection is complete ($\text{EF}=1$, dash-dot graph), which occurs for a $p_{\text{max}}$ of 26 mm Hg when the static pressure equals 14 mm Hg, and initial conditions were the default values. The stroke volume is 16 ml. Since the minimum value of $p_c$ equals zero when the chest is in its resting state, refilling occurs during the phase that $p_o$ falls below the corresponding atrial and venous pressures. The two flows displayed in the bottom half of fig 10 represent filling and emptying of the left ventricle. The ejection duration is 338 msec, considerably longer than in the normal ventricular cycle.

**Figure 10:** Left ventricular inflow ($Q_{\text{in}}$) and outflow ($Q_{\text{out}}$), as well as ventricular volume ($V_9$) under CPR conditions. The ventricle is completely emptied by application of $p_e(t)$. The tracings of $p_o$ and $p_e$, the ventricular cavity pressure are so close as to be indistinguishable. The ejection fraction (EF) equals 1 (complete emptying of the cavity).

- **Alteration of the stroke volume.** Intuition suggests that a higher value for $p_e$ might augment the small stroke volume in Figure 10 and this approach has been systematically advocated in clinical studies and practice. Equation I.81 shows that $p_6-p_e$ will increase if the left ventricular volume increases. This is the case as the mean circulatory filling pressure is increased (Figure 11). This pressure is raised to 25 mm Hg, as occurs under collapse conditions. Left ventricular volume during CPR diastole increases to 160 ml, well above the 16 ml in Figure 10. The maximum effective value for $p_e$ could be increased to 64 mm Hg, and the peak for $p_6$ became 80 mm Hg as a result. Nonetheless, the stroke volume decreased to 11 ml, well below the 16 ml in Figure 10. This undermines the intuitive expectation. Analysis identifies the cause of the decreased ejection by the ventricle: the ejection time is 200 msec, a value shorter than that in the normal cardiac cycle, with $V_9$ oscillating around the 155 ml. The right ventricle (Figure 12) demonstrates similar phenomena at a lower volume level.
Augmentation of stroke volume might lie in the increased duration of $p_c$ at its peak value, i.e., creation of a plateau. Such a procedure would bring the pressure curves in Figure 11 closer together, similar to those in Figure 12 for a smaller volume. A variable distance between atrial pressures and $p_c$ tracings is more clearly seen in Figure 13 and 14. Here the most conspicuous curves are those representing flows through the vеноatrial junctions which indicate major sloshing, allowed by to absence of valves at the junctions.

![Graph showing circulatory changes](image)

**Figure 11**: The left side of the circulation under CPR conditions. Shown is $Q_{H12}$, the flow over the mitral valve, $Q_{H14}$, the flow into the characteristic impedance of the aorta and $V_9$, the volume of the left ventricle, in relationship to $p_6$, the pressure in the left ventricle and $p_e$, the compression pressure as expressed on the ventricle. Note that these pressures are clearly distinguishable at the larger ventricular volume.

- **Collapse of veins.** In Figure 10, it was shown that application of a $p_c$ of 26 mm Hg could empty the ventricle under these specific conditions. For larger veins the corresponding number is much smaller, in the order of a few mm Hg, as found by Guyton. Consequently, chest compressions as practiced in clinical CPR will shut down large segments of the central veins during most of the chest compression phase, transferring blood primarily to the extrathoracic veins, and holding the volume there. Where high chest compression pressures are brought to bear, compression of the thoracic arteries might occur as well. There is a strong tendency to apply to the chest much higher peak values in clinical practice.

- **Achievement of high pressures in the left ventricle.** A few investigators have attempted to raise left ventricular pressures to higher levels in an effort to promote coronary and cerebral perfusion during CPR. Both goals have, occasionally, been crowned with success, both with little improvement in clinical outcome. The model can identify what happened during these efforts. In the model the mean circulatory filling pressure was
raised to 20 mm Hg while the peripheral resistances in the abdominal and leg circuits (\(R_2\) and \(R_{ss}\)) were raised to 30 times their default values and \(p_{\text{max}}\) was raised from a value of 15 to 30 mm Hg. The \(p_e\) was applied to the left atrium and ventricle, with the former including the remedy to prevent occurrence of negative volumes\(^{35}\). Compression frequency was at 1 Hz. These measurements raised peak left ventricular pressure from 22 to 48 mm Hg with left ventricular volume oscillation ranging from 70/67 to 102/93 ml compared to the application of \(p_e\) of 15 mm Hg only. Additional manipulation of \(R_{\text{esr}}\), but not \(R_{\text{esr}}\), by a factor of 10, and raising \(p_{\text{max}}\) to 70 mm Hg increased peak ventricular pressure to 80 mm Hg, and left ventricular volume swings to 138/127 ml. Such measures permit institution of high ventricular pressures albeit accompanied with tiny stroke volumes.

### 8.6 Discussion

In this paper we present a succinct model suitable for both general and specific examinations of the circulatory system. After introducing the model, and placing it in historical and scientific perspective, its operation is demonstrated in the resting state. This is followed by a compilation of physiological control effects, clearly recognizable from a life science point of view. Finally, a brief demonstration of the model’s value in cardiopulmonary resuscitation is presented.

#### 8.6.1 The Donders model in relationship to simulation and other models

In general, simulations have been suggested to be attempts to reproduce reality, using any mechanism which may produce the desired effect or result. Mathematical models, far more restricted, must use mechanisms in evidence to produce its effects.\(^{41}\) Models may, as is the case here, lead to conceptual interpretations of observations which can not be understood or answered via another medium. Detailed model studies of segments of the circulation have made it possible to distinguish between major and minor features in the operation of such segments. This has made it feasible to significantly reduce the number of equations needed to model such major features. An example is furnished by modeling of arterial systems where the number of equations could be reduced from a few hundred to less than ten.\(^{7}\) Framework models, built for use in experimentation, have also been presented.\(^{42}\)

We suggest that there were two primary reasons which support the development of a new model for the circulation, both of which apply here. First, to enable an investigator to carry out experiments that cannot be performed on the animal or on the human, or are ethically unacceptable to execute. Second, to develop, evaluate and integrate new insights, based on clinical observations, at a higher level of the closed circulatory loop. Such a study can make beneficial use of earlier reductions of segmental models, thereby enhancing readability and clarity of a more encompassing model. This improvement will also facilitate a better understanding of complex phenomena.

Examples of ethically imposed restrictions occur in the analysis of events surrounding cardiopulmonary resuscitation that baffled understanding for more than 60 years, even though other researchers have advocated the use of models to improve insights and clinical outcome.\(^{43}\) In 2006, in part based on data obtained from experiments in swine, new international guidelines will be introduced, even though investigators recognize physiological differences between swine and humans (e.g., the position of the heart in the chest).

#### 8.6.2 Control mechanisms and cardiac output

Exercise triggers sympathetic stimulation of the heart and augments the cardiac output by increase the contractile strength of the heart, increasing venous return, and increasing the heart rate. However, when isolated as an effect, the magnitude of this augmentation is limited by the filling of the right heart, the systemic venous pressure being low and falling owing to this
increased right ventricular uptake. Any increase in filling of the right heart will be transmitted to the left heart and induce a larger cardiac output. Neural stimulation in untrained persons tends to increase heart rate, reducing the duration of the filling period. This tends to encroach on the stroke volume. In exquisitely trained athletes, heart rate tends to decrease during heavy exercise, thereby increasing stroke volume. Metabolic stimulation, analogous to neural stimulation, operates on the cardiac musculature by rotating the cardiac function curve, as the representative of the original Starling’s law, counterclockwise, which makes it steeper and taller. Depression turns it clockwise, reducing stroke volume at the same filling level. Thus, the cardiac function curve adjusts itself in response to prevailing demands.

The normal system appears to rely on the contribution of all controls to answer a demand for above resting cardiac output for any human specimen. This means that magnification of cardiac output will depend on how well the individual contributions are attuned to one another. The reason for this is that the individual adjustments are arranged in series, implying that if even one fails, the system will find it difficult to adjust cardiac output properly. A parallel arrangement, as utilized in the distribution of flow among the peripheral organs, would tend to offer more flexibility if it were applied to all flow controls. During exercise, cardiac output has been found to increase up to six times normal in Olympic quality sporters during strenuous physical activity, four times being measured more commonly. These are changes in which the entire cardiovascular circuit is involved, and include interaction with the respiratory, metabolic, and neural systems.

Many experimental studies relating to cardiac output and venous return have been performed since Donders’ classical observations. The vast majority of the more recent studies in animals and on isolated animal parts share the difficulties of complex experiments: critical parameter values go unmeasured, complicating interpretation of the observations. The use of a model creates, in principle, a method to avoid such barriers. Table I may serve as an illustration. Cardiac output is shown as function of a variety of controls, all quantified and described is detail in section 5.2. The ratio between maximum and minimum cardiac output in Table I is 2.8. No effort was made to maximize this ratio. It is interesting to note that some control functions (such as an isolated increase of heart rate) can reduce cardiac output, rather than enhance it.

8.6.3 Impedance defined flow, Starling and Donders

We reported that broad concern was expressed about the ability of the heart to provide adequate venous return. What Starling called “the law of the heart” represented the relationship measured in his heart-lung preparation between filling pressure and ventricular output. Renamed Starling’s law, its validity became subject of doubt, the solution to which appeared to lie in the introduction of cardiac function curves, the latter allowing for neural and metabolic stimuli, in addition to the filling pressure and the concomitant introduction of impedance defined flow. Performance of the ventricle also was found to be sensitive to the arterial load and to ejection flow. Hence, it proved to be a function of not only the preload (filling), but also of the arterial load, flow generated, and stimuli from other systems. The heart, as the only generator of net flow, lost its perceived autonomous status and became a servant to the needs of peripheral organs.

Impedance defined flow deals quantitatively with net flow under conditions of chamber or vessel contraction and relaxation, chamber or vessel compression and release, alteration in gravity fields, acceleration and deceleration of the cardiovascular system as a whole, in situations where the circuit features one-way valves, has incompetent valves or is altogether free of valves. The availability of such a quantifiable impedance-defined flow concept facilitates the acquisition of additional analyses. Note, for example that in Figure 8A p_e goes negative.
This phenomenon is related to the horizontal shift of the cardiac function curve expected in impedance defined flow, and is directly related to the respiratory action (Figure 6).

These aspects are not contradictory with previous findings, if these are seen in their context. All of the early experiments were carried out at atmospheric pressure. If surrounding respiratory pressure is taken into account as demonstrated in Table I and Figure 6, the cardiac function curve is shifted to the left along the filling axis for negative air pressure and to the right for positive ones (as occurs by chest compression in CPR, as most clearly demonstrated by the thoracic pump CPR advocates. Intrathoracic pressure changes will operate on the vascular sections located in the thorax, including, but not limited to, the four cardiac chambers. The cardiac chambers, protected by the pericardium may, in fact, be far less sensitive to pressure changes than the pulmonary vasculature and the two cavae.

With respect to Donders’ claim about respiratory support of the circulation, comparison of columns 1 and 2 and Table I, as well as columns 10 and 11, shows augmentation of cardiac outputs by 30% and 21%, respectively, while all other parameters are left unchanged. This leaves little room for doubt about the validity of Donders’ claim.

8.6.4 Cardiopulmonary resuscitation and the Donders model
In more than 60 years of research into the treatment of cardiac arrest, little has been done with models, and then only in specific cases. Most of these models have been used to investigate and compare the effects of adjuvant techniques. Babbs et al. [1983] initiated mathematical descriptions during investigations into mechanisms most suitable for generating output. This team advocated use of $p_e(t)$ to influence abdominal pressure in order to raise impedance to caudal blood flow. To demonstrate this they developed a series of electrical models of the circulation, consistently and carefully demonstrating and motivating that and other potential aspects in CPR susceptible to change. Regrettably, the potential of models and their results have been studiously ignored.

Few others, such as Beyar and Goldstein and Halperin have made models suitable for CPR, in which only Halperin’s model contained a functional rudimentary heart. Although many of the adjuvant techniques involved impedance defined flow aspects (typically class II: external pumping of vessels) animal models remained the standard for research, despite concerns in anatomy, physiology, and morphology.

The $p_e(t)$ operating on the intrathoracic structures is still at issue. In the model we distinguish between $p_e^*(t)$ operating on the outside thoracic wall as opposed to $p_e(t)$ which actually operates on the organs, causing impedance defined flow up through successive collapse of structures as $p_e(t)$ increases. If peak esophageal pressure may be taken as indicative of $p_e(t)$ a range of 10-50 (max of 100) mm Hg seems reasonable, allowing for an attenuation factor of $10^{-1}$ to $10^{-2}$ from $p_e^*(t)$. Early indications are that clinical goals to create high intrathoracic pressures may, in fact, be counterproductive above a certain level of pressure. This corroborates expectations of vascular collapse due to $Z$-flow with collapse of interstitial structures and vessels. This phenomenon may have been observed, but not recognized, by Babbs.

Figure 10 illustrates the basic issue of small stroke volumes. At the bottom left ventricular inflow $Q_{h1}$ and ejection flow $Q_{h1}$ alternate under the influence of CPR pressure $p_e(t)$. Ventricular pressure $p_v$ is indistinguishable from $p_e(t)$ owing to the small ventricular volume $V_v$ (eq. I.81). Compression pressure $p_e(t)$ was selected such that the ejection fraction is 100%. Stroke volume is 16 ml. In an intuition based effect to enlarge stroke volume $V_v$ was increased. This separates $p_v$ and $p_e$ as $p_e$ is increased. Stroke volume becomes smaller instead of larger. Figure 12 demonstrates the phenomena of Figure 11 for the right ventricle with a volume just
Chapter 8: CVe2, the circulation in normo- and pathophysiology

large enough to separate the right ventricular pressure $p_4$ and $p_5(t)$. Figures 13 and 14 apply to the left and right atrium, respectively. The absence of inlet valves permits sloshing to occur, in turn causing variable separation between $p_5(t)$ and the atrial pressures $p_{501}$ and $p_1$. Total negative flow in Figure 13 and 14 equals 39 ml, total flow into the left ventricle equals 6 ml, while the total atrial inflow equals 44 ml.

Figure 12: As Figure 11, but for the right ventricle.

Figure 13: As Figure 11, but for the left atrium. Note vigorous sloshing and the resulting variable distance between the $p_5(t)$ and $p_{501}(t)$ tracings.

Figure 14: As Figure 13, but for the right atrium.
8.6.5 Limitations of the model

The present study was to produce and use a new flexible model of the circulation. It is defined by 88 differential and algebraic equations, and describes pressures and flows in detail in thoracic cavities and generally in the peripheral vasculature. A model remains a simplification of reality, in which choices have been made. Our intention was to focus on the essence of impedance defined flow, and $p_e(t)$ instead of incorporating all possible details. For example, the peripheral circulation is strongly simplified, and there is no vertebral circulation which may be a major factor in intracerebral pressure during resting and CPR conditions. In addition, the circulation has been modeled as a tightly closed loop, not allowing for lymphatic or third space shifts which should be recognized in both exercise and CPR.

Mechanical aspects, such as variable stiffness of the thoracic cavity, airway effects and lateral shift of the heart during CPR are difficult to model in a mathematical model. Different mathematical aspects also limit possibilities, such as the opportunity for the model to show negative volumes in vessels and chambers requiring a remedy.

8.7 Conclusions

The Donders model of the circulation is a moderately lumped RLC model, which is based in physiology, incorporates a detailed intrathoracic portion and introducing the concept of impedance defined flow into a functional model. The model is specifically suitable to be applied to a range of physiological and extreme conditions such as cardiopulmonary resuscitation.

The model is validated showing the effects of increased venous return, increased and decreased sympathetic effects, and heart rate, including motivation for the concepts involved in impedance defined flow.

In addition, conceptually simple but clinically impossible examples are shown involving the pure use of cardiac left ventricular and pericardial (all atria and ventricles) compression, and
the effects this has on the cardiac output. A preliminary concern was raised against the idea that more force application transition is better in CPR.
8.8 References

1 Harvey W. Exercitatio Anatomica, De Motu Cordis et Sanguinis in Animalibus. Frankford D. 1628.
2 Caesalpinus A. Quaestionum Medicarum, Liber secundus, p. 234, Venice I. 1593.
20 Patterson SW and Starling EH. On the mechanical factors which determine the output of the ventricles. J. Physiol. 1914; 48: 357-379.


Supporting the use of modeling in CPR: things (not) in evidence
Chapter 9: Things (not) in evidence

9.1 Introduction
The previous Chapters have approached cardiopulmonary hemodynamics from a theoretical or physiological point of view. They have focused on a broad, basic, analysis of what modeling can do, without focusing on explicit issues directly relevant to the clinician. This Chapter takes the first step towards functional, clinical medicine, and addresses some aspects within CPR using the Donders model, but coupling this to practical opportunities.

The use of modeling in CPR has, as yet, few hardy proponents, although it has been described to have found a 'niche'. The last major input with clinical consequences, was the 30:2 compression-relaxation ratio proposed by Babbs et al., and used in the 2005 guidelines. Research remains focused on applicability issues, attempting to use controlled trials, case series and animal experiments even though there are voices advocating some small change in this policy. None the less, even the resuscitation community has noted difficulties with that plan of attack: such as demonstrated in the ASPIRE study as well as in the HAT study.

With the 2010 guidelines looming in the horizon, central issues, such as mechanisms which might impact outcome in cardiopulmonary resuscitation, are very much of interest. The 2005 guidelines, clearly stating in the introductory editorial, that "choices had been made based on expert opinion" further recognized "that scientific evidence is scant and opinions are strong". This editorial may have opened up the road to new practice, which will allow for new insights to be achieved. If, for example a Randomized Controlled Trial (RCT) had been proposed in 2003 suggesting a single shock therapy for ventricular fibrillation, any ethical committee would have been challenged to accept this: a stacked, three shock, strategy had the benefit of a long history, as well as defibrillation having the status of the only evidence based therapy in CPR, with a 7% decrease in the probability of survival per minute, if not shocked. By placing techniques in context of overall goals, individual components in therapy may be highlighted and come to be better understood.

This Chapter, forming a bridge between the theoretical (modeling) aspects in CPR research and more finite, practical aspects in CPR, uses coronary flow, stroke volume per se, and a new concept of volume loss, to illustrate practical aspects, but which have not yet matured to full fruition. The Chapter also serves to wet the appetite for Chapter 16, which places the whole project in a more clinical perspective.

9.2 Qco: a non issue?

Few will challenge that the essential single factor in sudden cardiac death is coronary thrombosis or, more specifically, myocardial hypoperfusion. This has been suggested in at least 65% of cases of sudden cardiac death. However, research into the specific conduct of the coronary circulation, and potential opportunities to address issues, has been limited.

9.2.1 The coronary circulation in and out of no-flow conditions

The flow through the coronary circulation in man has been difficult to quantify, but has been estimated to be 0.7 – 0.8 ml gr-1 of cardiac muscle, or some 4-5% of left ventricular output at rest (i.e. ± 225 ml min -1). At rest, this flow allows 65-70% oxygen extraction. During strenuous
9.1 Introduction

The previous Chapters have approached cardiopulmonary hemodynamics from a theoretical or physiological point of view. They have focused on a broad, basic, analysis of what modeling can do, without focusing on explicit issues directly relevant to the clinician. This Chapter takes the first step towards functional, clinical medicine, and addresses some aspects within CPR using the Donders model, but coupling this to practical opportunities.

The use of modeling in CPR has, as yet, few hardy proponents, although it has been described to have found a ‘niche’. The last major input with clinical consequences, was the 30:2 compression-relaxation ratio proposed by Babbs et al., and used in the 2005 guidelines. Research remains focused on applicability issues, attempting to use controlled trials, case series and animal experiments even though there are voices advocating some small change in this policy. None the less, even the resuscitation community has noted difficulties with that plan of attack: such as demonstrated in the ASPIRE study as well as in the HAT study.

With the 2010 guidelines looming in the horizon, central issues, such as mechanisms which might impact outcome in cardiopulmonary resuscitation, are very much of interest. The 2005 guidelines, clearly stating in the introductory editorial, that “choices had been made based on expert opinion”, further recognized “that scientific evidence is scant and opinions are strong”. This editorial may have opened up the road to new practice, which will allow for new insights to be achieved. If, for example a Randomized Controlled Trial (RCT) had been proposed in 2003 suggesting a single shock therapy for ventricular fibrillation, any ethical committee would have been challenged to accept this: a stacked, three shock, strategy had the benefit of a long history, as well as defibrillation having the status of the only evidence based therapy in CPR, with a 7% decrease in the probability of survival per minute, if not shocked. By placing techniques in context of overall goals, individual components in therapy may be highlighted and come to be better understood.

This Chapter, forming a bridge between the theoretical (modeling) aspects in CPR research and more finite, practical aspects in CPR, uses coronary flow, stroke volume per se, and a new concept of volume loss, to illustrate practical aspects, but which have not yet matured to full fruition. The Chapter also serves to wet the appetite for Chapter 16, which places the whole project in a more clinical perspective.

9.2 Qco: a non issue?

Few will challenge that the essential single factor in sudden cardiac death is coronary thrombosis or, more specifically, myocardial hypoperfusion. This has been suggested in at least 65% of cases of sudden cardiac death. However, research into the specific conduct of the coronary circulation, and potential opportunities to address issues, has been limited.

9.2.1 The coronary circulation in and out of no-flow conditions

The flow through the coronary circulation in man has been difficult to quantify, but has been estimated to be 0.7 – 0.8 ml gr⁻¹ of cardiac muscle, or some 4-5% of left ventricular output at rest (i.e. ± 225 ml min⁻¹). At rest, this flow allows 65-70% oxygen extraction. During strenuous
exercise this flow may increase four to five-fold in the face of a cardiac workload increase of up to eight times. During contraction, an epicardial – endocardial (intra-myocardial) gradient develops, with the endocardium being exposed to pressures equal to those of blood, although flow in both epicardial and endocardial arteries is effectively limited to diastole. The endocardium is, however, more susceptible to hypoxia if its autoregulatory capacity is limited.14 Not surprisingly, during systole the flow in the arteries may be reversed, while more surprisingly venous outflow is initially augmented. This suggests that the vessels themselves do not collapse immediately.15, 16 Under conditions of normal flows and pressure (wave forms) a coronary stenosis of more than 70% is needed to adversely impact perfusion as intact autoregulatory systems control for this.8 The system of autoregulation is complex, involving local metabolism (oxygen, adenosine, etc) as well as autonomic regulatory mechanism (i.e. the sympathetic as there are few vasodilatory, vagal, parasympathetic fibers). The sympathetic receptor density varies based on location and therefore their effects can be variably expressed.

During cardiac arrest, this physiology is disturbed.17 Autoregulatory control is lost, and within 30 seconds maximal coronary vasodilation is present, making flow pressure gradient dependent.8 Ventricular fibrillation has been shown to obstruct both epicardial and endocardial blood flow via a high myocardial tonus by increasing coronary vascular resistance and increase oxygen extraction, even if coronary perfusion pressures are maintained.22 However, Downey et al. have suggested that the reduction in flow is uniformly distributed throughout the myocardial wall, after adenosine was used to create dilatation before VF and that perfusion pressure remains the principal regulatory mechanism.23

As suggested in the sections above, one of the principle goals of CPR is to induce sufficient coronary flow to re-energize the myocardium, allowing coordinated activity to restart. Issues are, of course that many of the (animal) models to not consider the effect of persisting coronary defects and thrombosis.

The effect of a coronary stenosis under low-flow or CPR conditions has also received some attention. In particular, Kern et al., as recently as during the 1990’s,24,25,26 suggested that the coronary perfusion pressure (CPP, Chapter 2) recognized since the 1980’s,21,28 and its clinical surrogate end-tidal CO2,29 become the important indicator for success (i.e. ROSC). The 15 mmHg boundary may even be useful for prognostification.30 CPP’s lower than 15 mmHg are indicative of failure and (30-) 40 mmHg suggestive of positive outcome.31 However, as Kern et al.,26 and Ditchey et al.,20 demonstrated, seemingly adequate CPP’s may not actually reflect myocardial perfusion. In line with this, a CPI (coronary perfusion index) has been suggested.32 The effects of medication, including vasopressin have given some insights.33 From the elegant studies by Kern et al.,34,35 both time sensitivity in percentage of flow, altering sensitivity to vaso-active substances, and the critical difference between epicardial flow and myocardial perfusion, have been addressed.16,17 This has, more recently been complemented by Berg et al.,36 and later by Steen et al.,37 who demonstrated in a swine model, that coronary perfusion falls rapidly, requiring some period of time to be re-established,36 and is an important factor in success in defibrillation, and that a negative coronary perfusion pressure is possible even in healthy vessels. This has led to voices advocating uninterrupted chest compressions.38

This step, creating the mathematically optimal CPP as a translation from physiology to external chest compressions may have neglected other factors. Are there other factors, such as how does the coronary circulation conduct itself under CPR: are ventilations beneficial or detrimental to flow and may brief interruptions for clinical tasks or monitoring be allowed without concern under both basic and advanced conditions.
This paragraph reports on the use of a flexible mathematical model of the human circulation (Donders-II), designed for CPR conditions and unique in its physiology to describe manipulation of intrathoracic pressure during simulated compressions.\textsuperscript{39} Approaching the circulation from a physiological standpoint,\textsuperscript{40} we focus briefly on mechanisms involved in coronary perfusion during CPR and introduce thinking about developments. It illustrates the potential value of mathematical modeling for those involved in clinical management during CPR.

9.2.2 Modeling Qco
Different factors seem to impact the coronary flow. These factors: a cumulative effect in a series of compressions, the impact of interruptions of flows and pressures, the effect of the compression per se on coronary vascular resistance, and the potential for ventilations to function as a pump during interruptions in chest compressions may have complementary or contradictory effects, each of which with a unique time sensitivity.

Presented to the ERC-UK meeting in 2006,\textsuperscript{*} we described this process of thinking using a modeling approach.

In this computer model (Donders-II), events in the coronary circulation have been strongly simplified, with, for this series of experiments, the resistance being sensitive to $p_6(t)$ as well as to the conduct of the diastolic properties of the myocardium. Coronary flows are small, with ca. 1.4 – 2 ml sec\textsuperscript{-1} and include a component of up to 65% of regurgitant flow.

In the model, the conduct of coronary circulation formulas (Donders II) are:

$$Q_{co}(t) = \frac{[p_7(t) - p_7(t)]}{[R_{cov} + R_{cov}]}$$  \hspace{1cm} (eq. 9.1)

$$R_{cov} = 0.6 \left[p_6(t) + p_{ec}c(t)\right] \text{ with } p_6 \geq p_{ec}$$  \hspace{1cm} (eq. 9.2)

When $p_7$ = filling pressure of the right heart, $p_7$ the pressure in the left ventricle, $p_7$ the pressure in the aortic root and $p_{ec}(t)$ the pressure applied to designated structures during compressions. Figure 1 shows that there is a brief period of ‘coasting’ in the Qco while the systemic load is redistributed to the venous capacitance vessels. The (mean) systolic pressure plummets, but the fall in diastolic pressure is slower. This effect is seen for some 5 seconds (Figure 2) after the last contraction, much more briefly than the 30 seconds until the flow suggested by the numeric CPP disappears as reported previously.\textsuperscript{28} Note that the rise in both right and left atrial pressure is not shown in the Figure, but follows the fall in diastolic pressure more than that of the systolic pressure.

Figure 1: Even though the heart starts beating ‘cardiac output’ does not immediately go to zero. This is supportive of impedance defined flow.

Figure 2: This coronary perfusion flow is reactive to changes in arterial to venous pressures causing both forward and regurgitant flow.

Ventilations, modeled as $p_{\text{end}}$, at 20 cmH$_2$O, have only a minor effect on cardiac output (flow through the aortic root), but a measurable, negative effect on forward Qco as $p_{\text{end}}$ impact right atrial pressures (and therefore the CPP) as well as flow gradients more than those in the left ventricle or aorta.

These data addressed two of the issues impacting coronary perfusion. First, that 5 seconds may be a more acceptable ventilation pause than the 10-15 seconds accepted for instruction purposes. And second, that ventilations, even at low pressures and of 1-second duration, have their own – negative- impact on residual Qco, inducing retrograde movement of up to 27%.

Where lack of coronary flow is the cause of cardiac arrest and resuscitation, external chest compressions generate flow through the (coronary) circulation, but also seem to interrupt this flow. During compressions – with forces at least sufficient to be effective (compression depth of...
2 cm, force of 25 kg\(^{42}\) – obstruct epicardial and endocardial flow. The optimal balance between compression time, relaxation time and interruptions has not yet been determined. That Qco is the central issue to improve ROSC and thereafter definitive positive outcomes is increasingly clear. The DIRE approach (Chapter 16) addresses this issue further.

Further analysis of the effects of all sorts of stenotic processes, as well as the use of epicardial and endocardial vascular structures, in addition to their connective vessels, each sensitive to their environment may give an impulse to clinicians in understanding the use of pressure, and support the development of devices which will limit Qco impact.\(^{43}\)

9.3 Stroke volumes in relationship to compression force

Chest compressions are intended to generate stroke volumes of sufficient volume to preserve life. The flows generated are low, classically suggested in the 30% range of normal cardiac output range, but are most likely even lower.\(^{44}\) Very little data is available in man,\(^{45}\) with experimental data from swine,\(^{46}\) and dogs\(^{47}\) of various weight categories being used more often in experimental conditions. End-tidal CO\(_2\) has also become the surrogate value for clinical use.\(^{48}\)

Measurement of cardiac output and more specifically stroke volume has eluded research scientists, despite the use of thermodilution techniques,\(^{49,50}\) cardiac ultrasound,\(^{51}\) radiolabeled spheres\(^{52,53}\) and dye systems as well as burr hole laser-doppler flowmetry.\(^{50}\) Central to the discussion of cardiac output during external chest compressions is the discussion of how deep these compressions should be.\(^{54}\) Since its inception in 1960,\(^{55}\) chest compression force has been expressed in inches or centimeters of downward movement of the sternum.\(^{56}\) The use of a root aortic, femoral or aortic root pressure waves to determine cardiac output has a long history in CPR, and has more recently been introduced as a minimal invasive method for general monitoring purposes.\(^{57}\) Aside from the issue of what can be measured, the base discussion of force has still not been addressed. Clinically, the adage of “harder is better” is well known, and the 2005 Guidelines\(^{3}\) generally support this. From a modeling aspect, this question can be analyzed in a selective and sensitive way, as the effect of (changing) compliance of the chest wall and intra-thoracic tissues as well as that of the vasculature and the myocardium itself, can be ignored. Pressure, p\(_e(t)\), can be defined.

Historically, stroke volume has been strongly associated with pump theory. (Figure 3) The classic, cardiac pump, with recruitable volume only that in the left ventricle, focuses on the ability of the compressions to close the mitral valve and empty the left ventricle into the aorta. Thoracic pump theory incorporated the circa 300 ml of post-pulmonary valve blood volume as recruitable, making a stroke volume fraction significantly larger.
Using the Donders model (II) to evaluate stroke volume generates large differences based on compression force and technique. Separation of location, i.e. only on the left ventricle or on both ventricles, with and without the atria, allow insights into forward movement of blood. As an experimental range, using a 60 mmHg compression wave with a sine function and a 'dead volume pressure of 15 mmHg, an effective stroke volume of 2 ml was shown with pressure being applied to all structures in the torso, going up to 36 ml compression if pressure was limited to the two ventricles and the arterial system. Note that these pressures are well within range or be clinically effective.

The use of a mathematical model offers the first steps in insights into the conflicting data reported over the years for supposedly optimal compressions techniques. Small shifts in volume, subtle changes in compression form and, through changing compliance in the chest wall (with inverse changes in the pulmonary tissues), and to the myocardium, may identify the technique needed to be clinically effective.

---

1 Dutch Society Anesthesiology Research meeting. Sept. 2006 (Amsterdam, NL) Oral presentation
These types of data are needed to stimulate clinicians in the recognition of the need for bio-feedback systems, and support the suggestion that nuances in compression force-, depth, and waveforms, when resuscitation is in the hands of professionals, need to be considered. Careful steps in this direction are being made with mechanical CPR devices and ETCO₂ monitoring, and setting out the first steps to Patient directed resuscitation (PDR). Modeling remains the only method reliably available to coach pump theory towards optimal performance, particularly for an individual patient.

9.4 Volume sequestration

A third example of the bridge which modeling may bring to clinical CPR is that of volume shifts and the mechanisms responsible for this. No other disease in man, including hemorrhagic shock demonstrates such a complex of inappropriate cumulative mechanisms, and, historically, so few therapeutic options.

9.4.1 Intravascular volume and cardiac arrest

Immediately after cardiac arrest, intravascular volume redistributes towards the venous system throughout the body.65 This relatively slow process (minutes to reach mean circulatory pressure)⁷⁹ has been shown to cause right heart dilation and decreased venous return from the cerebral circulation, including intraventricular septum shift.⁷⁷ At the same time, devices to improve venous return have been introduced, such as the inspiratory threshold device (ITD or ResqPod).⁵⁶ Furthermore, there is evidence that capillary leakage and plasma loss may decrease circulating volume by up to 20 ml kg⁻¹,⁶² while other experimental evidence suggests that perfusion of intra-abdominal organs may be decreased.⁶⁸

Intravascular volume, or more precisely pre-load, may be an essential aspect in generating good output in CPR. While there is little experimental⁶⁹ or clinical experimentation done in this direction, the Donders Model (III) suggests that external chest compressions cause sequestration of volume,⁷⁰ defined as the movement or limitation in distribution of volume, to outside the thoracic cavity.⁷ Exercise, volume loading,⁷¹ while studied, has been limited to cases with PEA (pulseless electrical activity) as one of the four so called "4 H’s and 4 T’s".³

The functionality of pre-loading volume ‘loss’ during CPR seems to have become lost,⁷²,⁷³ potentially since manual compressions are limited to techniques humans can perform. In addition, volume loading has been associated with contradictory results in neurological outcome.⁷⁴,⁷⁵ This is attributable to the effect of increased central venous pressures on cerebral perfusion. Early in external chest compressions, recruitment of peripheral volume, as well as selective increase in peripheral resistance (including but not limited to vascular resistance) was introduced.⁷⁶ The low pressures involved in artificial ventilation have been shown to cause sequestration, as well as impact the differing input by the superior and inferior caval veins.⁷⁷

The Donders model (III) allows for a further analysis in this area of contradictory evidence, where vessels collapse, intrathoracic pressures, actual intravascular volume and peripheral resistances seem to be operating in concert.

Volume sequestration and sloshing

The Donders model (III) has been extended to allow for investigation of the conduct of the central vascular structures. Central to this question, and relevant for clinicians, is the effect of (increasing) force and intra-thoracic pressures on this reservoir function.⁷⁸

---

¹ ERC Scientific Meeting, Cologne (D). October 2009 Oral presentation
Using the model, with standard conditions of a mean circulatory pressure of 15 mmHg, a compression frequency of 100 cpm, and a C:R ratio of 1:1, at pe(t) varying from 50 – 125 mmHg, the central veins close rapidly and early during compression systole. Volume changes caused by extra vascularization was not modeled.\(^6\) The purpose of this experimental series was to follow volume distribution over time.\(^9\) To our knowledge this has not been done in human or animal experimentation.

Under all conditions, particularly in the range of pe(t) representative for effective compressions, the normalized cross sectional area (i.e. the area at zero transmural pressure) of both the vena cavae as rapidly decreased to 0.35 (a factor 4, VCI), and to 0.5 (a factor 3.2, VCS) while the pulmonary veins required almost 325 seconds to decrease to this size, finally reaching a factor 6 reduction. This contrast, demonstrating high instantaneous, but brief, flows impacts on the reservoir function of the intrathoracic veins. The vessels open completely only during interruptions in compressions such as during ventilatory pauses. However, forward movement is, in part, undermined by extensive sloshing. Blood volume continues to redistribute to the kidney and mesentery circulation, pooling on the venous sides, as return to the thoracic cavity is hindered.

These data offer a handhold to the persisting interest and potential in the use of the abdominal compartment during chest compressions. The static compression, initiated by Redding et al.,\(^7\) and interposed abdominal compression, described by Ralston et al.,\(^8\) respectively refer to splinting of the diaphragm, to offer ‘protection’ or perhaps decrease loss of force from intra-thoracic pressure waves, focal increase in aortic peripheral resistance, and decreased venous pooling with improved (more rapid) flow towards the chest during CPR diastole. However, due to subtle limitations in modeling, these data, which suggest that the location and gradient of abdominal compression is relevant, were not cleared up.\(^7\)

Clinically, with strong voices suggesting that increasing continuous compressions at rates of 90-100 cpm, this handhold on unwelcome but physiological corrections with the complex of vascular reactions to CPR may need to be addressed. The numeric loss of CPP during brief interruptions to chest compressions may be compensated over time by the progressive trapping of volume outside the thoracic cavity. Whether these effects are important factors in the time sensitive real-life situation in humans (i.e. decreased survival over time despite good CPR) remains to be clarified and will be complex.

9.5 Summary

This Chapter, bringing the theoretical to the more clinical reality in CPR, uses three subtle, interrelated, aspects in chest compressions to illustrate the point that mechanistic thinking in CPR may offer solutions for clinicians.

Optimal coronary perfusion (Qco), perhaps one of the two principal endpoints, during chest compressions, seem to be hindered by the activity undertaken to produce it; the technique for generating stroke volume, may be time and patient sensitive; and volume loss, both undermining pre-load and as an resultant of volume sequestration, may need to be addressed before the length of uninterrupted chest compressions change. The advantage of fundamental modeling and the need to see the modeling incorporated in clinical strategy, seems clear.\(^8\)

---

While these aspects are used as indicators, more than as items in formal evidence, they hold enticing prospects for future, in depth, analysis.

9.6 Acknowledgements

Special thanks are due to ms. Y. Koeken, M.Sc. and I. Paulussen M.Sc. for their contributions to this Chapter and their insights.
9.7 References

40 Stephenson HE jr, Reid LC, Heyton JS. Pitfalls, precautions and complications in cardiac resuscitation; study of 1,200 cases. AMA Arch Surg. 1954; 69: 37-53.

Chapter 9: Things (not) in evidence


Chapter 9: Things (not) in evidence

Physical modeling in CPR: simple and complex


Physical modeling in CPR: simple and complex


10.1 Introduction

The physical model most commonly used in cardiopulmonary resuscitation (CPR) is the practice
manikin. A model, or perhaps more correctly a simulator, in the classic sense of the word: a
teaching tool (chapter 5). As with mathematical models, the use of this tool, its complexity and its
purpose have varied over time, but its role has only recently been challenged.

This role for the resuscitation manikin is unique. Inherent to the practice of CPR, the manikin has
achieved the status of surrogate for actual performance, and has been widely accepted in that role
by professionals and lay people alike.

This chapter will describe this model, its history and its relevance to cardiopulmonary
resuscitation today. The physical aspects and their use in an educational system will be
highlighted.

10.2 The physical model

The introduction of closed chest cardiac resuscitation, in 1960, also was the introduction of one of
the most consistent models or simulators in (para)-medical training. 1 Kouwenhoven
et al., set out
to involve the general public in a life saving system of care, and advocated that the steps needed
be practiced so that skills would be adequate.

While not actively supporting the introduction to the general public, the first Guidelines strongly
supported the use of ‘life-like’ manikins and required their development and use during courses
as early as the American Heart Association (AHA) Statement of 1966.2 This led to the
development of the Resusci Anne. 3 By the time the 1974 Guidelines were published, the
resuscitation committee of the AHA recognized the need to establish standards for the minimum
requirements in both training for trainees, and for instructors, as well as for course contents
including directions for training aids and materials. 4 The first (minimum) requirements for a
manikin were listed (Table 1), as well as an indication of the time needed to observe, transfer,
correct and integrate skills (at least four hours).

The importance of actual practice was highlighted, allowing for a learning curve and for feedback
to be given, under safe conditions, without a patient actually being involved. Careful and
repetitive practice, both with and later without feedback allows the student to integrate skills and
correct errors. Nonetheless, as will be elaborated on later (chapter 10.5), concerns about the
model remained.

Table 1: 1974 requirements for manikins3

<table>
<thead>
<tr>
<th>Requirement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Airway Compressions</td>
</tr>
<tr>
<td>Airway obstruction when the neck is flexed</td>
</tr>
<tr>
<td>Adequate resistance to chest compressions</td>
</tr>
<tr>
<td>Effective movement of the chest when adequate ventilation is performed</td>
</tr>
<tr>
<td>Effective movement of the sternum when compressions are properly applied</td>
</tr>
<tr>
<td>Objective feedback in the form of lights, gauges, strip chart is desirable</td>
</tr>
<tr>
<td>Palpable carotid pulse is desirable</td>
</tr>
</tbody>
</table>

Physically, different manikins have been produced over the years. The Laerdal line of manikins
historically has been the golden standard, with Simons (1986), 5 following the Emergency Care

...
10.1 Introduction

The physical model most commonly used in cardiopulmonary resuscitation (CPR) is the practice manikin. A model, or perhaps more correctly a simulator, in the classic sense of the word: a teaching tool (chapter 5). As with mathematical models, the use of this tool, its complexity and its purpose have varied over time, but its role has only recently been challenged.

This role for the resuscitation manikin is unique. Inherent to the practice of CPR, the manikin has achieved the status of surrogate for actual performance, and has been widely accepted in that role by professionals and lay people alike.

This chapter will describe this model, its history and its relevance to cardiopulmonary resuscitation today. The physical aspects and their use in an educational system will be highlighted.

10.2 The physical model

The introduction of closed chest cardiac resuscitation, in 1960, also was the introduction of one of the most consistent models or simulators in (para)-medical training. 1 Kouwenhoven et al., set out to involve the general public in a life saving system of care, and advocated that the steps needed be practiced so that skills would be adequate.

While not actively supporting the introduction to the general public, the first Guidelines strongly supported the use of ‘life-like’ manikins and required their development and use during courses as early as the American Heart Association (AHA) Statement of 1966.2 This led to the development of the Resusci Anne.3 By the time the 1974 Guidelines were published, the resuscitation committee of the AHA recognized the need to establish standards for the minimum requirements in both training for trainees, and for instructors, as well as for course contents including directions for training aids and materials.4 The first (minimum) requirements for a manikin were listed (Table 1), as well as an indication of the time needed to observe, transfer, correct and integrate skills (at least four hours).

The importance of actual practice was highlighted, allowing for a learning curve and for feedback to be given, under safe conditions, without a patient actually being involved. Careful and repetitive practice, both with and later without feedback allows the student to integrate skills and correct errors. Nonetheless, as will be elaborated on later (chapter 10.5), concerns about the model remained.

Table 1: 1974 requirements for manikins3

<table>
<thead>
<tr>
<th>Airway</th>
<th>Compressions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Airway obstruction when the neck is flexed</td>
<td>Adequate resistance to chest compressions</td>
</tr>
<tr>
<td>Effective movement of the chest when adequate</td>
<td>Effective movement of the sternum when compressions</td>
</tr>
<tr>
<td>ventilation is performed</td>
<td>are properly applied</td>
</tr>
<tr>
<td></td>
<td><strong>Objective feedback in the form of lights, gauges, strip chart is desirable</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Pulspable carotid pulse is desirable</strong></td>
</tr>
</tbody>
</table>

Physically, different manikins have been produced over the years. The Laerdal line of manikins historically has been the golden standard, with Simons (1986),5 following the Emergency Care
Research Institute's (1981) description as well as expressing concerns on the efficacy of manikins in popular use. Only (much) later were actual investigations into correlations between manikins and humans’ published.

In chapter 2.3 the basic course in CPR was described, including the psychomotor skills required to perform. The manikin, and more particularly the Laerdal Resusci Anne™, was briefly touched on, and the difficulty involved in teaching mentioned. Not addressed there was the actual interaction between the model (i.e., an actual manikin or even a pillow) and the trainee either in general, or as a function of the model itself. These aspects are summarized in the next sections as two, independently published, brief papers.

10.3 Are there requirements for manikins and how does they impact skills?

The use of “sophisticated” manikins for cardiopulmonary resuscitation (CPR) training has been advocated. These manikins provide information in terms of modalities such as ventilation volume, compression depth, frequency, etc., and have been shown to assist in evaluating skills. However, immediately after a CPR course, psychomotor skills are often poor. As early as 1986, Seaman surmised that the time spent on “hands-on” training is a factor in skills quality. This conclusion is supported by Moser et al., which showed only a mean of 2.4 minutes (or 6% of practice time) to be available for the “hands-on” part of the one-rescuer CPR training. They suggested that more trainee satisfaction and possibly an improvement in skills could be achieved by having trainees spend significantly more time utilizing manikins.

Sophisticated manikins are costly to purchase, use, maintain, and transport. These features led to one manikin being shared by trainees in a ratio of 6-7:1 trainees-manikin. Simple, individual manikins were developed to address these issues. They are uncomplicated, compact, light and inexpensive, and can be used in a 1:1 trainee-manikin ratio during training. The Actar 911™ (Actar Airforce Inc., NJ, USA / MediScore, Bodegraven, NL), and the Laerdal Little Anne™ (Laerdal Corp., Stavanger, NO) are examples.

This study addressed the fear that trainees may “learn incorrect skills” when practicing without the feedback offered by a sophisticated manikin. It presents a direct comparison of one-rescuer skills levels following initial training using both sophisticated and individual manikins. Relevant didactic and logistic aspects are also discussed.

10.3.1 Materials and methods

The study was performed during the first session of 30 hours of training in Emergency Medicine. The 8.5 hours of basic (BLS) and advanced cardiac life support (ACLS) training were based on the American Medical Association’s 1992 guidelines and was given to about 24 first year medical students each time. (Students with acceptable experience were exempted from the course). These students were divided into three cohorts. Two study cohorts used “Actar 911™ (Act) or the Laerdal “Little Anne™ (LA) in a 1:1 trainee-manikin ratio. These light weight manikins have mechanical airway-blocking systems (insufficient extension of the neck blocks the airway) and disposable lungs. The head and thorax can be replaced. When the correct compression depth is reached the LA gives an audible ‘click’; the Act features a maximum compression depth indicator. The LA purchase price is twice that of an Act, a Recording Resusci Anne (RRA) about 28 times. The third (control) cohort used the (sophisticated) Recording Resusci Anne (RRA, type 20.10.00, Asmund Laerdal, Stavanger, NO) in a ratio of 4-5:1 trainees-manikin. Modalities with direct (visual or auditory) feedback in the RRA manikin are compression frequency (metronome),

<table>
<thead>
<tr>
<th>Scoring modality</th>
<th>criteria</th>
<th>error points</th>
</tr>
</thead>
<tbody>
<tr>
<td>frequency (cpm)</td>
<td>&lt;= 21</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>21 - 25</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>25 - 30</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td>30 - 40</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td>40 - 50</td>
<td>25</td>
</tr>
<tr>
<td></td>
<td>50 - 60</td>
<td>30</td>
</tr>
<tr>
<td></td>
<td>60 - 70</td>
<td>35</td>
</tr>
<tr>
<td></td>
<td>70 - 80</td>
<td>40</td>
</tr>
<tr>
<td></td>
<td>80 - 90</td>
<td>45</td>
</tr>
<tr>
<td></td>
<td>90 - 100</td>
<td>50</td>
</tr>
<tr>
<td></td>
<td>100 - 120</td>
<td>55</td>
</tr>
<tr>
<td></td>
<td>120 - 140</td>
<td>60</td>
</tr>
<tr>
<td></td>
<td>140 - 160</td>
<td>65</td>
</tr>
<tr>
<td></td>
<td>160 - 180</td>
<td>70</td>
</tr>
<tr>
<td></td>
<td>180 - 200</td>
<td>75</td>
</tr>
<tr>
<td></td>
<td>200 - 220</td>
<td>80</td>
</tr>
<tr>
<td></td>
<td>220 - 240</td>
<td>85</td>
</tr>
<tr>
<td></td>
<td>240 - 260</td>
<td>90</td>
</tr>
<tr>
<td></td>
<td>260 - 280</td>
<td>95</td>
</tr>
<tr>
<td></td>
<td>280 - 300</td>
<td>100</td>
</tr>
</tbody>
</table>

This scoring system assigns penalty points to each modality, weighing them as to severity. The correct technique is given “zero” points. Summation of the six modalities leads to a total score; up to and including 15 points is considered satisfactory performance. A severe limitation of a training model (i.e., margin of measurement error) is directly associated with a negative outcome.
compression depth (light display and mechanical resistance) and ventilation volume (light display). Cohort assignment was made by drawing a random number at course registration.

Following presentation of theory, trainees in the Act and LA cohorts used their own manikin to practice. They also used a Recording Resusci Anne (RRA) once during the training period. This use, limited to three minutes, served to familiarize themselves with this manikin as well as to produce a paper "printout" to evaluate their progress. The students in the control (RRA) cohort practiced by taking turns on the RRA manikin. Each cohort had a student assistant instructor, who had completed 60 hours of instructor training, with one physician supervising the course. Following 1.5 hours of training, each trainee made a two- to three-minute printout of one-rescuer CPR using an RRA manikin with the lights and metronome rendered inoperable. The printouts were scored by one experienced supervisor blinded to the cohort/number combinations.

The Recording Resusci Anne printout is a real time record which has been demonstrated to give a reliable impression of lung insufflation and external heart massage technique, within the limitations of a training model (i.e., margin of measurement error = 10-15%). (Figure 1) The scoring system developed by Berden (1993) was used to objectify skills. It uses the printout to describe independent modalities within one-rescuer CPR (Table 2). These modalities are compression frequency and depth, the compression-relaxation ratio, ventilation volume and interval (Figure 1).

A sixth modality, hand position, is scored by instructor observation. Each modality is scored independently. The penalty (error) points are weighted with respect to severity and lead to a cumulative score. A score of ≤ 15 penalty points is considered satisfactory performance. A severe error (10 or more penalty points for that modality) is considered by the Berden system to be directly associated with a negative outcome.

Table 2: Berden Scoring table (2000 ICOR criteria). This scoring system assigns penalty points to each of five independent modalities within one-rescuer CPR using the printout of the Laerdal Recording Resusci Anne. The sixth modality, hand position, is judged by an observer. The number of penalty points assigned to each modality is weighted as to severity. The correct technique is assigned "zero" points. Summation of the six modalities leads to a total score; up to and including 15 error points is considered an adequate resuscitation attempt. Ventilation interval is the time between the last compression of a 15:2 cycle and the first compression of the next cycle.

<table>
<thead>
<tr>
<th>Scoring modality</th>
<th>criteria</th>
<th>error points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Compression frequency (cpm)</td>
<td>≤ 39</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td>40 - 59</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td>60 - 79</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>80 - 100</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>101 - 120</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>121 - 140</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>&gt; 140</td>
<td>15</td>
</tr>
<tr>
<td>Compression depth (mm)</td>
<td>≤ 21</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td>22 - 29</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>30 - 37</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>38 - 52</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>53 - 60</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>&gt; 60</td>
<td>10</td>
</tr>
<tr>
<td>Compression relaxation ratio</td>
<td>≤ 0.6</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>0.6 - 1.4</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>&gt; 1.4</td>
<td>10</td>
</tr>
</tbody>
</table>
10.3.2 Results

Of the 165 students in the investigation, data could be analyzed for 161 (97.5%). Of those lost to evaluation, three printouts were not clearly labeled and one trainee failed to complete the study. There was no difference among the three cohorts with respect to age or gender. All were healthy first year medical students. None had prior training in CPR.

Overall, 55% of the students ‘passed’, (≤15 penalty points), with no significant difference among the three cohorts (Chi², p = 0.18). The distribution of actual skills and penalty points (p = 0.94) are shown in Table 3 and Figure 2 respectively. The number of errors and severe errors (i.e., those receiving 10 or more error points) made per trainee did not change significantly between the cohorts (p = 0.92 and 0.66, respectively). On average 2.2 errors were made.

Scores were entered in SPSS version 6.0. The numeric results are reported as mean ± SD; penalty point analysis is reported as median unless otherwise noted. Statistical analysis was performed using ANOVA, and multivariable analysis between groups. Penalty point analysis was performed using Kruskal-Wallis; p< 0.05 was considered to represent a significant difference.

Figure 1: The printout of a Recording Resusci Anne.
The printout gives a real time impression of the one-rescuer CPR attempt. From top to bottom, ventilation volume, resting and chest impression depth, and a time (seconds) track is shown.
Table 3: Table of results (Means +/- SD). Composition and results achieved by each cohort defined by the scoring modalities. A "p" is shown for each modality. Statistical significance was taken to be p<0.05. Note that the modality hand position is not presented: effectively 100% of trainees were judged as having "correct" hand positions. An "*" = severely insufficient depth and volume.

<table>
<thead>
<tr>
<th>parameters / modalities</th>
<th>Significance</th>
<th>cohort Actar 911 n=57 (ACT)</th>
<th>cohort Little Anne n=63 (LA)</th>
<th>cohort Recording Resusci Anne n=41 (control, RRA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>compression freq. (cpm)</td>
<td>NS</td>
<td>98.2 ± 14.4 95% CI = 94.3-101.9</td>
<td>100.5 ± 15.7 95% CI = 96.6-104.5</td>
<td>105.4 ± 16.8 95% CI = 110.1-110.7</td>
</tr>
<tr>
<td>compression depth (mm)</td>
<td>P = 0.04*</td>
<td>43.7 ± 4.7 95% CI = 42.4-44.9</td>
<td>42.0±6.6 95% CI = 40.9-44.3</td>
<td>48.4±5.5* 95% CI = 46.6-50.1</td>
</tr>
<tr>
<td>Compression-relaxation ratio</td>
<td>NS</td>
<td>0.73 ± 0.24 95% CI = 0.6-0.8</td>
<td>0.70 ± 0.31 95% CI = 0.7-0.9</td>
<td>0.78 ± 0.34 95% CI = 0.6-0.9</td>
</tr>
<tr>
<td>Ventilation volume (li)</td>
<td>NS</td>
<td>1.1 ± 0.3 95% CI = 1.0-1.2</td>
<td>1.2 ± 0.4 95% CI = 1.1-1.3</td>
<td>1.1±0.4 95% CI = 1.0-1.2</td>
</tr>
<tr>
<td>Ventilation interval (sec)</td>
<td>NS</td>
<td>6.0±1.4 95% CI = 5.7-6.4</td>
<td>6.3±1.7 95% CI = 5.9-6.7</td>
<td>5.9±1.3 95% CI = 5.5-6.3</td>
</tr>
<tr>
<td>Trainees with:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-penalty points (mean)</td>
<td>NS</td>
<td>15.4</td>
<td>16.9</td>
<td>17.2</td>
</tr>
<tr>
<td>-≥ 1 errors</td>
<td>NS</td>
<td>55 (96%)</td>
<td>62 (98%)</td>
<td>49 (95%)</td>
</tr>
<tr>
<td>-severe errors (&gt;10 PP)</td>
<td>P = 0.04*</td>
<td>38 (67%)</td>
<td>46 (73%)</td>
<td>31 (76%)</td>
</tr>
<tr>
<td>-inadequate CPR*</td>
<td>NS</td>
<td>5 (9%) compdepth</td>
<td>14 (16%) compdepth</td>
<td>3 (8%) compdepth</td>
</tr>
<tr>
<td></td>
<td>NS</td>
<td>4 (8%) ventol</td>
<td>7 (12%) ventol</td>
<td>4 (10%) ventol</td>
</tr>
<tr>
<td>Num. of trainees passing (≤15 PP)</td>
<td>NS</td>
<td>30 (53%)</td>
<td>35 (56%)</td>
<td>24 (59%)</td>
</tr>
</tbody>
</table>

Figure 2: Total number of penalty points acquired.
The performance of each trainee, by cohort, is shown by the acquired number of error points. 55% of students had 15 or less penalty points, which is considered indicative for a good CPR technique (p = 0.18 between scores).

Modalities with direct (visual or auditory) feedback in the RRA manikin were examined further (Table 2). The distribution of compression frequency was similar in all cohorts (p = 0.69), with a mean of 108 cpm. In the compression depth modality the Act cohort showed 89% of trainees in the 38-51 mm range in comparison to 94% and 95% for the LA and the RRA cohorts (p = 0.88). Actual measured compression depth varied significantly (p = 0.04) but this was not reflected in the penalty points. When errors were made, overly deep compressions (>53 mm) were seen three times more often than shallow (30-37 mm) compressions. 47% ventilated correctly (p = 0.53). 30% of trainees insufflated 1.51-2.0 L (a severe error).
In the modality ventilation interval, only 16% (LA), 18% (Act) and 22% (RRA) avoided errors \( (p = 0.35) \), in contrast with the modality compression depth 85% (RRA), 86% (LA), and 88% (Act) in which the trainees performed optimally \( (p = 0.93) \).

The Act and LA torso manikins did not move more during training than the RRA. Although the torso-model manikins bear little resemblance to a patient, neither trainees nor instructors found this a limitation to training or a (psychological) shortcoming. Instructors confirmed that trainees using the individual manikins had much more hands-on training than those in the RRA cohort. The Act and LA manikins seemed cost-effective: use of the RRA paper printout roles decreased by a factor of three during the study.

### 10.3.3 Discussion

Our study showed similar BLS-CPR one-rescuer skills following initial training when comparing the Actar 911, the Little Anne and the Recording Resusci Anne. There was no difference in overall quality of modalities such as compression frequency, compression depth and ventilation volume, although the trainees in the RRA cohort should have corrected mistakes immediately. One significant difference in the compression depth measurements \( (p = 0.04) \), was not reflected in the penalty points assigned. These findings support the notion that without “learning” mistakes, trainees can learn CPR skills without extensive use of a sophisticated manikin.

Little has been documented about the need for sophisticated manikins, with studies emphasizing instructor feedback and its effect.\(^7\) The use of sophisticated manikins has also, due to logistical reasons such as expense, storage and transportation limitations as well as manikin wear and tear, created a situation in which trainee-manikin ratios of 3-4:1 has been accepted by the AHA, and ratios of up to 12:1 are not unknown.\(^2\) In effect, it seems that sophisticated material has led to an important decrease in practice of CPR psychomotor skills.\(^1\) Skills evaluation shows that few can perform CPR even under optimal conditions.\(^1\) Looking at our data, 95% of trainees made errors during the skills evaluation, with about 63% making two or more.

Concern seems legitimate. Starting BLS has a positive effect on patient outcome: in our opinion, this presumes that BLS skills are at least adequate and that we can motivate trainees by offering adequate time to train. While we know of no systematic studies, all too regularly (anecdotal) accounts of poor bystander BLS and outcome are noted. Since many enroll in CPR courses to aid relatives, intensive training might be expected to increase confidence and improve psychological preparation in addition to increasing skill.

Different authors have shown that while 12 minutes (range 2.5-16 minutes) were available for practice per trainee, (3-4:1 ratio), poor performers were judged not to have had enough time to make corrections.\(^1\) The suggestion that trainees should be able to integrate skills and spend time practicing is logistically difficult with only one or two manikins.\(^1\) The use of individual manikins allows trainees to work at their own tempo, integrating, bringing up to speed, and then merging skills, without pressure to ‘perform’ under the watchful eyes of peers. This also frees instructors to circulate.\(^1\) In line with this, Tweed et al.\(^1\) demonstrated that ‘overtraining’ of motivated and mature trainees resulted in a significantly better skill level. This term of ‘overtraining’ is, of course, a contradiction.\(^2\)

A puzzle remains in our findings: while we set out to investigate if trainees using individual manikins would be limited by the nature of the manikin (i.e., ‘learn errors’) post factum, we wonder why the trainees in the Act and LA cohorts did not score better.\(^1\) This remains incompletely addressed. A possible explanation is that the trainees using the individual manikins were subjected to a course protocol set up for a 4-5:1 trainee-manikin ratio, thereby limiting their learning curve. Improved layout with structured exercises (i.e., step-by-step "hands-on"
instructions), overhead sheets allowing improved self-instruction, adapted instructor training are tools as yet unused in combination with the individual manikins. During the study, individual manikins were used in conjunction with the RRA. Our reason for this was twofold: first, to retain the RRA printout as a teaching tool, allowing trainees to see what they did and to discuss possible improvements in technique, and second, to use the RRA as a generally accepted evaluation tool. This may have had a (negative) influence on skills, but the limitations set on this use were intended to standardize any influence over the two study cohorts. For didactic reasons, we intend to continue combining sophisticated manikins with the individual ones (optimal use of both) during training.15 Both the Act and the LA seem to be compatible with the RRA. Baubin et al.22, while investigating manikin compression characteristics, reported that the physical buildups were not uniform, varying in thoracic diameter, compression depth/pressure relationship and effect of use of resistance (the chest plate of the RRA stays at a somewhat lower position after extensive use). They suggested the use of multiple manikins as most realistic.

Further study, in closely controlled layperson CPR training, is needed to confirm our preliminary results and impressions. Skills retention may also be improved by further evaluation of didactic techniques involving manikins used in CPR training courses.

10.3.4 Conclusions
Although little is known about the need for expensive, sophisticated manikins in relation to the quality of skills to be learned, their use is generally accepted, but appears to limit practice time. We investigated whether two types of individual, simple manikins (the "Actar 911"™ and the Laerdal "Little Anne”™) can be used effectively during one-rescuer CPR skills training of medical students in a 1:1 trainee-manikin ratio without trainees “learning errors”. No major significant differences in skills, as measured by the number of errors made and penalty points assigned, were found between the two study cohorts and the control cohort using Recording Resusci Anne manikins in a ratio of 4-5:1 trainees-manikin. Adapting didactics with plenary training and overheads may allow students, and other laypersons, to use individual manikins optimally in conjunction with a sophisticated manikin. Further controlled study is needed to confirm these preliminary results.

10.4 Integrated resuscitation simulators should retain “basic” options.
In this section (a letter to the editor) we ask attention for a training and clinical concern. We have observed that manikins available for teaching advanced life support (ALS) may actually limit adherence to the Guidelines 2005.23 These Guidelines state that chest compressions must be an enduring facility, and manikins are available to teach these skills in a variety of course settings.24 The Guidelines further emphasize the significance of team intervention: timely intervention to avoid full arrest as well as excellent basic life support continuing in the face of ALS techniques.25 However, in furtherance of the paper by Perkins,24 we would put forward that two issues may be caused by current manikins. These are the issues of full scenario training for early intervention teams and objectifying basic skills when using integrative manikins.

Our institution, a non-university teaching hospital with 650 beds, dispatches a medical emergency team (QRT: Quick Response Team) employing the MegaCode system first described by Kaye et al.8 This team is called in for all threats to vital functions within the hospital, including, but not limited to, actual circulatory or respiratory arrest. Our findings parallel the Guidelines 2005 in that the implementation of a QRT leads to a reduction in full resuscitations (i.e., those requiring chest compressions) of 60 to 75%: in our case, such incidents have been reduced by 67% over the last 5 years. This means that two out of three calls are initially not for full resuscitations.
From a clinical as well as training point of view, this means that the QRTs should be trained in a wider range of cases than just full resuscitations on integrated clinical simulators. We have noted that cases which are not directly algorithm driven accrue more adverse events than those driven by algorithms. As a natural consequence, strict training in CPR should be retained as well as complemented with training in scenarios where critical events such as when BLS becomes ALS or the patient reaches ROSC.

At the moment there are different types of manikins available, as recently described.24 These are BLS, ALS and “intermediate” task-and-team oriented manikins. Their characteristics, which may have an impact on training, have been described.5,6 None of these manikins are able to represent a patient in poor condition sliding towards CPR, and perhaps into arrest, while recording the quality of each of the central skills required and care given. If the focus is chest compressions one manikin is needed. As the focus moves towards specific ALS skills, another manikin is needed, and our most direct concern, chest compression simulation becomes completely unrealistic and/or un-scored. This makes training very much instructor driven and assessment needlessly subjective.26,27

Scenario training recognizes that individual skills are, just as the chain of survival, being incorporated into one whole, functional, unit.28 In our view, it is vital to be able to train a team in the whole range of care relevant to their situation. Force needed for adequate compression depth in ALS manikins is not realistically related to the size of the manikin, and initial (mouth-to-mouth or mouth-to-face mask) ventilations are discouraged as the moisture may damage airway mechanisms. BLS and intermediate training manikins lack the possibility of airway maneuvers such as Mayo tubes or intubation. In order to show the team how well they are performing, it is vital that all the skills be scored during the scenario, including the important basic life support components.

We would suggest that, with the Guidelines 2005, a new era has also arrived for simulations. Even without exorbitant expenditures, particularly in ALS, we must be able to deal with the full scope of cases including (all) the important skills involved. Therefore the ideal manikin, useful in general hospitals and other facilities where teams provide coordinated BLS and ALS care, would have to be able to start of as a critically ill patient, perhaps still talking, breathing and with a pulse, worsening to a patient in need of full resuscitation. All interventions should be objectified for the trainer, so that debriefing is focused and positive. It is hard to accept that none of the new generation of integrative, computer supported, ALS manikins are capable of supporting the concept of early intervention scenarios through to ALS with retention of all BLS scoring abilities. Changing the manikin during the scenario is strongly distracting and detracts from the essence of the guidelines 2005. We strongly suggest that the philosophy of the design of manikins be adjusted to create room to follow the guidelines completely.

10.5 Is the model needed for skills?

Having presented two viewpoints on specific aspects in physical models, we have not addressed the question of what contribution the models have actually made: instituted to train lay- and professional caregivers, have they achieved this goal?

Manikins have been an integral, part of resuscitation training for decades, starting in the 1960s. Many partial task simulators have been developed, such as devices to learn the correct application of cricoid pressure.29 Increasingly voices have been raised to suggest alternatives to classic, group-oriented, centrally led courses.30 A central aspect in this is that students do not seem to
learn the skills during classic training, as well as not being able to appraise their own skills correctly (see also chapter 11).31,32 Scores for the quality of chest compressions remain low, with scores at completion of 2%,33 3.4%,34 12%20 correct compression rates, even in closely monitored teaching environments.35,36 This has been the source of terms such as ‘overtraining’ which led to a 97% adequate compression score.19 This concern is not new,36-39 but certainly persistent.37 The reoccurring thread during lay- and professional training seems to be the further introduction of feedback- and instructor quality may be needed in getting skills up to a mastery level.38,39 since achieving this level also impacts retention of the acquired skills.40 Early computer-based interactive forms demonstrated rapid knowledge improvement, but lack of skills acquisition,41 while other authors have demonstrated that knowledge is a prerequisite for (retention of) skills, but do not guarantee them (39% good compressions).52,53 There has also been a propensity to use complex, integrated, manikin systems (i.e., SimMan) where part task trainers would be more appropriate.43 However, Laerdal has introduced the VREM (virtual reality enhanced manikin) as an interesting combination of part task and integrated systems.44

Recently, Perkins described the position of simulation in more general terms, using learning theory as an approach to skills and knowledge acquisition while applying this to resuscitation training.45 There is good evidence that the ‘core business’ of CPR is being understated and perhaps even somewhat neglected. Laypeople not sufficiently practiced to be optimally effective (just doing something is better than nothing?)46, and professionals being of the opinion that chest compressions is a simple skill and therefore unworthy of serious practice since they have staff to perform it, while they think and cause interruptions (perhaps the reason that the guidelines 2005 de-emphasis the role of physicians in resuscitation?).

Despite voices to the contrary, the need for and the usefulness of a model - a manikin - with good, objective feedback, acting as closely to reality as possible in conjunction with motivated and skilled instructors cannot be sufficiently reiterated.47 With the further development of new manikins, and other technical devices giving feedback or even instructions, the intent of model making, even in the strict sense used in the pervious chapters, can still be realized.48,49

### 10.6 The manikin and the model

The previous two sections approach modeling from a physical simulator format. The use of a training manikin to develop and maintain skills has been, without doubt, a mainstay in resuscitation medicine since the 1960s. New technology has even suggested that instructor interaction (during refresher exposure of ca. five minutes) can be done without.50 Despite this, getting the improvement into the real world of CPR, both in- or out-of-hospital, has been proven to remain a challenge.51 There seems to be no issue that the general public wants to learn these skills.52,53 And there seems to be little issue with the relationship between good quality CPR and better outcome.54,55 The logical consequence seems to be that less procedure instruction (as suggested in the 2005 guidelines) and more aggressive, skills orientation (i.e., accepting 90-95% of ‘in range’ compressions as adequate instead of a mean depth between 4-5 cm) during a representative time frame of circa five minutes of chest compressions seems proper use of a suitable manikin model.51 Teaching can only be done on a model (the manikin) which generates feedback, or if a separate (stand-alone or incorporated in a defibrillator-monitor system) feedback device is used in addition to a non-feedback manikin- to ensure that skills are being taught correctly.56 The feedback is a supporting tool, and testing should be done without this aid. Practice should continue in cycles until a level of mastery is achieved, and both evaluation of

---

individual skill components, as well as the process supporting the skills must be adequate and without a ‘knockout’ across the board.57

10.7 Conclusions

Despite the limited chance that laypersons will be confronted with cardiac arrest, interest and motivation is not lacking. By extension, this seems to be the inverse for professionals. A wide range of resuscitation manikins offer controlled conditions for skills acquisition and mastery if the boundary conditions are carefully defined and vigilantly monitored.58 Where public telephone booths have disappeared from street corners, perhaps a mini-manikin station (VAM) should be placed, enticing and stimulating the layperson to reach quality, instead of just meeting minimal requirements. Professionals need to be offered the opportunity to use manikins with basic options, not only to learn the skills, but also to objectify the performance of core skills during scenario training. Feedback must be available if professionals are involved in resuscitation, both to support and objectify quality.
10.8 References

Chapter 10: Physical modeling in CPR

58 Hoke RS, Handley AJ. A reference basic life support provider course for Europe. Resuscitation. 2006 Jun; 69(3); 413-419.
Training needs and qualifications of anesthesiologists not exposed to ACLS.

Training needs and requirements

Training needs and qualifications of anesthesiologists not exposed to ACLS.

Abstract

Our goal was to establish which needs exist for specific training in Advanced Cardiac Life Support (ACLS) in Anesthesiology residents and interns not exposed to structured ACLS courses.

48 residents, and 7 interns accepted for training in Anesthesiology, were tested in an unannounced, blind, cross sectional, prospective assessment using a recording manikin with validated scoring system, a questionnaire, and 35 multiple-choice questions.

65% said not to have had any CPR training within the last two years. Answers were correct in 55 ± 14% of the cases, increasing significantly with length of training (p = 0.001). One-rescuer CPR skills were inadequate: 13% (n = 7) of participants scored within acceptable limits when using the Berden Scoring system. No correlation with skill was noted with increased length of residency, confidence, ER or ICU experience, or participation in CPR incidents.

Anesthesiology residents and interns were not able to demonstrate BLS skills properly even while in training and while recognizing this themselves. CPR-related knowledge is poor and increases only incidentally over the years of residency even though participants were frequently exposed to seminars, resuscitation situations and interact with structured protocols daily. The use of multiple-choice questions and the Berden Scoring system avoids difficulties in evaluating case scenario-type of tests. We suggest that trainees should be motivated to take standardized, intensive, recognizable ACLS courses, emphasizing BLS skills and requiring (re)certification.
Abstract

Our goal was to establish which needs exist for specific training in Advanced Cardiac Life Support (ACLS) in Anesthesiology residents and interns not exposed to structured ACLS courses. 48 residents, and 7 interns accepted for training in Anesthesiology, were tested in an unannounced, blind, cross sectional, prospective assessment using a recording manikin with validated scoring system, a questionnaire, and 35 multiple-choice questions. 65% said not to have had any CPR training within the last two years. Answers were correct in 55 ± 14% of the cases, increasing significantly with length of training (p = 0.001). One-rescuer CPR skills were inadequate: 13% (n = 7) of participants scored within acceptable limits when using the Berden Scoring system. No correlation with skill was noted with increased length of residency, confidence, ER or ICU experience, or participation in CPR incidents. Anesthesiology residents and interns were not able to demonstrate BLS skills properly even while in training and while recognizing this themselves. CPR-related knowledge is poor and increases only incidentally over the years of residency even though participants were frequently exposed to seminars, resuscitation situations and interact with structured protocols daily. The use of multiple-choice questions and the Berden Scoring system avoids difficulties in evaluating case scenario-type of tests. We suggest that trainees should be motivated to take standardized, intensive, recognizable ACLS courses, emphasizing BLS skills and requiring (re)certification.
11.1 Introduction

Knowledge of advanced cardiac life support (ACLS), defined as basic life support (BLS) and techniques such as airway management, IV-access, drug therapy, cardiac monitoring arrhythmia management including defibrillation and post-resuscitation care, is presupposed in anesthesiology interns and residents who form the core of resuscitation teams world over. This assumption, however, is unsupported by evidence. Earlier studies attempting to analyze skills and knowledge in a broad professional population have been done primarily in English speaking countries with an ACLS-course tradition (Table 1). These studies are difficult to compare and reported strongly divergent results. On mainland Europe, standardized ACLS courses have been implemented only on a very limited scale, making an extrapolation of these results to the needs of anesthesiology residents and interns in a country without ACLS courses impossible. In our institutions, exposure to "ACLS" type training consists of an estimated two hours within the annual continuing education sessions, with manikins available continuously, in addition to eight hours of training preparatory to the second year National Board Examinations.

Table 1: Earlier investigations into physicians' CPR skills. This table demonstrates the diversity of investigational methodology as well as the divergent results obtained in the studies performed during the last 13 years. EMT-D: Emergency Medical Technician - Defibrillation qualified.

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Participants (mode)</th>
<th>Methods</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bell et al.1</td>
<td>30 Trainee anesthesiologists (unannounced?)</td>
<td>Management of VF and asystole in scenario with stand-in anesthesiologist</td>
<td>3% (1) competent in BLS; 27% in VF; 30% in ACLS for asystole</td>
</tr>
<tr>
<td>Makker et al.7</td>
<td>225 Consecutive cardiac arrests in-hospital (unannounced)</td>
<td>Actual diagnostics and therapy as registered during the incident were evaluated</td>
<td>12% errors overall; 3.6% rhythm errors and 9.8% therapy errors</td>
</tr>
<tr>
<td>Quiney et al.1</td>
<td>24 Anesthesiologists of three grades (announced)</td>
<td>Management of one of three scenario's using an ALS trainer together with a team</td>
<td>79% competent</td>
</tr>
<tr>
<td>Schneider et al.8</td>
<td>162 Out-of hospital CPR cases (prospective)</td>
<td>Using on-line tape recording time intervals to ACLS steps were analyzed and physicians vs. EMT-D compared</td>
<td>Physicians more rapid in diagnostics, but slower in therapy</td>
</tr>
<tr>
<td>David &amp; Prior-Willeard19</td>
<td>30 Senior house officers and registrars in Internal Medicine (unannounced)</td>
<td>Scenario on manikin of an in-hospital cardiac arrest, including drug treatment and defibrillation</td>
<td>3% (one candidate) judged proficient</td>
</tr>
<tr>
<td>Buss et al.11</td>
<td>88 Pediatric residents (unannounced)</td>
<td>Telephone questionnaire</td>
<td>41% expressed confidence, 45% knew correct endotracheal tube size</td>
</tr>
<tr>
<td>Schwid &amp; O’Donnell10</td>
<td>30 Anesthesiologists (10 residents, 10 attending staff, and 10 in private practice) (announced?)</td>
<td>Used Anesthesia Simulator Consultant computer program, criteria: ACLS guidelines in treatment</td>
<td>40% correct for residents, 30% for attending staff and 20% for those in private practice</td>
</tr>
<tr>
<td>Thwaites et al.11</td>
<td>24 Unspecified consultants (announced)</td>
<td>Manikin-oriented scenario scored by own system visually</td>
<td>42% scored zero correct, no 100% in pre-test</td>
</tr>
<tr>
<td>Donofrio et al.13</td>
<td>86 Staff members Department of Internal Medicine (announced)</td>
<td>Self-administered questionnaire consisting of 15 multiple-choice questions</td>
<td>75-76% correct for interns and residents, 57% for internists</td>
</tr>
<tr>
<td>Goucke &amp; Dobb10</td>
<td>80 Medical staff, 80 nursing staff (unannounced)</td>
<td>12 Multiple choice questions for</td>
<td>70.9% correct for staff, 32.5% passed practical test</td>
</tr>
</tbody>
</table>
11.1 Introduction

Earlier investigations into physicians' CPR skills. This table demonstrates the diversity of results to the needs of anesthesiology residents and interns in a country without ACLS courses. Courses have been implemented only on a very limited scale, making an extrapolation of these results to the needs of anesthesiology residents and interns who form the core of resuscitation teams the world over.

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Participants</th>
<th>(mode)</th>
<th>Methods</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kaye &amp; Mancini\textsuperscript{18} 1986</td>
<td>21 Medical residents (and 17 RN’s &amp; 21 laypersons) (unannounced?)</td>
<td>Manikin-oriented scenario for one-rescuer CPR using recording strip and AHA Heartsaver checklist</td>
<td>No physician performed all steps correctly, 57% with compression and ventilation difficulties</td>
<td></td>
</tr>
<tr>
<td>Skinner et al.\textsuperscript{12} 1985</td>
<td>29 Unspecified house officers (unannounced?)</td>
<td>10-Question multiple-choice and one-rescuer CPR on standard manikin</td>
<td>55% performed CPR correctly, correct mean 37.6%</td>
<td></td>
</tr>
<tr>
<td>Mancini &amp; Kaye\textsuperscript{17} 1984</td>
<td>33 Medical house officers (unannounced)</td>
<td>Manikin-oriented scenario scored using AHA Heartsaver checklist.</td>
<td>Showed strong deterioration one year after training</td>
<td></td>
</tr>
<tr>
<td>Gass &amp; Curry\textsuperscript{10} 1983</td>
<td>20 Unspecified physicians (and 19 RN’s) (announced?)</td>
<td>Multiple-choice test and one-rescuer CPR on a recording manikin</td>
<td>Pre-test: 75% correct answers, with 62% making error on manikin</td>
<td></td>
</tr>
</tbody>
</table>

This assessment was performed to inventory the need for ACLS training as well as to define factors that might influence skills and knowledge in a population frequently confronted with resuscitation situations. In this paper we describe theoretical and practical CPR knowledge as well as experience using a questionnaire and multiple-choice questions in conjunction with a BLS skills session scored by a standardized technique and make suggestions for suitable training.

11.2 Materials and Methods

We programmed the assessment during a one-hour, mandatory, departmental continuing education session. Advance notice was avoided by placing a nondescript title on the schedule. Each intern or resident was arbitrarily assigned a number and was subsequently assigned to one of four groups, each with a non-participating mentor. The assessment involved four successive 15-minute sessions. In one session, a questionnaire was used to inventory experience and training in CPR, the participants’ opinion of their own skills and knowledge, time lapse since skills training and demographic information.

In two sessions 35 multiple-choice questions were presented. The questions were based on the 1992 American Heart Association (AHA) "Standards and Guidelines" since standards based on the European Resuscitation Council Guidelines had only recently been implemented.\textsuperscript{1,3} We included 16 research or comprehension-oriented questions as well as 19 practical questions. Evenly divided over these categories were 10 pediatric and 12 medication-oriented questions (Appendix IV).

The remaining session involved a one-rescuer CPR assessment. Twenty-one Laerdal Recording Resusci Annes (type 20.00.10, Laerdal, Stavanger, Norway) were used. Indicator lights and metronomes had been rendered inoperable. Participants were allowed time to familiarize themselves with their manikin after which a representative written record (printout 'stroke') of about two minutes was made.\textsuperscript{6} The mentors gave no instructions on rhythm, frequency, etc. Each printout, as well as notes concerning performance and “position of hands on sternum” made by the mentor, were evaluated using the scoring system developed and described by Berden (Appendix 2). This system is based on ‘error points’ (EP), weighted as to severity that are assigned to various modalities in one-rescuer CPR. The Berden system has been validated extensively and can be used in conjunction with the "chest expansion" parameter for sufficient ventilation.\textsuperscript{7} The maximum number of error points (EP) associated with an adequate one-rescuer CPR attempt is 15. All printouts and multiple-choice lists were scored by one blinded mentor.

Data were entered into a database using SPSS\textsuperscript{\textregistered}, version 6.0. Statistical analysis was performed using SPSS\textsuperscript{\textregistered} version 7.0. A two-tailed t-test was used. Multi-varient analysis was done using
11.3 Results

Of those eligible, 55 (75%) participated in the investigation. This included 7 interns who had been accepted for training and 48 first- through fifth-year residents. Sixty three percent of the participants were male, reflecting the male-female ratio within our department. The distribution of participants was due to leave, duty- and rotation schedules. All participants completed the whole investigation.

11.3.1 Questionnaire

CPR courses had been attended by all of the participating physicians. Eight (15%) noted training within the previous 12 months while 65% stated not to have received formal CPR training in over two years. Almost three-quarters of the participants remarked that their last CPR training had occurred during medical school. Most of the participants (81%) had no idea of the Guidelines used during their earlier training. One participant claimed familiarity with the Utstein style of reporting data, 16 (29%) admitted to training on some aspects of ACLS-CPR even though no such formal courses were offered at our University. Prior to the test, the participants rated their proficiency level at 5.4 ± 1.7 on a 10-point scale.

As part of residency training, 14 (26%) noted emergency room experience, including pre-hospital medic responses, and 32 (58%) cited ICU experience including cardiac cases. The interns through third-year residents noted significantly more participations in CPR interventions (p<0.05). The "opinion of own skills and knowledge" as reported by the participants (Figure 1) showed a significant increase with length of residency (p = 0.01). In addition, male trainees rated themselves significantly better in skills and knowledge than did female trainees (p=0.02). Third-year residents, who had just passed National Board Examinations, considered their CPR knowledge "outdated" significantly more often than other residents (p<0.05).

Figure 1: Questionnaire results with regard to experience and participants own opinion. The number of participants who consider their BLS skills acceptable shows a significant decrease for second and third year residents. Surprising is the almost linear increase in the percent of participants who state they have had some form of ACLS training. The "opinion of CPR skills and knowledge" remains steady through the third year, but increases (significantly) in the fourth and fifth year of residency. The actual number of CPRs also differ significantly, but inversely, decreasing between the interns through third year residents and the last two year residents.
11.3.2 Multiple-choice assessment

The multiple-choice (MC) answers were correct in 55 ± 14% of the cases, with the mean for "medication" answers being 39 ± 22% (Table 2A). A significant improvement in overall correct responses was correlated with length of training (r1 = 0.6, p<0.001). This was attributed to improvement in the category "theory", less so to "medication". No improvement was seen in the category "pediatric". While there was a significant difference in the scores in the "practical" category, it was the interns who scored significantly better (p<0.05). A strong, inverse correlation between the scores for "practical" and "theory" answers (r = -0.99, p = 0.001) was apparent at all trainee levels. Participants with "pre-hospital CPR" experience (n = 24) demonstrated significantly better MC scores than other participants (63 ± 15 vs. 49 ± 15, p<0.05). Similar comparisons for ER (n = 14) or ICU (n = 32) experience, as well as for "some form of ACLS training" (n = 16) elicited no significant differences.

Table 2A: Results (± standard deviation) of the multiple-choice questions by category and year. Mean score of 55% correct for all answers decreased to a mean of 39% for questions concerning medication. * indicates a significant differences (p< 0.05) between the labeled groups, ** with p<0.01. Number of participants passing, as represented by 60% of answers correct, is given by year.

<table>
<thead>
<tr>
<th>Category</th>
<th>1st year</th>
<th>2nd year</th>
<th>3rd year</th>
<th>4th year</th>
<th>5th year</th>
</tr>
</thead>
<tbody>
<tr>
<td>All questions (%)</td>
<td>50 ± 9 (17%) passed</td>
<td>51 ± 6 (0%) passed</td>
<td>54 ± 13 (0%) passed</td>
<td>60 ± 7 (2%) passed</td>
<td>66 ± 17 (3%) passed</td>
</tr>
<tr>
<td>Theory (n=16)</td>
<td>30 ± 7</td>
<td>44 ± 15</td>
<td>44 ± 8</td>
<td>49 ± 15**</td>
<td></td>
</tr>
<tr>
<td>Practical (n=19)</td>
<td>62 ± 10</td>
<td>56 ± 15</td>
<td>56 ± 8*</td>
<td>51 ± 15*</td>
<td></td>
</tr>
<tr>
<td>Pediatric (n=10)</td>
<td>42 ± 17</td>
<td>47 ± 17</td>
<td>49 ± 17</td>
<td>51 ± 19</td>
<td></td>
</tr>
<tr>
<td>Medication (n=11)</td>
<td>28 ± 16**</td>
<td>40 ± 19</td>
<td>49 ± 14*</td>
<td>58 ± 19**</td>
<td></td>
</tr>
</tbody>
</table>

11.3.3 Practical skills assessment

The participants assembled a median of 35 error points (EP) while seven (13%) accrued the 15 or fewer EP for a satisfactory one-rescuer CPR attempt (range: 5-65 EP). No significant improvement in skill could be found with increasing length of residency (Table 2B).

Table 2B: Results of the one-rescuer CPR attempt by the modality in the Berden Scoring system. The numeric results, its commensurate number of error points (EP) as well as the median and mean EP are reported. * indicates a significant difference (p<0.05) between the labeled groups. Both ventilation interval (vent. int.) and ventilation volume (vent. vol.) are shown to be particularly poorly performed. Please see Appendix for descriptions and abbreviations.

<table>
<thead>
<tr>
<th>Modality</th>
<th>Mean ± sd (n = 55)</th>
<th>Intern</th>
<th>1st year</th>
<th>2nd year</th>
<th>3rd year</th>
<th>4th year</th>
<th>5th year</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hand position</td>
<td></td>
<td></td>
<td>67% Correct</td>
<td>40% Correct</td>
<td>86% Correct</td>
<td>72% Correct</td>
<td>79% Correct</td>
</tr>
<tr>
<td>Comp.depth (mm)</td>
<td></td>
<td></td>
<td>43 ± 11 4 &amp; 5 EP</td>
<td>43 ± 9 3 &amp; 10 EP</td>
<td>44 ± 11 3 &amp; 0 EP</td>
<td>39 ± 8 3 &amp; 5 EP</td>
<td>41 ± 8 3 &amp; 0 EP</td>
</tr>
</tbody>
</table>
Of the modalities within one-rescuer CPR, the mean “ventilation time” of 10 ± 3 sec. lay well outside accepted limits of circa 5 seconds with 46 (84%) participants being slow in restarting compressions. Most participants found mouth-to-mouth ventilation difficult: only 20 (36%) participants carried this out correctly. Participants overinflated the lungs in 75% of attempts, with exaggerated overinflation (> 1.5 l) occurring in 29%; only 7 (13%) inflated correctly. In the modality "compression frequency" 23 (42%) participants performed correctly. The median of compression frequencies (cpm) lay in the 101-120 cpm (5 EP) range, with a significant difference between interns and first-year residents (p<0.05). A 15:2 rhythm was used by 29 (52%) participants, while 20% used an unrecognizable rhythm and 21% a 10:2 or 5:2 compression-ventilation rhythm.

No correlations were found between either "opinion of own skills and knowledge" with actual EP ($r_s = -0.3$), or the results of the multiple-choice questions with actual EP ($r_s = -0.26$), though results from the multiple-choice category "practical" correlated with actual EP assigned ($r_s = 0.83$, p<0.01).

### 11.4 Discussion

This study inventoried anesthesiology interns' and residents' ACLS skills, in order to evaluate needs for a well-defined training program. The study demonstrated that 55% of the multiple-choice questions were answered correctly. In the skills assessment, the Berden Scoring system was employed. (Table 3) The findings revealed a median of 35 EP, with only 13% of the participants demonstrating satisfactory skills.

**Table 3:** Berden Scoring table. This scoring system assigns error points to six independent modalities within one-rescuer CPR using the printout of the Laerdal Recording Resusci Anne. The number of error points assigned is weighted as to severity, with "zero" error points for the correct technique. Summation of the points for the six modalities leads to a total score. Up to and including 15 error points is considered an adequate resuscitation attempt (to the left of the vertical line in Figure 2). Hand position is judged by the observer. Ventilation interval is the time between the last compression of one cycle of 15:2 and the first compression of the next cycle.
It is difficult to compare these results with those assembled in Table 1, since focus as well as methodology in those studies ranged extensively. Information concerning judgment of competence is often incomplete or lacks validation. In addition, those directed at anesthesiologists were performed in countries with a strong ACLS course and certification tradition. Our study represents the first broad investigation into the relevance of ACLS done in continental Europe with a large homogeneous group of participants and looks both at individual skills and knowledge as well as for correlations with other factors. Our study is no exception in demonstrating generally poor results with 4 (7%) scoring adequately in both the multiple-choice and one-rescuer CPR skills. Thirty one (56%) participants were judged to have scored unsatisfactorily in both sections of the evaluation. The findings of this new study appear to be supported in part by the results obtained by Bell et al. They reported a lack of BLS skills in ACLS certified trainee anesthesiologists using as cutoff "70% correct" as measured by a Skillmeter Resusci Anne. They also noted poor adherence to accepted algorithms for arrhythmia management.

Our study does, however, show a positive correlation between length of training and CPR-related knowledge, which was not seen in the study by Bell et al. We assessed the knowledge needed to understand algorithms rather than adherence to them. A number of studies support our findings of medication and pediatric knowledge including some involving recently (re)certified ACLS physicians. Interestingly, Buss et al. described the same difficulties with accurate

<table>
<thead>
<tr>
<th>Table 3: Scoring modality</th>
<th>Criteria</th>
<th>Error points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hand position</td>
<td>correct</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>incorrect</td>
<td>5</td>
</tr>
<tr>
<td>Compression frequency (cpm)</td>
<td>&lt;= 39</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td>40 - 59</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td>60 - 79</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>80 - 100</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>101 - 120</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>121 - 140</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>&gt; 140</td>
<td>15</td>
</tr>
<tr>
<td>Compression depth (mm)</td>
<td>&lt;= 21</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td>22 - 29</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>30 - 37</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>38 - 52</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>53 - 60</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>&gt; 60</td>
<td>10</td>
</tr>
<tr>
<td>Compression relaxation ratio</td>
<td>&lt; 0.6</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>0.6 - 1.4</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>&gt; 1.4</td>
<td>10</td>
</tr>
<tr>
<td>Ventilation volume (l)</td>
<td>&lt; 0.49</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td>0.50 - 0.79</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>0.80 - 1.20</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>1.21 - 1.50</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>1.51 - 2.0</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>&gt; 2.0</td>
<td>15</td>
</tr>
<tr>
<td>Ventilation interval (sec)</td>
<td>&lt;= 4.9</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>5.0 - 6.0</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>6.1 - 8.0</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>8.1 - 10.0</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td>&gt; 10</td>
<td>20</td>
</tr>
</tbody>
</table>

Chapter 11: Training needs and qualifications
knowledge and stated that their pediatric residents counted on the on-duty anesthesiologist for aid during an intervention.

Other studies, with 55% to 79% being judged competent, contrast strongly with the 7% in our study. These studies all stated that frequent training as well as frequent CPR interventions were the reasons for these results. Of our participants, 65% stated that no CPR training had been attended in over two years even though the preparatory course for the national Board includes eight hours of intensive training in ACLS related subjects. In our study multiple-choice scores as well as EP for second- and third-year residents does not reflect this training at all: clearly, well-defined goal-oriented training may improve recall and performance.

Frequent CPR interventions has also been suggested as an effective factor in maintaining knowledge, while others describe insufficiently frequent practice in interventions as an important contributing factor for the poor scores. Actual presentation of this data is nonetheless scarce, as is correlation with proficiency in relation to a minimum number of interventions. In our study, the number of CPR interventions per year, confidence, extent of ER, ICU or EMT experience as well as training in what the participant had experienced as advanced cardiac life support, all proved to be poor indicators of ability. In 1990, Marteau et al. stated that frequent interventions increase confidence though not skill in a non-anesthesiologist evaluation. Our results, while corroborating their work in part, also show that our participants were aware of their proficiency.

For professionals, carefully structured, intensive ACLS courses are popular in English-speaking countries leading to (re)cetification. However, the term ACLS is used as a gold standard even in countries where such courses are not taught (i.e., used for general, related lectures) and leading to "ACLS-trained" being misconstrued. As yet, training of anesthesiologists in ACLS has not been accepted as sufficiently important in most of mainland Europe as to require separate certification, leaving responsibility for training at local or individual levels.

As in several countries where mandatory (re)cetification has been welcomed by anesthesiologists for improvement of their skills in a positive, directed, and controlled way, we feel that regular and intensive (standardized) ACLS training including emphasis on BLS skills, medication and pediatric aspects and also perhaps specific management techniques is the key to eliminating the lapses shown by our data.

We recommend that interns and residents on the European continent be given regular, mandatory, hands-on ACLS training at a high frequency, preferably using European-oriented Guidelines. This training should be in course format and focused on BLS skills and aspects relevant to the anesthesiologist or other specific physician groups' needs.

### 11.5 Acknowledgments

This study was supported in part by Laerdal, Inc. (Brussels, Belgium) and MediScore (Bodegraven, The Netherlands).
11.6 References

Chapter 11: Training needs and qualifications

Applicability of insights in force and pressure: A CPREzy study.
The quality of chest compressions by trained personnel:
The effect of feedback, via the CPREzy, in a randomized controlled trial using a manikin model.

Part of this material was presented as preliminary reports at the “3rd Med Conference on Emergency Medicine, Feb 9-13 2005 Leuven B, as well as at the 3rd Mediterranean Emergency Medicine Conference, Sept 1-5 2005, Nice, Fr.
Applicability of insights in force and pressure: A CPREzy study.

The quality of chest compressions by trained personnel: The effect of feedback, via the CPREzy, in a randomized controlled trial using a manikin model.


Part of this material was presented as preliminary reports at the “3rd Med Conference on Emergency Medicine, Feb 9-13 2005 Leuven B, as well as at the 3rd Mediterranean Emergency Medicine Conference, Sept 1-5 2005, Nice, Fr."
Abstract

The ability, after training, to perform effective cardiac compressions has been found to be poor and to decrease rapidly. We assessed this quality with and without a noninvasive feedback device, the CPREzy™, during a 270 second CPR session in an unannounced, single blinded manikin study using 224 hospital employees and staff chosen at random and using a non-crossover design.

The two groups assessed their knowledge and skills as adequate. However, the control group (n = 111) had significantly more difficulty compressing more than 4 cm deep at all (25 versus 1 candidate in the CPREzy group) P = 0.0001. The control group compressed ineffectively 36% (± 41%) of all compressions as opposed to 6 ± 13% in the CPREzy group (n =112, P = 0.0001). If compressions were effective initially, the time until > 50% of compressions were less than 4 cm was 75 ± 81 in the control group versus 194 ± 87 seconds in the CPREzy™ group (P = 0.0001 [-180 to -57.5]). Even without training in its use, our candidates used the CPREzy™ effectively, without coaching, after only seconds of instruction.

Against the background knowledge that estimation of compression depth by the caregiver or other team members is difficult, and that performing effective compressions is the cornerstone of any resuscitation attempt, our data strongly suggests that a feedback device such as the CPREzy™ should consistently be used during resuscitation.
Chapter 12: Applicability of force

Abstract
The ability, after training, to perform effective cardiac compressions has been found to be poor and to decrease rapidly. We assessed this quality with and without a noninvasive feedback device, the CPREzy ™, during a 270 second CPR session in an unannounced, single blinded manikin study using 224 hospital employees and staff chosen at random and using a non-crossover design. The two groups assessed their knowledge and skills as adequate. However, the control group (n = 111) had significantly more difficulty compressing more than 4 cm deep at all (25 versus 1 candidate in the CPREzy group) P = 0.0001. The control group compressed ineffectively 36% (±41%) of all compressions as opposed to 6 ± 13% in the CPREzy ™ group (n = 112, P = 0.0001). If compressions were effective initially, the time until > 50% of compressions were less than 4 cm was 75 ± 81 in the control group versus 194 ± 87 seconds in the CPREzy ™ group (P = 0.0001 [-180 to -57.5]). Even without training in its use, our candidates used the CPREzy ™ effectively, without coaching, after only seconds of instruction.

Against the background knowledge that estimation of compression depth by the caregiver or other team members is difficult, and that performing effective compressions is the cornerstone of any resuscitation attempt, our data strongly suggests that a feedback device such as the CPREzy™ should consistently be used during resuscitation.
12.1 Introduction

Hospitals are settings to which the ‘Chain of survival’ applies. Interest has recently shifted from improvements in Advanced Cardiac Life Support equipment toward improvements in Basic Life Support (BLS). As in any other emergency medical system, in our hospital first responders provide basic life support until the Quick Response Team (QRT) arrives. The first responders continue to assist as needed, for example in performing the chest compressions. The importance of consistent and adequate chest compressions has been stressed\(^1,2\) with extrapolation suggesting that chest compressions may be the crucial factor in improving outcome.\(^3,4\)

While all personnel in our hospital, not only nursing staff, participate in structured, intensive training and certification, training frequency may be too low to guarantee adequate skills. Loss of skills has been observed as early as days after training.\(^5,6\) A further complicating factor is the poor ability of caregivers to assess their own,\(^7,8\) as well as others’ resuscitation skills, especially in actual resuscitation cases.\(^9\) Feedback has now been introduced\(^10\) to reduce subjective evaluation of caregivers during external chest compressions. Initial tests have been encouraging.\(^11,12\)

For the purpose of expanding these tests and to gain further understanding of the mechanical engineering involved, we performed two types of evaluations with the CPREzy™ (Health Affairs, London, UK). These were (a) assessment of compression skills of hospital personnel with and without the use of the noninvasive feedback device and (b) description of the indicators on the CPREzy as a mechanical model on a stiff surface with and without an underlying manikin or human chest.

12.2 Materials and methods

All medical, nursing and support staff members in our hospital were eligible for participation in the study. During 10 days, on which no training was being given, and using a roster to ensure representation of the different departments, the investigators toured the hospital, recruiting staff as they were seen. By referring to management support if needed, staff could not “bow out” using work load, other tasking, or even coffee breaks as excuses.

Candidates were individually “asked” to come to a quiet, private, location where the assessment was to be performed. The first questionnaire (Appendix Va) was filled out, followed by randomization to the ‘control’ or ‘Ezy’ group, and a standardized briefing given. Assessment followed using one of four SkillReporter Resusci Annes (Laerdal, Stavanger, NO). The practical session, with ventilation being performed by a non-obstructive investigator who offered neither suggestions nor comments, continued for at least 240 seconds, but no longer than 270 seconds. This time period reflects the maximal time a caregiver might be expected to perform chest compressions in one uninterrupted period in our hospital, as allowed by the hospital resuscitation standing orders. It was deemed to be long enough to allow for fatigue, as suggested by Baubin.\(^13\) After the practical session, a second questionnaire (Appendix Vb) was filled out. The candidate was also asked to sign a form giving permission to use the data and to access their training database. The two questionnaires and the SkillReporter’s written reports were labelled with a unique randomization number. Two investigators experienced in the use of the standardized scoring system, blinded for the randomization, scored the results based on a fixed list of parameters (Table 1), and entered directly into the SPSS database. The candidate was not told how he/she had performed.
standing orders. It was deemed to be long enough to allow for fatigue, as suggested by Baubin. 

This time period reflects the maximal time a caregiver might be expected to perform chest 
suggestions nor comments, continued for at least 240 seconds, but no longer than 270 seconds.

Candidates were individually "asked" to come to a quiet, private, location where the assessment 
was to be performed. The first questionnaire (Appendix Va) was filled out, followed by 

...followed using one of four SkillReporter Resusci Annes (Laerdal, Stavanger, NO). The practical 
randomization to the 'control' or 'Ezy' group, and a standardized briefing given. Assessment 
was to be performed. The first questionnaire (Appendix Va) was filled out, followed by 

...followed using one of four SkillReporter Resusci Annes (Laerdal, Stavanger, NO). The practical 
randomization to the 'control' or 'Ezy' group, and a standardized briefing given. Assessment 
was to be performed. The first questionnaire (Appendix Va) was filled out, followed by 

All medical, nursing and support staff members in our hospital were eligible for participation in 
the study. During 10 days, on which no training was being given, and using a roster to ensure 

12.1 Introduction

Hospitals are settings to which the 'Chain of survival' applies. Interest has recently shifted from 

improvements in Advanced Cardiac Life Support equipment toward improvements in Basic Life 

While all personnel in our hospital, not only nursing staff, participate in structured, intensive 

training and certification, training frequency may be too low to guarantee adequate skills. Loss of 

of consistent and adequate chest compressions has been stressed 1,2 with extrapolation suggesting 

continue to assist as needed, for example in performing the chest compressions. The importance 
provides basic life support until the Quick Response Team (QRT) arrives. The first responders 

of the different departments, the investigators toured the hospital, recruiting staff 
i...from management support if needed, staff could not "bow out" using 

Table 1: Listing of points scored for each candidate. Scoring was done by one of two investigators 
b...blinded for the randomization and entered into a database directly.

<table>
<thead>
<tr>
<th>Overall</th>
<th>Per compression set (15:2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Randomization (blinded)</td>
<td>Block number</td>
</tr>
<tr>
<td>Manikin code</td>
<td>Time block started (sec)</td>
</tr>
<tr>
<td>Total # compressions</td>
<td>Number leaning</td>
</tr>
<tr>
<td>Total time (sec)</td>
<td>Actual depth / leaning compression</td>
</tr>
<tr>
<td>Total number 15:2</td>
<td>Direction leaning</td>
</tr>
<tr>
<td>Mean compression freq</td>
<td>Actual depth / compression</td>
</tr>
<tr>
<td>Mean compression depth</td>
<td>Mean depth / 15 compressions</td>
</tr>
<tr>
<td>Compression depth lost (mm)</td>
<td>Depth loss / 15 compressions</td>
</tr>
<tr>
<td>Total # compressions</td>
<td>Number ‘out of range’ / 15 compressions</td>
</tr>
<tr>
<td>Total time (sec)</td>
<td>Compression frequency</td>
</tr>
</tbody>
</table>

The candidates randomized to the 'Ezy' group used the CPREzy™ (Health Affairs, London, UK). 
This device was not in use in our hospital and was unknown to all the candidates. It is a small, 
light (260 gm) device, with a resting height of 5 cm, a base of 5.5 x 18 cm, and is powered by a 9 
volt battery (Figure 1).

Figure 1: The CPREzy as used in the trial. The large surface for compression (A) is labelled, as 
is the LED indication for pressure applied (B). The on/off button (C) is next to the metronome 
light (D). The arrows indicate the moving planes.

Placement is on the sternum, and it features a drawing to assist herein. It has a non-slip pad on its 
bottom. When turned on (green) indicator lights show impression force as correlated to the size 
(weight) of the patient. The indications start with one light for a 'child' (40 kg/90 lbs); two lights 
for a ‘small adult’ (55 kg/120 lbs), three for an ‘average adult’ (75 kg/165 lbs), four lights for a 
‘large adult’ (90 kg/200 lbs) and a fifth (red) light for ‘extra large adult’. The force needed to 
activate the indicator lights at the different sizes is 23 kg, 32 kg, 41 kg, 50 kg, and 54 kg, 
respectively. 11,12 A metronome flashes an orange light and emits a 60 dB beep at 100 min⁻¹ in
accordance with International Liaison Committee on Resuscitation (ILCOR) Guidelines.\textsuperscript{14} Candidates randomized to the ‘control’ group performed regular (hands on sternum) chest compressions. Candidates were also stringently requested to maintain secrecy if asked about their temporary absence. Approval for the study was given by the Medical Ethics Committee.

For the registration of skills the Resusci Anne \textsuperscript{TM} was connected to its external monitor (i.e., the “SkillReporter”) which uses lights to give feedback and which also produces a written record although not in ‘real time’. During the assessment period, the lights on the SkillReporter where not used and the device turned so that they, nor the print out, were visible. On the written record, or stroke, time is a fixed parameter. Movement of the chest is recorded as curves, as are ventilation volumes. Compression-relaxation ratios must be calculated. The SkillReporter also has an internal data log system which can be printed at the end of a stroke. The usefulness of this data in our experimental setup was limited as we analyzed 270 seconds and worked on a compression-to-compression basis.

Each stroke was inspected carefully, using a standard technique first developed by Berden \textit{et al.}\textsuperscript{15} as a standardizing evaluation tool. This system is in regular use for the courses given in our hospital and has been adapted to current ILCOR Guidelines. A measuring ‘ruler’ is used by us to further improve accuracy and limit interobserver scoring variations.

The SkillReporter Resusci Annes were tested beforehand by the hospital medical instrumentation service to validate the methods. In the first series, a standardized force was applied to the manikin and correlated with the SkillReporter written record for compression depth. No relevant (<5\%, \pm 2 mm inter-manikin variation) differences could be found in the range expected to be relevant during the study of 20, 30, 40, 50 and 60 kg. A second test evaluated these forces applied to the CPREzy\textsuperscript{TM} in relation to the indicator lights and the actual depth in the manikin while on a firm surface. The force to CPREzy indicator lights confirmed the manufacturer’s specifications,\textsuperscript{12} but also demonstrated a larger spread during the dynamic testing, as the interpretation of the indicator light scale is approximate with (see above) steps between lights of 4 to 9 kg. This reflected itself in a maximal 5 mm spread, both within and between manikins. The difficulty lay in applying the exact force needed to just light the indicator light during dynamic testing. During static testing, we confirmed the data from Boyle and Perkins\textsuperscript{11,12} in that at least three lights, and optimally, four lights should (just) be illuminated to achieve adequate depths in our model (Appendix V). The results of theses tests were also used to check for interobserver variability, which was negligible.

This pilot used a technique described elsewhere.\textsuperscript{16} The validation tests were performed using the DPM3 Universal Biometer (DPM3, BIO-Tek Instruments Inc., Winooski, VT, USA), which can measure pressure applied to 1\% of accuracy.

The principal endpoint was the number of correct compressions a caregiver could perform during the test session. Numeric assessment of compression depth, incomplete relaxation, compression to relaxation ratio and compression frequency were scored as defined by the 2000 ILCOR guidelines. As each candidate had been trained, these scores were related to their skills during the course exit test. More specifically, hand position was scored as in error if lateral force was registered by the manikin, and incomplete relaxation if the ‘resting’ chest position was not returned to (\geq 1 mm) between compressions. Each variable was scored independently of each other. Secondary endpoints were: the ability of the candidates to assess their compressions and the work required, the loss of compression depth during the course of the session, the frequency with which the cardiac compressions were performed, and whether individual characteristics, such as age, weight, body mass index (BMI) influenced their capabilities.

For each candidate, the results were entered into an SPSS (v12) database for statistical analysis. P < 0.05 was taken to be significant, with data being presented as mean (\pm standard deviation), with
95% confidence intervals presented in brackets. After testing for normality using the Kurtosis test and the Levene’s test for equality in variance, the (two-tailed) independent samples t-test was used for parametric data, with correlations sought using the (two-tailed) Pearson’s coefficient, with missing cases excluded pair wise, for parametric values. Using the general linear model, repeated measures analysis as performed with time and depth as variables. The Huynh-Feldt epsilon was used if sphericity conditions were not demonstrated. The Mann-Whitney U and Chi-squared were used for independent sample analysis of non-parametric data. The (2-tailed) Spearman’s rho, was then also used, with missing cases excluded pair wise.

12.3 Results

Two hundred twenty four candidates, including physicians, nursing and non-nursing staff, were included (Table 2). One candidate was not randomized due to recent neck hernia complaints and was not replaced. The 223 remaining were randomized to the ‘control’ and the ‘Ezy’ groups (n = 111 and 112 candidates, respectively). All candidates were able to produce usable records that were entered into the database for analysis. Two were unable to complete the nominal 240 seconds of chest compressions (protocol deviation) due to technical difficulties with the manikin. In four cases the candidate stopped early due to shortness of breath (n = 1) or other physical discomfort (n = 3). Their data remained in the database for analysis. Failures of the CPREzy™ did not occur.

Table 2: Descriptive statistics for the candidates. Data is presented as mean ± standard deviation and the 95% confidence interval in square brackets, except when non-parametric (median + 95% confidence interval). n = number of cases. This is reported as the actual number for that individual variable if data was missing. No significant differences were found between the two groups, for any category even though there was a tendency to more physicians in the control group. The candidate’s abilities are reported on the basis of the post-test skill measurements.

<table>
<thead>
<tr>
<th>Gender</th>
<th>Control Mean (+ SD)</th>
<th>CPREzy Mean (+ SD)</th>
<th>P [ 95% CI]</th>
</tr>
</thead>
<tbody>
<tr>
<td>N =111</td>
<td>Female: 72 Male: 39</td>
<td>Female: 82 Male: 30</td>
<td>N.S. (0.521)</td>
</tr>
<tr>
<td>N =112</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>36.6 (± 10.5)</td>
<td>35.4 (± 10.2)</td>
<td>N.S. (0.401)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>72.8 (± 14.4)</td>
<td>71.6 (± 11.9)</td>
<td>N.S. (0.518)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>173.2 (± 9.5)</td>
<td>172.2 (± 8.8)</td>
<td>N.S. (0.414)</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>23.8 (23.5 -24.8)</td>
<td>23.9 (23.5 -24.9)</td>
<td>N.S. (0.822)</td>
</tr>
<tr>
<td>Ability at end of training (number &amp; % of ineffective compressions)</td>
<td>155 (± 50)</td>
<td>139 (± 48)</td>
<td>N.S. (0.626)</td>
</tr>
<tr>
<td></td>
<td>4.5 (± 9)</td>
<td>4.8 (± 10)</td>
<td>N.S. (0.880)</td>
</tr>
<tr>
<td>Time since training (months)</td>
<td>16.4 ± 15</td>
<td>14.5 ± 14</td>
<td>N.S. (0.09)</td>
</tr>
<tr>
<td>Actual (recent) BLS experience (n)</td>
<td>26</td>
<td>23</td>
<td>N.S. (0.603)</td>
</tr>
</tbody>
</table>

The two groups were well balanced in their physical characteristics and potential skills. In particular, similarity in the ‘time since last training’ (16.4 versus 14.5 months) and the
‘compressions skills at the end of training’ (95% versus 95% effective compressions) in the control and the CPREzy™ groups, respectively, suggest that randomization was adequate (Table 2). In the pre-assessment questionnaire all the candidates reported that they were capable of performing BLS-CPR (6.9 (± 1.4) versus 7 (± 1.3); P = 0.536) at the end of their last course (Table 3). They estimated that their current skills were 5.7 (± 1.7) out of 10 versus 5.8 (± 1.8), (control and CPREzy groups, respectively; P = 0.538) with knowledge estimations essentially the same. In the post-assessment questionnaire these estimations became 5.9 (± 1.6) and 5.9 (± 1.8), P = 0.591. Both groups reported that they felt that they had performed circa 53% of the compressions correctly, while the CPREzy group was less confident in actually having achieved and maintained adequate depth (4.7 (± 1.8) versus 5.2 (± 1.8) for the control group, respectively, with P = 0.039 [5% CI = 0.025-0.98]). Similar results were reported for the physical work needed to perform chest compressions. These were 4.8 (± 2.2) versus 4.7 (± 2.2), respectively (P = 0.714), on a 10-point scale with zero being ‘extremely tiring’ and 10 being ‘no effort at all’ (Table 3).

Table 3: Pre- and post assessment questionnaire. The VAS score is used, with a non-calibrated 10 cm line on which the candidate marked their answer. In the two questions labeled ‘knowledge’, the candidate was asked to answer with the ILCOR guideline parameters. This was scored as correct or as not correct. N.S. = not significant. Data is presented as mean (± standard deviation), except when non-parametric. If significant the 95% confidence interval [95% CI] is reported. N = number of cases. This is reported in the individual variable if data was missing. No significant differences were found between the two groups, except for in the difference between estimation of compression depth (P = 0.039). This difference does not seem clinically relevant.

<table>
<thead>
<tr>
<th></th>
<th>Control Mean (±SD)</th>
<th>CPREzy Mean (±SD)</th>
<th>P [95% CI]</th>
</tr>
</thead>
<tbody>
<tr>
<td>VAS score</td>
<td>N=111</td>
<td>N=112</td>
<td></td>
</tr>
<tr>
<td>Pre assessment</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Knowledge: at the end of last training course?</td>
<td>7.0 (± 1.4) (N = 109)</td>
<td>7.0 (± 1.3) (N = 109)</td>
<td>N.S. (0.891)</td>
</tr>
<tr>
<td>Knowledge: this minute?</td>
<td>5.7 (± 1.7)</td>
<td>5.8 (± 1.8)</td>
<td>N.S. (0.538)</td>
</tr>
<tr>
<td>Post assessment</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Opinion: (0-10) How good were compression?</td>
<td>5.9 (± 1.6) (N=110)</td>
<td>5.9 (± 1.8) (N=110)</td>
<td>N.S. (0.591)</td>
</tr>
<tr>
<td>Opinion: Depth (5 = correct)</td>
<td>5.2 (± 1.8)</td>
<td>4.7 (± 1.8)</td>
<td>0.039 [0.025-0.98]</td>
</tr>
<tr>
<td>Opinion: effective compressions (%)</td>
<td>53 (± 21) (N = 110)</td>
<td>53 (± 20) (N = 110)</td>
<td>N.S. (0.911)</td>
</tr>
<tr>
<td>Opinion: how tiring was CPR?</td>
<td>4.8 (± 2.2)</td>
<td>4.7 (± 2.2)</td>
<td>N.S. (0.714)</td>
</tr>
<tr>
<td>Correct depth is? (knowledge)</td>
<td>32</td>
<td>39</td>
<td>N.S. (0.338)</td>
</tr>
<tr>
<td>Opinion: (0-10) How good was frequency?</td>
<td>6.3 (± 1.6) (N=109)</td>
<td>6.2 (± 1.7) (N=109)</td>
<td>N.S. (0.407)</td>
</tr>
<tr>
<td>Correct freq. is? (knowledge)</td>
<td>46</td>
<td>40</td>
<td>N.S. (0.381)</td>
</tr>
<tr>
<td>Opinion of own confidence</td>
<td>5.9 (± 1.8)</td>
<td>6.0 (± 1.9)</td>
<td>N.S. (0.688)</td>
</tr>
</tbody>
</table>

Practical skills differed strongly between the two groups (Table 4). The control group had significantly more difficulty with achieving and maintaining effective compressions over time even though this was not reflected in their opinion during the post-assessment questionnaire (P = 0.591). This lack of performance could be expressed in the number of candidates unable to perform compression of more than 4 cm (25 in the control group versus 1 in the CPREzy group; P = 0.0001). Of the remaining candidates, 48 members of the control group started with adequate compressions but lost compression depth progressively, reaching < 4 cm by 75 (± 81) seconds, without recognizing or correcting this inadequacy for the remainder of the trial period. In the
CPREzy group this minimal effective depth threshold was not maintained by only 11 members, and then some two minutes later at 194 (± 87) seconds (P = 0.0001 between groups for time as well as number of candidates reaching threshold, see also Figure 2 [I -180 to -57.5]

Table 4: Practical skills assessment. This table summarizes the practical skills. mm = millimeters of impression; sec = seconds after initiation of CPR; N = number of cases; cpm = compressions per minute. Ineffective compressions are those outside the ILCOR range. If a subgroup is reported, the actual number of cases is listed. Leaning was scored as present if the registration showed > 1 mm non return to resting position. Hand position was scored if an ‘incorrect hand position’ exclamation mark was listed on the written record (not specific for the four potential sites of the error: see text for explanation).

<table>
<thead>
<tr>
<th></th>
<th>Control Mean (± SD)</th>
<th>CPREzy Mean (± SD)</th>
<th>P</th>
<th>[95% CI]</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>111</td>
<td>112</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Actual time (sec)</td>
<td>260 (± 26)</td>
<td>258 (± 23)</td>
<td>N.S. (0.589)</td>
<td></td>
</tr>
<tr>
<td>Actual number of 15:2 cycles</td>
<td>21 (± 4)</td>
<td>21 (± 3)</td>
<td>N.S. (0.784)</td>
<td></td>
</tr>
<tr>
<td>Mean depth (mm)</td>
<td>40 (± 9)</td>
<td>45 (± 4)</td>
<td>0.0001</td>
<td>[-0.32--2.15]</td>
</tr>
<tr>
<td>Depth loss (mm)</td>
<td>4 (± 5)</td>
<td>3 (± 4)</td>
<td>N.S. (0.143)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(N = 106)</td>
<td>(N = 109)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Incidence of ineffective chest compressions (N)</td>
<td>25 (never correct)</td>
<td>1 (never correct)</td>
<td>0.0001</td>
<td>0.001</td>
</tr>
<tr>
<td>N of candidates compressing &lt; 4 cm consistently (after an adequate start)</td>
<td>48</td>
<td>11</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time until compressions become &amp; remained &lt; 4 cm (sec)</td>
<td>75 (± 81)</td>
<td>194 (± 87)</td>
<td>0.0001</td>
<td>[180- -57.5]</td>
</tr>
<tr>
<td>Total ineffective compression (% of all compressions)</td>
<td>36 (± 41)</td>
<td>6 (± 13)</td>
<td>0.0001</td>
<td>[22.7 - 38.7]</td>
</tr>
<tr>
<td>Ineffective compressions (n)</td>
<td>94 (± 104)</td>
<td>15 (± 33)</td>
<td>0.0001</td>
<td>[59 - 100]</td>
</tr>
<tr>
<td>Leaning (incidence)</td>
<td>13</td>
<td>14</td>
<td>N.S.</td>
<td></td>
</tr>
<tr>
<td>Incorrect hand position (incidence 15:2)</td>
<td>136</td>
<td>88</td>
<td>0.001</td>
<td></td>
</tr>
<tr>
<td>Actual compression frequency (cpm)</td>
<td>106 (± 21)</td>
<td>102 (± 10)</td>
<td>N.S. (0.056)</td>
<td></td>
</tr>
</tbody>
</table>

Time (sec)

Chapter 12: Applicability of force 241
The number of ineffective compressions for the control group, within each block of 15 compressions, ranged from a mean of 4 (± 6) initially to 7 (± 7) at 240 seconds, as opposed to 1 (± 4) consistently throughout the trial for the CPREzy group (see also Figure 3, P = 0.0001 CI = 59 - 100).

Figure 2: Compression depth over time (data presented as mean ± SD for each time segment). Compression depth measured from zero (resting). Measurements of a compression-to-compression basis, with the series of compressions being scored closest to the time interval.

Figure 3: Number of ineffective compressions by group over time (data presented as mean ± SD). Measured on a compression-to-compression basis. Ineffective defined as compressions (measurably) less than 4 cm depth.

The overall percentage of candidates not reaching a mean 4 cm impression depth at each block of 15 compressions ranged from 21% at 15 seconds to 38% at 270 seconds. The incidence of incomplete relaxation (e.g., leaning on the chest) was limited in both groups (13 and 14 cases, respectively), while 50 candidates in the control group and 98 in the CPREzy group demonstrated consistently correct hand positions (P = 0.001). Compression frequency did not differ significantly between groups (Table 4).

Correlations between the use of the CPREzy, the adequacy of their compressions and potential confounders such as caregiver physiognomy, their time since last training, weight, and their own opinions of their abilities, could not be demonstrated. Notably, a caregiver’s (high) opinion could not be correlated to their actual ability when the skill was expressed as adequate compressions (P = 0.38, r = -0.084).

12.4 Discussion

This unannounced study assessed the compression skills of 224 trained caregivers with and without the use of an unfamiliar noninvasive feedback device, the CPREzy™ in a manikin setting. Two investigators, blinded for the randomization, and using a standardized scoring system, evaluated the effectiveness on a compression-to-compression basis and scored pre- and post-assessment questionnaires. The use of a feedback device in a large, non-crossover, manikin
study with caregivers at known levels of training and time since training, and a questionnaire, had not been performed.

We found, as expected, a generally moderate ability to perform adequate chest compressions at an average of 15 months after the most recent training, with a mean of 6 of each 15 compression series being inadequate over time. While the assessment lasted up to 270 seconds, it demonstrates (Figure 3) that the period during which one caregiver may compress the chest effectively, may need to be further limited to about 120 seconds. When the CPREzy™ is used, the overall percentage of adequate compression during the assessment period was increased significantly, from an average of 9 (± 8) sufficiently deep compressions in the control group to at least 14 (± 2) per 15 compression series. This improvement in effectiveness is both for consistency of depth as well as for adequacy of depth. Use of the CPREzy™ was neither correlated with improvements in frequency, nor did it increase incomplete relaxation between compressions. However, the low incidence of these errors may have contributed to this outcome.

Our study demonstrates that better definition and consistency of force during chest compressions is feasible in a population of caregivers not specifically trained in the use of the CPREzy. This is a significant prerequisite for application in patient resuscitation. Compression force and depth assessable during chest compressions has received little attention. This may have been caused by early emphasis on getting the lay public to perform compressions to the exclusion of aspects difficult to teach. Outcome studies have been careful to score ‘early access’ while avoiding estimation of adequacy.14, 17

Early work, performed by Thomas et al.10 reports on the use of a force indicating gauge to improve depth estimation. They report improvement of compression efficacy from 33 to 96% in a manikin study of trained flight nurses using a crossover design.10 However, they drew no general conclusions. Their impressive results could not be reproduced entirely by Elding et al.,17 although this study also demonstrated statistically significant improvement in compression efficacy and general technique.

The CPREzy™, first described by Boyle et al.11 in 2002 in a limited group assessed without and with the CPREzy™ on consecutive days, found a threefold improvement (13 to 42%) in effective compressions if the CPREzy™ was used. The suggestion that this improvement may not translate to clinical improvement seems justified in their study, as the rate of effective compressions remains low even after instruction and introduction of the feedback device. Perkins et al.12 validated Boyle’s results using a small group of medical students when resuscitating on a bed. In this later study, the students received instructions, practiced, and were told how many indicator lights were optimal, with some of the test candidates having been active in the validation series.

Our data confirms but also expands on this experience. While our improvement, expressed as a percentage, is smaller, it brings the percentage of adequate compressions in line with what seem to be realistic clinical demands. In addition, we used an interrupted compressions model in order to simulate the discontinuity this brings with it, incorporating a maximum time that one caregiver may need to perform BLS before being relieved. We avoided a crossover design to exclude a learning curve as may have occurred in Boyle’s and Perkins’s studies. A potential individual learning effect of uncertain magnitude cannot be ruled out. We increased the number of participants to limit any bias caused by skills (Table 2). Perhaps most importantly, we did not train our caregivers in the use of the CPREzy™: they were confronted with it as randomized, and were instructed to “use the lights as indicated and begin immediately” so as to standardize unfamiliarity most likely to occur in the clinical setting. We also demonstrate that improvement is independent of time since training, caregiver weight and function within the hospital. The mean
time since training in our assessment was 15 months (range 0–37 months with a normal distribution). A subgroup analysis of those with training less than 6 months earlier and those with training between 12 and 18 months demonstrated that the CPREzy maintains skills, as suggested by others. These results demonstrated that the use of the CPREzy might increase the useful interval extensively, giving it both a teaching as well as practical role.

The need for quality in basic life support, both early and during the advanced stages of a resuscitation effort need not be explained. Without a feedback device, such as the CPREzy, the caregiver as well as the physician have to depend on their ‘experience’ and ‘memory’ to evaluate the effectiveness of the compressions, while evidence suggests that this is neither taught in courses nor clinically possible for instructors or caregivers. Their experience relates to manikins in the training situation, and negates understanding of the variables such as the loss of caregiver capabilities over time, individual chest wall stiffness and chest diameter. The characteristics of a device, such as the CPREzy, with its affect on the force applied to the individual patient (Appendix 2) is, perhaps regrettably, unclear to many. The CPREzy provides one of these variables and empirically a second, thereby reducing the number of unknowns in a user-friendly manner. The feedback device also gives the caregiver a tool on which to benchmark compressions, allowing physician delegation and monitoring of this basic life support task. Its use can be considered an adjuvant to CPR as are ETCO₂ monitoring and expensive techniques such as impedance evaluation built into defibrillators.

In our hospital the CPREzy is brought to the scene by the advanced life support team and implemented as the first step in their protocol: although the first minutes may be suboptimal, it allows improved maintenance of chest compressions during the advanced intervention which may continue for up to an hour.

The importance of chest compressions has been rediscovered. The exact amount of force required to create an optimal artificial circulation in humans is still a matter of discussion. Force needed for compressions labelled as adequate vary from manikin to manikin, and in humans, from 20-70 kg for adults. Careful suggestions that force and depth should be individualized have been expressed, but may need to remain in the realm of advanced skills. Perkins et al. described the force needed when working with the CPREzy as a range from child to extra large adult as 23 to 54 kg. Timerman et al. working with a novel chest compression device, reported that they used 51 ± 20 kg of peak force in their population. Experimental determination of the force to compression relationship with the CPREzy on a rigid surface demonstrated agreement with the numbers listed above. Doubts have been expressed in the past about whether such force indicators retain their meaning when the CPREzy is used on a flexible or compressible support, such as the human chest. Our experience confirms this accuracy, while recognizing that the amount of physical work on a multiple layer support exceeds that of compression on a rigid surface. Clinically, this allows the caregiver performing chest compressions to recognize that, should the patient be lying in a hospital bed, the total distance their hands move may be more than 4-5 cm, and he may, initially, need to do extra work to achieve correct depth, even on modern hospital beds, as this position is often not the position used for training.

The Resusci Anne uses its SkillReporter lights to specify incorrect hand position. Technically, both an actual incorrect position of the hands, as well as applying force in a non vertical direction while the hands are correctly placed, will be scored as such. During our study we used the written record which does not specify the location/direction of the error. Earlier investigation found a propensity for too low a position of the CPREzy; our study design, which relied on the written record, does not allow us to confirm or repudiate this aspect in chest compressions.

Our study has a number of limitations. We did not use a cross over design as discussed above, including large groups of candidates at random, allowing for variables such as motivation,
physiognomy, and skills to correct themselves in the sampling. As those including the candidates were unaware of which task their candidate would perform next, any bias should be limited. Human evaluation of the written record was also used: while the blinded investigators, dedicated resuscitation officers, have extensive skills and practices in evaluating the records, and the benchmarking did not demonstrate relevant differences, more checks during scoring may have increased security as to the value of the scores. However, a post hoc analysis of the database, using the investigator as a variable, could not demonstrate any systematic differences.

While the device increases the possibility for the caregiver to choose a force and judge the consistency of their compressions, it does not allow insight into the force which might be optimal for that patient. In manikin studies this effect is difficult to simulate and the manikin may not be related to actual conditions. Our study also used manikins, and relied on the written record as an accurate representation of reality. However, even the CPREzy does not allow for more accuracy than increments of 3-9 kg of force. Actual force required, or even optimal impression depths in humans are still subject to debate and may be greater than currently thought (i.e., 70 kg or more needed).

In order to optimally evaluate compression skills we de-emphasized ventilation. We used the compression-ventilation ratios for one-caregiver CPR, but supplied a nonobstructive caregiver to perform these ventilations. While introducing interruptions for the ventilations, our protocol did not require the movement of hands to open the airway and the additional fatigue caused by mouth-to-mouth ventilation was lost to evaluation. This did allow for a large number of compressions to be evaluated in a brief period of time.

### 12.5 Conclusions

Our study demonstrates strong improvement in achieving and maintaining adequate depth during chest compressions when a feedback device, the CPREzy, is used. This difference was found even though the caregivers were not specifically trained in its use. Although it requires more work, the variation in depth is significantly smaller than without the device, regardless of physiognomy of the caregiver. It also shows that this device de-emphasises the interval after training without compromising quality. The improvements in efficacy should be an important factor in optimization of the ‘chain of survival’.

### 12.6 Acknowledgements

The authors would like to thank F. Jansen and B. Tax for their technical assistance, and Dr. J. Giele for her help with the statistics.
12.7 References

Work and force in CPR: a practical discussion

Does use of the CPREzy™ involve more work than CPR without feedback?

Abstract

Feedback during CPR may facilitate quality in chest compressions, but has also been associated with caregiver complaints such as stiff wrists, the need for more force and increased fatigue. This concern about extra work is, when using the CPREzy™ with its own spring-loaded surface, particularly relevant in the face of an increased number of successive compressions. This manuscript evaluates the objective workloads for caregivers with and without the CPREzy.

An air pressure driven, piston device was used to generate controlled compressions in a manikin model. The pressure was applied for chest compressions with each of the following: the cylindrical end of the piston, a wooden block as dummy for the CPREzy, and the CPREzy itself. Three manikins with subjectively different spring compliances were selected for the tests. Series of 20 compressions were performed over a wide range of pressures.

No additional force is required to achieve a given depth of compression with or without the CPREzy. However, some additional work is required, ranging from 21 to 26.5%. This work is caused by the longer compression distance associated with the need to compress two springs (e.g., the CPREzy and the chest wall) instead of one (e.g., the chest wall).

The subjective feeling of increased rescuer fatigue with the CPREzy can, at least in part, be attributed to the extra work required for compressing the spring of the CPREzy. Improved accuracy in chest compression depth is likely to be another, more significant, factor in rescuer fatigue.
Feedback during CPR may facilitate quality in chest compressions, but has also been associated with caregiver complaints such as stiff wrists, the need for more force and increased fatigue. This concern about extra work is, when using the CPREzy™ with its own spring-loaded surface, particularly relevant in the face of an increased number of successive compressions. This manuscript evaluates the objective workloads for caregivers with and without the CPREzy.

An air pressure driven, piston device was used to generate controlled compressions in a manikin model. The pressure was applied for chest compressions with each of the following: the cylindrical end of the piston, a wooden block as dummy for the CPREzy, and the CPREzy itself. Three manikins with subjectively different spring compliances were selected for the tests. Series of 20 compressions were performed over a wide range of pressures.

No additional force is required to achieve a given depth of compression with or without the CPREzy. However, some additional work is required, ranging from 21 to 26.5%. This work is caused by the longer compression distance associated with the need to compress two springs (e.g., the CPREzy and the chest wall) instead of one (e.g., the chest wall).

The subjective feeling of increased rescuer fatigue with the CPREzy can, at least in part, be attributed to the extra work required for compressing the spring of the CPREzy. Improved accuracy in chest compression depth is likely to be another, more significant, factor in rescuer fatigue.
13.1 Introduction

The quality of chest compressions, particularly compression depth,\(^1\) is a central issue in cardiopulmonary resuscitation. Studies\(^2,3,4\) have suggested that real-time feedback may be essential in maintaining adequate depth,\(^5\) and has led to the development of voice prompts,\(^6\) analysis of electrical impedance,\(^7\) accelerometers\(^8\) as well as of force recognition feedback devices.\(^9\) The CPREzy\(^{10}\) <www.cprezy.com, Health Affairs, London, GB> correlates force to the desired 4-5 cm compression depth and patient weight. It contains a metronome and five lights for different patient weights which are consecutively lit as appropriate pressure is applied. In addition to the direct feedback, the CPREzy also seems to have psychological value for the caregivers.\(^9\)

During clinical resuscitation attempts in our hospital, rescuers commented on the work involved in performing good chest compressions as well as on stiff wrists. Whether the CPREzy requires extra pressure and/or work to reach the same compression depth,\(^9\) has not been addressed quantitatively. We conducted this study in a manikin setting to determine whether this subjective increase in effort is attributable to the improved depth of compressions and/or to the spring-on-spring design in the CPREzy and to clarify the mathematics involved in the concepts of force and work (Figure 1).

13.2 Materials and Methods

A pressure driven piston (Figure 2), using a standard hospital-wall air pressure source and mounted on a self-contained framework was developed and validated for accuracy of force-distance relationships. During preparatory studies, pressures needed for the full range of compression depths, stability, the position of the manikin as well as the area over which the pressure would be applied were determined. Three different CPREzy's (brand new ± 10 hours and ± 90 hours of use) were tested using this bench arrangement to determine if usage over time altered the force-distance relationships (compliance) when on a firm surface. This was done using a medical grade caliper on an immobile surface with the lights as reference. No statistical or relevant differences between the devices could be demonstrated, allowing one CPREzy to be chosen at random for the actual test series.
Chapter 13: Work and force in feedback

13.1 Introduction

The quality of chest compressions, particularly compression depth, is a central issue in cardiopulmonary resuscitation. Studies have suggested that real-time feedback may be essential in maintaining adequate depth, and has led to the development of devices like the CPREzy (www.cprezy.com, Health Affairs, London, GB). This device correlates force to the desired 4-5 cm compression depth and patient weight. It contains a metronome and five lights for different patient weights which are consecutively lit as appropriate pressure is applied. In addition to the direct feedback, the CPREzy also seems to have psychological value for the caregivers.

During clinical resuscitation attempts in our hospital, rescuers commented on the work involved in performing good chest compressions as well as on stiff wrists. Whether the CPREzy requires extra pressure and/or work to reach the same compression depth, has not been addressed quantitatively. We conducted this study in a manikin setting to determine whether this subjective increase in effort is attributable to the improved depth of compressions and/or to the spring-on-spring design in the CPREzy and to clarify the mathematics involved in the concepts of force and work (Figure 1).

![Figure 1: A schematic representation of the pair of springs as used in the bench tests and during clinical use. The "d" and "D" represent stiffness (1/compliance) of the springs, "F" the loading performed and the "x's" the resulting spring compressions. These are related to the work "W" by equation 1: W1 = (F • x1) ÷ 2 and equation 2: W2 = [(F • x2) ÷ 2 + (F • x1) ÷ 2] = (F ÷ 2) (x1 + x2) = (F • x1 ÷ 2) [1+ (d ÷ D)]. For the detailed mathematical derivations see Reference 9, Appendix 2.]

13.2 Materials and Methods

A pressure driven piston (Figure 2), using a standard hospital-wall air pressure source and mounted on a self-contained framework was developed and validated for accuracy of force-distance relationships. During preparatory studies, pressures needed for the full range of compression depths, stability, the position of the manikin as well as the area over which the pressure would be applied were determined. Three different CPREzy’s (brand new ± 10 hours and ± 90 hours of use) were tested using this bench arrangement to determine if usage over time altered the force-distance relationships (compliance) when on a firm surface. This was done using a medical grade caliper on an immobile surface with the lights as reference. No statistical or relevant differences between the devices could be demonstrated, allowing one CPREzy to be chosen at random for the actual test series.

Three different manikins, a Laerdal Simulator 4000™ and two Laerdal Skillmeter Annes™ (Laerdal Medical, Stavanger, N), were selected based on their subjectively different chest compression characteristics. The manikins were connected to a laptop computer using the PC Skillmeter (Laerdal Medical, Stavanger, N). Force was applied by each of three techniques: either the 45 mm diameter inflexible ring on the piston of the compression device; a wooden block under the piston identical in area to the CPREzy as control, or the CPREzy itself between the piston and the manikin’s sternum. A series of 20 compressions were averaged for mean compression depth per pressure setting. The settings were changed after all measurements with all devices were completed in any one series.

All data were entered into an SPSS v14 database (SPSS Inc., Chicago, IL, USA). The one-way ANOVA (repeated measures) was used to compare mean depths. The independent measures t-test was used to evaluate the workloads with and without the CPREzy.

13.3 Results

The pilot study demonstrated that there is no change in usage-based compliance, from 20-45 PSI, for the CPREzy (p = 0.39 – 0.95). This pressure range includes the full recommended compression depths in our manikins. Pressures from 20 to 50 PSI (138 – 345 kPa) were chosen to include a range from 25-55 mm effective compression depth. Results, expressed in terms of mean compression depths as a function of pressure applied, for the three different compression techniques, are displayed in Figure 3. When comparing these depths for any given pressure, no significant differences were found between the piston, CPREzy or the placebo wooden block.

![Figure 3: Graph showing the mean compression depths as a function of pressure applied. The graph compares piston only, wooden block, and CPREzy. There is no significant difference between the techniques at any given pressure.]

Chapter 13: Work and force in feedback
Figure 3: Depth of compression (means, n = 20 per set) as measured by the actual movement of the manikin’s chest plate in the arrangement depicted in Figure 2, reached with each preset pressure. Data is from all three manikins. Note that there are no significant differences between the use of the piston, the block or the CPREzy (range of p = 0.35 – 0.71). Error bars represent one SEM. The range of compression distances was chosen to extend well beyond the 4-5 cm clinical range.

More work is required when using the feedback device: this in direct proportion to the extra compression distance moved (Table 1). This effect varies between the 21% and 26.5% of the total work required (Figure 4), with the largest difference in the lower range, at less than the clinically relevant compression depths. The difference between the workloads is statistically significant at each force (p = 0.001, overall). There are consistent, small, nonsignificant, differences between the compression characteristics of the CPREzy when on a firm surface as opposed to when lying in the sternum of the manikin. The total work, in Joules, required for optimal quality compressions in our manikin model, are 45 to 82 Joules performed without the CPREzy (Figure 4). This range of work is similar to that which caregivers would perform, by lifting a container with 3.75 litres of water from the ground to their lips, for each single compression.

While not a principal endpoint, the subjective differences in the compression characteristics of the manikins turned out to be small. At similar compression pressure and technique, very similar depths to those by Beckers et al.8 suggest that training with the device allows the students to understand sternal compression depths of 4-5 cm even though the hands move a greater distance.13

Table 1: Compression effects of pressure on the manikin alone (x1), and on the CPREzy (x3), when lying, alone, on a firm surface. Results expressed in terms of depth (mm). When the pressure is applied on the CPREzy (x3) as it lies on the manikin (x1), the compression effect that each pressure has on the manikin and the CPREzy is shown as well as the total travel of the rescuers hands. These are related by the equation W2 = (F ÷ 2) • (x1 + x2). The extra work required by the use of the CPREzy is listed for each series. Note that the only purpose of the subscript ‘s’ (eq. x1s) is to signify other conditions.

<table>
<thead>
<tr>
<th>Compression effect of</th>
<th>PSI 20</th>
<th>21</th>
<th>24</th>
<th>25</th>
<th>30</th>
<th>35</th>
<th>37</th>
<th>39</th>
<th>40</th>
<th>45</th>
<th>50</th>
</tr>
</thead>
<tbody>
<tr>
<td>Manikin only (mm, x1)</td>
<td>24.2</td>
<td>24.6</td>
<td>26.3</td>
<td>25</td>
<td>31</td>
<td>35</td>
<td>40.5</td>
<td>45</td>
<td>46.5</td>
<td>48.5</td>
<td>53</td>
</tr>
<tr>
<td>CPREzy only on firm surface (mm, x2)</td>
<td>8</td>
<td>9</td>
<td>10</td>
<td>11</td>
<td>11</td>
<td>12.5</td>
<td>13</td>
<td>13.5</td>
<td>14</td>
<td>14.5</td>
<td>15</td>
</tr>
<tr>
<td>Manikin (compressed via CPREzy, mm, x1s)</td>
<td>25</td>
<td>25</td>
<td>29</td>
<td>30</td>
<td>36</td>
<td>44</td>
<td>46</td>
<td>49</td>
<td>50</td>
<td>54</td>
<td>55</td>
</tr>
<tr>
<td>CPREzy (when lying on manikin mm, x2s)</td>
<td>8</td>
<td>9</td>
<td>10</td>
<td>10</td>
<td>12</td>
<td>13.5</td>
<td>13.8</td>
<td>14</td>
<td>15</td>
<td>15</td>
<td>16</td>
</tr>
<tr>
<td>Total travel (both in series, x1 + x2, mm)</td>
<td>33</td>
<td>34</td>
<td>39</td>
<td>40</td>
<td>48</td>
<td>57.5</td>
<td>59.8</td>
<td>63</td>
<td>63</td>
<td>69</td>
<td>71</td>
</tr>
<tr>
<td>% extra work</td>
<td>24.2</td>
<td>26.5</td>
<td>28.6</td>
<td>25</td>
<td>33</td>
<td>37.5</td>
<td>27.8</td>
<td>22.2</td>
<td>23.1</td>
<td>21.7</td>
<td>22.5</td>
</tr>
</tbody>
</table>
Table 1: The compression distances moved (mm, x1) are related to the total travel of the rescuer’s hands. These are compression distances was chosen to extend well beyond the 4-5 cm clinical range. The work needed for the desired depth of compression, as well as the work of performing a series of 30 compressions instead of a series of 15, has raised concerns about the physical demands made on the caregiver. This study addresses this issue in evaluating a possible side effect of a feedback device. Our results suggest that, while the amount of force required to perform good quality chest compressions is not altered by the CPREzy, some additional work is required since the distance the hands travel is increased. As a secondary result, we found that there is no decay in the quality of the device itself, even when used intensively.

The CPREzy strongly promoted the quality of compressions when performed, even well after training, in a diverse, large population of hospital staff, who had been trained both with and without the feedback device. The participants were able to perform the workload asked of them for more than 240 seconds, far longer than the two minute change cycle currently recommended. Although Table 1 demonstrates a difference in compression travel with or without the CPREzy, both our results and those by Beckers et al. suggest that training with the device allows the students to understand sternal compression depths of 4-5 cm even though the hands move a greater distance.

The work needed to reach a desired compression depth is increased in comparison with an accelerometer. This as a function of the extra travel by the hands to reach the desired sternal compression depth. Confusion may have been caused by the use of force or pressure: where none extra is required. As Figure 1 and its caption indicate, the extra work done if the CPREzy lies on a firm surface for each force-distance relationship. Work (Joules)

<table>
<thead>
<tr>
<th>Pressure (PSI)</th>
<th>CPREzy-only (Wc)</th>
<th>Manikin only (Wm)</th>
<th>CPREzy + manikin (Wt)</th>
</tr>
</thead>
<tbody>
<tr>
<td>20</td>
<td>20</td>
<td>21</td>
<td>21</td>
</tr>
<tr>
<td>21</td>
<td>24</td>
<td>24</td>
<td>24</td>
</tr>
<tr>
<td>25</td>
<td>30</td>
<td>30</td>
<td>30</td>
</tr>
<tr>
<td>30</td>
<td>35</td>
<td>35</td>
<td>35</td>
</tr>
<tr>
<td>35</td>
<td>37</td>
<td>37</td>
<td>37</td>
</tr>
<tr>
<td>37</td>
<td>39</td>
<td>39</td>
<td>39</td>
</tr>
<tr>
<td>39</td>
<td>40</td>
<td>40</td>
<td>40</td>
</tr>
<tr>
<td>40</td>
<td>45</td>
<td>45</td>
<td>45</td>
</tr>
<tr>
<td>45</td>
<td>50</td>
<td>50</td>
<td>50</td>
</tr>
</tbody>
</table>

Figure 4: Relative amounts of work being done. CPREzy-only (Wc, first column, defined by eq. 1 in Figure 1) is the amount of work being done if the CPREzy lies on a firm surface for each force-distance relationship. Manikin only (Wm, also defined by eq. 1) is the amount of work being done for each force-distance relationship without a second spring (i.e., the non-compressible wooden block or a rescuer’s hands) on the manikin’s sternum. CPREzy + manikin (Wt, defined by eq. 2 in Figure 1) is the work required to compress both CPREzy and manikin at each force-depth point. Note that the extra work is directly related to the increased total compression depth. Area of compression is constant, p = 0.001 overall. Note that the CPREzy approximates the nonlinear force depth relationship as described by Tomlinson et al.
performing chest compressions, and detract from confidence in the CPREzy. The confusion in units may also have, incorrectly, suggested that rescuers with low body weights might be inferior in performing good chest compressions based on that fact alone.

The subjective feelings of rescuer work cannot be defined, as this was a mechanical-compression oriented study. However, the circa 25% added work of compression, as shown by the two studies listed above, seems less significant than the important improvements in compression quality. Further work, involving oxygen consumption testing, may clarify this.\textsuperscript{14}

This study was performed on manikins. One potentially important concern is that the one-piece manikin chest plates, incorporating a centrally located spring system, may only partially simulate the flexibility of the human chest (e.g., movement of parts of the sternum, the sternocostal junctions and the ribs), cloaking somewhat the relationship between the small area of the circular piston in relationship to the large surface area of the CPREzy.\textsuperscript{15}

13.5 Conclusions

The CPREzy\textsuperscript{TM}, with its novel mechanical technique and retaining its force-to-depth relationship even after intensive use, does not require extra force in the range needed for chest compressions in humans, though an extra effort (work of 21\%–26.5\%) must be made by the caregiver. In absolute terms of Joules, the total work remains well within the manageable range for a lay- or professional rescuer. The subjective feeling of increased rescuer fatigue with the CPREzy can, at least in part, be attributed to the extra work of compressing a second spring. Improved compression depth is likely to be another, more significant, factor in rescuer fatigue.
13.5 Conclusions

The CPREzy™, with its novel mechanical technique and retaining its force-to-depth relationship even after intensive use, does not require extra force in the range needed for chest compressions in humans, though an extra effort (work of 21%-26.5%) must be made by the caregiver. In absolute terms of Joules, the total work remains well within the manageable range for a lay- or professional rescuer. The subjective feeling of increased rescuer fatigue with the CPREzy can, at least in part, be attributed to the extra work of compressing a second spring. Improved compression depth is likely to be another, more significant, factor in rescuer fatigue.

13.6 References


The effects of mattresses and backboards on in-hospital CPR

The impact of compliant surfaces on in-hospital chest compressions: Effects of common mattresses and a backboard.

The effects of mattresses and backboards on in-hospital CPR

The impact of compliant surfaces on in-hospital chest compressions: Effects of common mattresses and a backboard.

Abstract

To evaluate, in a hospital setting on different, common mattresses, with and without a backboard, chest movement during CPR.

Sixty CPR sessions (140 seconds each, 30:2, compression-relaxation ratio 1:1) were performed using a manikin on standard hospital mattresses, with or without a backboard in combination with variable weights. Sternum-to-spine compression distance was controlled (range 30 to 60 mm) allowing evaluation of the underlying compliant surface on total hand travel.

Movement of the caregiver’s hands was significantly larger (up to 111 mm at 50 mm compression depth, p< 0.0001) when sternum-to-spine compressions were performed without a backboard than with one. The extent of this variable extra travel effect depended on the type of mattress as well as the force of compression. Foam mattresses and air chamber systems act as springs and follow hand movement, while ‘slow’ foam mattresses incorporate time delays, making depth and force sensing harder. A backboard decreases the extra hand movement due to mattress effects by more than 50%, strongly reducing caregiver work.

Total vertical hand movement is significantly and clinically relevantly much larger than sternum-to-spine compression depth when CPR is performed on a mattress. Additional movement depends on the type of mattress and can be strongly reduced, but not eliminated, when a backboard is applied. The additional motion and increased work load adds extra complexity to in-hospital CPR. We propose that this should be taken into account during training by in-hospital caregivers.
Table of contents

14. THE EFFECTS OF MATTRESSES AND BACKBOARDS ON IN-HOSPITAL CPR ...... 257
14.1 Introduction ............................................................................................................................. 260
14.2 Materials and methods............................................................................................................. 260
14.3 Results..................................................................................................................................... 262
14.4 Discussion ............................................................................................................................... 267
14.5 Conclusions ............................................................................................................................. 269
14.6 Acknowledgements ................................................................................................................. 270
14.7 References ............................................................................................................................... 271
14.1 Introduction

Success following in-hospital resuscitations (CPR) is poor.\(^1,2\) Many factors contribute to this, including comorbidity, lower incidence of observed arrests, delay in basic and advanced life support, as well as poor quality of CPR itself.\(^3,4,5\) Quality emphasizes the need to perform deep compressions, but many recent studies underscore continuing difficulty in the performance and understanding of this skill both out-of-hospital and in-hospital.\(^6,7,8\) Further, mattress properties may effect this process since patients remain on a stretcher or in bed during transportation and in-hospital resuscitation.\(^9,10\)

Hospitals, challenged to prevent bed sores, have introduced different pressure relieving support systems, in particular for patients confined to bed, starting with simple foam mattresses. Increasingly so called ‘slow’ foam or ‘hybrid’ foam-based constant low-pressure’ mattresses are used as the baseline mattress.\(^11,12\) These systems incorporate multiple layers of material with different viscoelastic properties so that weight is distributed equally. Air-filled systems have been replaced by this technology.\(^13\)

Studies show that patients most at risk for in-hospital arrest are typically found on pressure distribution systems.\(^13\) Surprisingly, initial attention for the relationship between mattresses and CPR quality suggested little negative impact on compression quality attributable to the non-firm surfaces.\(^1,14,15,16\) Two of these studies also suggested that a backboard did not contribute to the effectiveness of chest compressions while introducing a time delay.\(^14,15\) One paper commented that firm surfaces may be a cause of additional injuries.\(^9\) All these studies focussed on CPR quality with expert caregivers intuitively adapting compressions, not on quantifying the effects of the compliant surface on vertical hand movement (i.e., the sum of sternum-to-spine and mattress compressions). Most recently, this intuitive effect was objectified using feedback devices on compliant surfaces, demonstrating a 35-40% overestimation of sternum-to-spine movement and overly shallow compressions regardless of training.\(^17\)

This study evaluates the effect of typical compliant surfaces on total vertical hand movement by using controlled sternum-to-spine compressions (range 30–60 mm) in a manikin model. This approach offers data about distances, forces and work involved when using different mattresses, with or without a backboard and with or without additional weights.

14.2 Materials and methods

Mattresses on which our in-hospital resuscitations frequently occur were identified. (Table 1) A brief telephone questionnaire with 10 similar and university hospitals in Europe and the United States suggested that these three types of mattresses are representative for those in general use. Hospital steel bed frames are rigid, with a stiffness of 2500 N/cm. The backboard, in our case part of the foot end frame of the bed, is semi-rigid synthetic Trespa\(^\text{TM}\) (Trespa International, Weert, NL). The width and height are 80 and 30 cm, respectively. It has a stiffness of 200 N/cm. This stiffness is typical of backboards and CPR boards in general use. The backboard is placed crosswise on top of the mattress, underneath the patient from the upper edge of the shoulders (approximately at the 2\(^\text{nd}\) thoracic vertebra) supporting the patient’s torso to the top of the lumbar spine. There is no direct contact between the backboard and the bed frame.
Table 1: Details of the mattresses involved in the study. All of these mattresses are used in regular ward settings. No specific outside markers make recognition easy, although the use of the mattress is included in the electronic nursing and medical files.

<table>
<thead>
<tr>
<th>Mattress type</th>
<th>Code</th>
<th>Location / use</th>
<th>Dimensions (cm)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cliniplot III</td>
<td>A</td>
<td>Selected high risk groups (ward)</td>
<td>192 x 85 x 16</td>
<td>Simple foam mattress</td>
</tr>
<tr>
<td>(Hill Rom)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Meditherm (Medibol, Valkenswaard)</td>
<td>B</td>
<td>Default on all hospital beds</td>
<td>198 x 84 x 14</td>
<td>Two-layer construction, with different hardness i.e., ± 70 N/cm upper and 105 N/cm lower, and 40% of total mattress depth.</td>
</tr>
<tr>
<td>Atmos Air 9000</td>
<td>C</td>
<td>Selected high risk group (ward)</td>
<td>202 x 82 x 18</td>
<td>Includes centrally located air mattress: 145 x 70 x 12</td>
</tr>
<tr>
<td>(Hill Rom)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EMS stretcher</td>
<td>EMS</td>
<td>Pre-hospital / Emergency Room</td>
<td>Foldable</td>
<td>Schnitzler 404020404 Ferno</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Mattress thickness ± 5cm</td>
<td></td>
</tr>
</tbody>
</table>

While previously standard training manikins were used, our experiments were performed using an especially prepared version. It includes a linear potentiometer (Type S13FLP100A, Sakae Tsushin Kogyo Co., Japan) to measure movement of the anterior chest wall relative to the back (sternum-to-spine distance), replacing the less accurate training systems of the SkillReporter™ or VAM™. A linear voltage differential transformer (LVDT, Type SM100, WayCon, Taufkirchen, DE.) was attached to the manikin’s rigid back plate and to the bed frame as its reference. This LVDT measures movement of the manikin into the mattress, movement not detected by the potentiometer. This system has a 1 mm cumulative measurement error as listed for the potentiometer <http://sakae-tsushin.co.jp/eng_page/pdf/pot/e_13FLP.pdf> and the LVDT <http://www.waycon.de/fileadmin/pdf/Inductive_Transducer_SM.pdf>. The spring system simulating the elasticity of the manikin’s chest wall was the standard Laerdal Skillrecorder Resusci Anne system (Laerdal, Stavanger, NO).

Sixty sessions of CPR (140 seconds duration each) were performed on the three mattresses and an EMS stretcher. Experiments were performed per target compression depth without a backboard, then with a backboard, followed by the weight series. After each series (i.e., when the new mattress was placed on the bed frame), equipment baseline stability was revalidated.

Chest compressions were done according to the one-rescuer 2005 ILCOR Guidelines, with about 140 seconds (6 cycles) of 30:2 compressions to ventilation motions. The ventilation pause was standardized to five seconds of no-compression time. Compression depths (from 30 to 60 mm sternum-to-spine impression representing clinically relevant depths) were chosen in random order. For a session to be used for analysis, all of the following criteria had to be satisfied: quality (depth ± 2 mm) met during at least 90% of the 140 seconds compressions, a compression frequency of 95-105 cpm, a ± 20% range of the 1:1 compression-relaxation ratio, complete relaxation of the chest wall between compressions, and a stable measurement base. The 30:2 compression to ventilation rhythm was used in order to evaluate whether the mattresses, and in particular the ‘slow’ foam mattress (B), ‘recuperate’ to their neutral position, possibly affecting compressions after the ventilations.

When the backboard was used, weights (20 or 40 kg) could be added. This theoretically corrected for patient weight up to 110 kg assuming circa 35% of that weight is the torso. Weights were not placed elsewhere since the mattress characteristics should negate any impact on our measurements. All sessions were performed with the bed at 60 cm above the floor, and the caregiver standing next to it. The ambient temperature was standardized at 20°C, since the ‘slow’ foam mattresses can change characteristics under very cool or hot conditions.
Data was collected and stored in real time using a laptop computer. Data collection started after steady state was reached (see Figures 1 and 2). Potentiometer feedback from the laptop screen was provided so that manual compressions could be reproducibly maintained at the required depth. Post hoc, we generated cumulative depth of compression (the total impression depth or vertical hand movement), LVDT and potentiometer graphs, as well as depth-time relationship figures. Data are presented as means ± standard deviations. Under SPSS, v16, the student’s t-test was used to test for significance between single measurements, while repeated ANCOVA with Bonferroni’s correction was used for multiple analyses. P < 0.05 was taken to be significant.

14.3 Results

At chest compression depths (i.e., the sternum-to-spine) of 30–60 mm, consistent, large extra movements by the manikin into the mattresses were detected. All were both clinically and statistically significant when compared to the same compression on a firm surface (p = 0.0001). The total movement was also clinically and statistically different when compared with the intended sternum-to-spine depth on each specific mattress (p = 0.0001).

Chest compressions without ventilatory pauses on mattress B is shown in Figure 1, continuing until steady state was reached. Both sternum-to-spine movement and impression of the manikin into the mattress are displayed. At 40 mm target sternum-to-spine compression, this ‘slow’ foam mattress requires 34 seconds to reach steady state. Examples of the sensor data of total and sternum-to-spine movement sessions using the 30:2 cycle on mattress B are shown in Figure 2a and 2b. The time to steady state when 60 mm is the target (the LVDT measurement in Figure 2b) is approximately 6 cycles or 138 seconds, reflecting both rebound of the mattress during ventilation pauses and the reaction of the mattress to that force. Total vertical hand movement is shown in Figures 3a-d and in Table 2 for the three mattresses as well as an EMS stretcher, under different CPR conditions (i.e., target depth, backboard, weights).
Data was collected and stored in real time using a laptop computer. Data collection started after steady state was reached (see Figures 1 and 2). Potentiometer feedback from the laptop screen was provided so that manual compressions could be reproducibly maintained at the required depth. Post hoc, we generated cumulative depth of compression (the total impression depth or vertical hand movement), LVDT and potentiometer graphs, as well as depth-time relationship figures. Data are presented as means ± standard deviations. Under SPSS, v16, the student’s t-test was used to test for significance between single measurements, while repeated ANCOVA with Bonferroni’s correction was used for multiple analyses. P < 0.05 was taken to be significant.

14.3 Results

At chest compression depths (i.e., the sternum-to-spine) of 30–60 mm, consistent, large extra movements by the manikin into the mattresses were detected. All were both clinically and statistically significant when compared to the same compression on a firm surface (p = 0.0001). The total movement was also clinically and statistically different when compared with the intended sternum-to-spine depth on each specific mattress (p = 0.0001).

Chest compressions without ventilatory pauses on mattress B is shown in Figure 1, continuing until steady state was reached. Both sternum-to-spine movement and impression of the manikin into the mattress are displayed. At 40 mm target sternum-to-spine compression, this ‘slow’ foam mattress requires 34 seconds to reach steady state. Examples of the sensor data of total and sternum-to-spine movement sessions using the 30:2 cycle on mattress B are shown in Figure 2a and 2b. The time to steady state when 60 mm is the target (the LVDT measurement in Figure 2b) is approximately 6 cycles or 138 seconds, reflecting both rebound of the mattress during ventilation pauses and the reaction of the mattress to that force. Total vertical hand movement is shown in Figures 3a-d and in Table 2 for the three mattresses as well as an EMS stretcher, under different CPR conditions (i.e., target depth, backboard, weights).

Figure 2a: Example of a 30:2 cycle on a ‘slow’ foam mattress. Middle section of a 30:2 resuscitation session (with as goal: 60 mm sternum-to-spine depth). Abbreviations used in the figure are: VP = ventilation pause, R = retention of depth of the patient into the mattress. Conditions are 60 (58) mm actual sternum-to-spine compression, mattress B, no backboard, 30:2 compressions to ventilation pause (5 seconds), with 100 cpm and caregiver next to the bed. Two curves are shown in the 88-120 second section of the session: The broken (lower) curve is the potentiometer (sternum-to-spine compression depth), the fully drawn line is the summation of potentiometer and LVDT (movement of the manikin into the mattress). Note the slow rebound effects of the mattress (upper line, below the R) during the 5 second ventilation pause. Line R demonstrates that the patient rebounds to 22 mm depth (from the unloaded top of the mattress) between compressions.
Figure 2b: Demonstration of time to steady state on 'slow' foam mattress B. Graphic display of the data in Figure 2a. Note that the sternum of the manikin moved some 110 to 120 mm downwards during this experiment. When compared with Figure 1, the effect of ventilatory pauses and increased sternum-to-spine depth is shown. One 30:2 cycle is 23 seconds in length.

Differences between target sternum-to-spine depth and actual vertical hand movement were found to vary from 20% to up to 120%, (figure 3a-d) and alter caregiver work significantly. This is also true (>30% added travel) for compressions on the thin (5 cm) EMS stretcher mattress, despite stiffness offered by its transport position. While the use of a backboard reduces the excess motion by more than 50%, the impression into the mattress is not eliminated. There is a strong, consistent, statistically significant effect between any mattress and compression depth with versus without the backboard, and thus on caregiver work (p = 0.0001, Table 2). The effect of adding additional weight to the backboard was different in mattresses A and B (increase in total depth) compared with mattress C (decrease in total depth). This anomalous behaviour (Figure 3) was systematic.
Figure 2b: Demonstration of time to steady state on ‘slow’ foam mattress B. Graphic display of the data in Figure 2a. Note that the sternum of the manikin moved some 110 to 120 mm downwards during this experiment. When compared with Figure 1, the effect of ventilatory pauses and increased sternum-to-spine depth is shown. One 30:2 cycle is 23 seconds in length. Differences between target sternum-to-spine depth and actual vertical hand movement were found to vary from 20% to up to 120%, (figure 3a-d) and alter caregiver work significantly. This is also true (>30% added travel) for compressions on the thin (5 cm) EMS stretcher mattress, despite stiffness offered by its transport position. While the use of a backboard reduces the excess motion by more than 50%, the impression into the mattress is not eliminated. There is a strong, consistent, statistically significant effect between any mattress and compression depth with versus without the backboard, and thus on caregiver work (p = 0.0001, Table 2). The effect of adding additional weight to the backboard was different in mattresses A and B (increase in total depth) compared with mattress C (decrease in total depth). This anomalous behaviour (Figure 3) was systematic.

Figure 3a: Summary of data with 30 mm sternum-to-spine compressions. Without a backboard mattress A allows 42 mm of additional travel (140% of target sternum-to-spine, total hand movement is 72 mm), while this is reduced to 9 mm with the backboard. Similar changes, of a smaller order of magnitude (32 mm and 7 mm) are demonstrated for mattress B. Plots of actual hand travel under controlled sternum-to-spine compression depth of 30-60 mm. Abbreviations: A = foam mattress; B = ‘slow’ foam; C = combined system (see Table 1 for details). nbb = no backboard; bb = backboard; bb+20 and bb+40 are series with backboard with respectively 20 and 40 kg of added weight distributed over the board. Cycle indicates the cycle 30:2 of basic life support. Statistics are not shown for clarity in the figures. (see Table 2)

Figure 3b: Summary of data with 40 mm sternum-to-spine compressions. Mattress C shows relatively little travel (66 mm) in comparison with mattress A (94 mm) and mattress B (83 mm). However, the impact of the backboard is relatively small on mattress C, attributable to the air-filled section and limited elastic foam. Note that with a backboard the total travel is 8-13 mm on mattresses A and B.
Chapter 14: The effects of mattresses and backboards on in-hospital CPR

Figure 3c: Summary of data with 50 mm sternum-to-spine compressions. In this Figure, the different degrees of stiffness, and their effects are demonstrated. The added weight of 40 kg makes mattress C stiff (13 mm added travel), while this effect is not seen in mattresses A and B.

Figure 3d: Summary of data with 60 mm sternum-to-spine compressions. This Figure also demonstrates that a pure foam mattress (A, of 16 cm thickness) is only slightly sensitive to increasing stiffness due to compressions.

Table 2: Compression data and total hand travel data under different compression targets and conditions. Abbreviations used in the table are nbb = no backboard; bb = backboard; w = weight added (20 and 40 kg are shown); n.a. = data not available (experimental series not performed or insufficient to report. This is a ‘slow’ foam mattress on a “backboard” used to immobilize a (trauma) patient without a formal backboard, and has been suggested to alleviate bed sores or decubitus dangers while adequately immobilizing a patient during initial screening. Vertical statistics shown per 30-60 mm target is the actual hand travel compared to the sternum-to-spine compression target. In horizontal rows, different p-values between subgroups are labelled with superscripts.

Table 2

<table>
<thead>
<tr>
<th>Conditions</th>
<th>Mattress A</th>
<th>Mattress B</th>
<th>Mattress C</th>
<th>EMS</th>
<th>p between mattresses</th>
</tr>
</thead>
<tbody>
<tr>
<td>30 mm nbb</td>
<td>71 ± 1</td>
<td>61 ± 1.3</td>
<td>54 ± 0.3</td>
<td>40  ± 1.8</td>
<td>p = 0.0001</td>
</tr>
<tr>
<td>30 mm bb</td>
<td>39 ± 0.5</td>
<td>37 ± 0.4</td>
<td>46 ± 0.7</td>
<td>n.a.</td>
<td>p = 0.001</td>
</tr>
<tr>
<td>30 mm bb + 20</td>
<td>41 ± 0.8</td>
<td>43 ± 1</td>
<td>38 ± 0.6</td>
<td>n.a.</td>
<td>p = 0.0001</td>
</tr>
<tr>
<td>30 mm bb + 40</td>
<td>43 ± 0.9</td>
<td>39 ± 0.4</td>
<td>n.a.</td>
<td>n.a.</td>
<td>p = 0.0001, except 0.01*</td>
</tr>
<tr>
<td>40 mm nbb</td>
<td>94 ± 0.6</td>
<td>83 ± 0.9</td>
<td>66 ± 0.5</td>
<td>56 ± 2.1</td>
<td>p = 0.0001</td>
</tr>
<tr>
<td>40 mm bb</td>
<td>53 ± 1.0</td>
<td>49 ± 0.8</td>
<td>57 ± 0.4</td>
<td>n.a.</td>
<td>p = 0.0001</td>
</tr>
<tr>
<td>40 mm bb + 20</td>
<td>54 ± 1.4</td>
<td>56 ± 0.6</td>
<td>51 ± 0.9</td>
<td>53 ± 0.9*</td>
<td>p = 0.0001, except 0.04*</td>
</tr>
<tr>
<td>40 mm bb + 40</td>
<td>55 ± 0.4</td>
<td>50 ± 0.8*</td>
<td>54 ± 0.4</td>
<td>53 ± 0.9*</td>
<td>p = 0.0001, except 0.42*</td>
</tr>
<tr>
<td>50 mm nbb</td>
<td>111 ± 1.1</td>
<td>100 ± 1.5</td>
<td>82 ± 0.7</td>
<td>65 ± 1.3</td>
<td>p = 0.0001</td>
</tr>
<tr>
<td>50 mm bb</td>
<td>66 ± 0.7</td>
<td>62 ± 1.5</td>
<td>71 ± 1.0</td>
<td>n.a.</td>
<td>p = 0.0001</td>
</tr>
<tr>
<td>50 mm bb + 20</td>
<td>70 ± 1.1</td>
<td>71 ± 1.5</td>
<td>66 ± 1.0</td>
<td>68 ± 0.9</td>
<td>p = 0.0001, except 0.04*</td>
</tr>
<tr>
<td>50 mm bb + 40</td>
<td>67 ± 1.4</td>
<td>63 ± 0.8</td>
<td>n.a.</td>
<td>n.a.</td>
<td>p = 0.0001, except 0.42*</td>
</tr>
<tr>
<td>60 mm nbb</td>
<td>121 ± 1.2</td>
<td>115 ± 0.7</td>
<td>91 ± 0.8</td>
<td>87 ± 0.6</td>
<td>p = 0.0001</td>
</tr>
<tr>
<td>60 mm bb</td>
<td>85 ± 0.9#</td>
<td>77 ± 1.0</td>
<td>85 ± 0.9#</td>
<td>n.a.</td>
<td>p = 0.0001, except 1.0#</td>
</tr>
<tr>
<td>66 mm bb + 20</td>
<td>86 ± 0.8##</td>
<td>85 ± 0.8</td>
<td>79 ± 1.6</td>
<td>80 ± 0.8</td>
<td>p = 0.0001, except 0.42##</td>
</tr>
<tr>
<td>60 mm bb + 40</td>
<td>86 ± 0.8##</td>
<td>80 ± 0.8</td>
<td>80 ± 0.9##</td>
<td>74 ± 0.8</td>
<td>p = 0.0001, except 0.42##</td>
</tr>
</tbody>
</table>

Discussion

This study demonstrates that compressions of 50 mm (sternum-to-spine movement) on hospital mattresses may require hand movement of 111 mm. This extra travel is variable, dependent on factors such as target depth, patient weight, type of mattress and the use of a backboard. These findings highlight concerns that the ability to perceive ‘good’ chest compressions may be taught but remain difficult to apply, even on non-compliant surfaces.6,20,21  Our findings impact caregiver abilities to meet the consensus that optimal chest compressions are an essential prerequisite for good outcome.6,7,22

Our findings, and other recent work, strongly suggest that performing good chest compressions on hospital mattresses requires knowledge of the mechanics of mattress and backboard effects (impression target versus vertical hand movement), feedback devices, force and work.20  Additional compression depth will increase the amount of work required for the compressions even though the force required does not change. Work is equal to the integral of force over distance and our data shows that the increase can be as large as 100%.23

Hospital mattresses have complex, sometimes nonlinear, mechanical characteristics. Mattress C behaved most ideally for compressions, with its thin foam layer limiting damping in the air-cushion, and increasing stiffness as compression depth increases. Mattress B is highly viscoelastic, creating strong damping and large nonlinear effects. Mattress A, the classic foam mattress, resembles a simple spring (i.e. linear effects), with stiffness lower than in either B or C. These mattress properties not only impact compression but also the recoil between compressions. The strong viscous properties in mattress B delay return to the neutral position, allowing the
decubitus dangers while adequately immobilizing a patient during initial screening. Vertical statistics shown per 30-60 mm target is the actual hand travel compared to the sternum-to-spine compression target. In horizontal rows, different p-values between subgroups are labelled with superscripts.

### Table 2: Compression data

<table>
<thead>
<tr>
<th>Conditions</th>
<th>Mattress A</th>
<th>Mattress B</th>
<th>Mattress C</th>
<th>EMS</th>
<th>p between mattresses</th>
</tr>
</thead>
<tbody>
<tr>
<td>30 mm nbb</td>
<td>71 ± 1</td>
<td>61 ± 1.3</td>
<td>54 ± 0.3</td>
<td>40 ± 1.8</td>
<td>p = 0.0001</td>
</tr>
<tr>
<td>30 mm bb</td>
<td>39 ± 0.5</td>
<td>37 ± 0.4</td>
<td>46 ± 0.7</td>
<td>n.a.</td>
<td>p = 0.001</td>
</tr>
<tr>
<td>30 mm bb + 20</td>
<td>41 ± 0.8</td>
<td>38 ± 0.6</td>
<td>43 ± 0.9</td>
<td>n.a.</td>
<td>p = 0.0001</td>
</tr>
<tr>
<td>30 mm bb + 40</td>
<td>43 ± 1</td>
<td>40 ± 0.3</td>
<td>39 ± 0.4</td>
<td>n.a.</td>
<td>p = 0.0001</td>
</tr>
<tr>
<td>30 mm nbb</td>
<td>94 ± 0.6</td>
<td>83 ± 0.9</td>
<td>66 ± 0.5</td>
<td>56 ± 2.1</td>
<td>p = 0.0001</td>
</tr>
<tr>
<td>40 mm bb</td>
<td>53 ± 1.0</td>
<td>49 ± 0.8</td>
<td>57 ± 0.4</td>
<td>n.a.</td>
<td>p = 0.0001</td>
</tr>
<tr>
<td>40 mm bb + 20</td>
<td>54 ± 1.4</td>
<td>51 ± 0.9</td>
<td>55 ± 0.4</td>
<td>n.a.</td>
<td>p = 0.0001</td>
</tr>
<tr>
<td>40 mm bb + 40</td>
<td>56 ± 0.6</td>
<td>53 ± 0.9</td>
<td>50 ± 0.8</td>
<td>n.a.</td>
<td>p = 0.0001, except 0.01</td>
</tr>
<tr>
<td>50 mm nbb</td>
<td>111 ± 1.1</td>
<td>100 ± 1.5</td>
<td>82 ± 0.7</td>
<td>65 ± 1.3</td>
<td>p = 0.0001</td>
</tr>
<tr>
<td>50 mm bb</td>
<td>66 ± 0.7</td>
<td>62 ± 1.5</td>
<td>71 ± 1.0</td>
<td>n.a.</td>
<td>p = 0.0001</td>
</tr>
<tr>
<td>50 mm bb + 20</td>
<td>70 ± 1.1</td>
<td>66 ± 1.0</td>
<td>67 ± 1.4</td>
<td>n.a.</td>
<td>p = 0.04, except 0.0001</td>
</tr>
<tr>
<td>50 mm bb + 40</td>
<td>71 ± 1.5*</td>
<td>68 ± 0.95</td>
<td>63 ± 0.8</td>
<td>n.a.</td>
<td>p = 0.0001</td>
</tr>
<tr>
<td>60 mm nbb</td>
<td>121 ± 1.2</td>
<td>115 ± 0.7</td>
<td>91 ± 0.8</td>
<td>87 ± 0.6</td>
<td>p = 0.0001</td>
</tr>
<tr>
<td>60 mm bb</td>
<td>85 ± 0.9*</td>
<td>77 ± 1.0</td>
<td>85 ± 0.9</td>
<td>n.a.</td>
<td>p = 0.0001, except 1.0*</td>
</tr>
<tr>
<td>60 mm bb + 20</td>
<td>86 ± 0.8*</td>
<td>79 ± 1.6</td>
<td>80 ± 0.9*</td>
<td>n.a.</td>
<td>p = 0.0001</td>
</tr>
<tr>
<td>60 mm bb + 40</td>
<td>85 ± 0.8</td>
<td>80 ± 0.8</td>
<td>74 ± 0.8</td>
<td>n.a.</td>
<td>p = 0.42, except 0.0001</td>
</tr>
</tbody>
</table>

### 14.4 Discussion

This study demonstrates that compressions of 50 mm (sternum-to-spine movement) on hospital mattresses may require hand movement of 111 mm. This extra travel in variable, dependent on factors such as target depth, patient weight, type of mattress and the use of a backboard. These findings highlight concerns that the ability to perceive ‘good’ chest compressions may be taught but remain difficult to apply, even on non-compliant surfaces. Our findings impact caregiver abilities to meet the consensus that optimal chest compressions are an essential prerequisite for good outcome. Our findings, and other recent work, strongly suggest that performing good chest compressions on hospital mattresses requires knowledge of the mechanics of mattress and backboard effects (impression target versus vertical hand movement), feedback devices, force and work. Additional compression depth will increase the amount of work required for the compressions even though the force required does not change. Work is equal to the integral of force over distance and our data shows that the increase can be as large as 100%.

Hospital mattresses have complex, sometimes nonlinear, mechanical characteristics. Mattress C behaved most ideally for compressions, with its thin foam layer limiting damping in the air-cushion, and increasing stiffness as compression depth increases. Mattress B is highly viscoelastic, creating strong damping and large nonlinear effects. Mattress A, the classic foam mattress, resembles a simple spring (i.e. linear effects), with stiffness lower than in either B or C. These mattress properties not only impact compression but also the recoil between compressions. The strong viscous properties in mattress B delay return to the neutral position, allowing the
patient to be driven into the mattress, as shown in Figures 1 and 2b. Even the five seconds non-compression time we chose for ventilation, was too short for full recoil (Figure 2a). This introduces a complication for the caregiver, as the distance his hands must move is now history dependent. Total compression depths follow from the mechanical properties described above: mattress A, being the most compliant, has the largest extraneous movement but is also the easiest to understand. Sensitivity to this effect on actual spine-to-sternum target depths is relevant even on thin EMS gurney mattresses. Table 2 shows large (addition of >30% of target depth) total hand travel differences under conditions where the use of a backboard is impossible.

A backboard reduces movement into the mattress by about 50%, with the order of reduction mattress A > B > C. This can be attributed to the backboard roughly doubling the force-transferring contact area, leading to an increased effective stiffness and thus to less mattress compression at the same applied force. The effect is contingent on the size of the backboard in relation to the chest: length outside the force-transferring area of the chest or torso may not contribute as effectively. The effects of weights on the backboard, simulating the weight of the torso pushing into the bed, were small and also different than we expected. Our assumption had been that heavy patients compress the mattress, increasing its density and stiffness, thereby limiting excess motion. The extra weight, however, seems to have little impact on density characteristics, increases inertia of the backboard-manikin combination, by generating extra acceleration into the compliant surface. These are small but recognizable effects on the total compression depth (Table 2, Appendix VI). This limiting effect is most apparent in mattresses A and B, in which stiffness is independent of compression depth and explain earlier findings of similar compression quality on inflated and empty air mattresses.

While, to our knowledge, this is the first paper to approach mattress effects on chest compressions from a fixed sternum-to-spine depth, different authors have explored aspects of this question. Sensitivity to this effect on actual spine-to-sternum target depths is relevant even on thin EMS gurney mattresses. Table 2 shows large (addition of >30% of target depth) total hand travel differences under conditions where the use of a backboard is impossible.

Conclusions

Other studies have focused on efficacy of chest compressions in manikin models, with Tweed et al., testing the effects of foam and air-containing mattresses using a small, expert caregiver group. They found a mean sternum-to-spine depth of 42.5 mm on the floor, but only 49% of compressions being marginally adequate on any other surface. The authors mention, that while the mattresses were used at random, a positive learning curve in quality of compression was recognizable. This suggests that their caregivers intuitively focussed on resistance and work instead of on performing 4-5 cm compression depths. Applying this model to foam and other ICU oriented mattresses, as well as to caregiver body-to-bed position on force, they confirmed their earlier results of caregiver adaptation, as well as the prevalence of shallow compressions on compliant surfaces. These results have been confirmed by others. Work related aspects due to non-firm surfaces has also been investigated using variable bed height and an added backboard. In this study a mean compression depth of ± 30 mm was found overall. The backboard did not improve this overly shallow depth but did required some 11 seconds to place.

Our results expand, but also differ from those described above. Dissatisfied with the quality of compressions, none directly addresses the question of why compressions were as good as they were. In our sample, additional hand travel of 61 mm without a backboard and about 25 mm with one, can be demonstrated. If a caregiver were to adhere to goals of 40-50 mm hand movement, cardiac output would plummet. Output to compression depth relationship, including trained caregivers has been described as challenging. Even feedback-directed sternum-to-spine
compression depth on compliant surfaces must be approached carefully, as pointed out by Perkins et al.\textsuperscript{17} They concisely point out the clinical impact of mattress systems when using accelerometer-based feedback, nicely imitating the difficulty caregivers have if they focus on 40–50 mm compression depths.\textsuperscript{17} This study touches on the heart of in-hospital compression quality and the need to understand both feedback technology\textsuperscript{21,22,23} and limitations caused by the compliant surface. Their findings of 35-40\% under estimation of compression depth adjoin the low end of the range found by Andersen et al.\textsuperscript{10} and those we found. Difference in the experimental setup, measurement techniques, type of backboard and mattress systems explain the numeric differences in hand travel within the results, while both studies are complementary in methodology and general conclusions.

The findings by Tweed et al.,\textsuperscript{14} Perkins et al.,\textsuperscript{15,17} as well as our own, suggest that caregivers may, if offered the opportunity, focus on compression force instead of distance per se. Teaching this to in-hospital caregivers involves moving the manikin from the floor.\textsuperscript{18} Students are taught to recognize displacements of 40-50 millimetres as well as the range of resistance and work needed to reach this goal even if the manikin moves relative to the surface of the bed. Schooling should be on beds comparable to the local situation, carefully monitored and feedback may be considered.\textsuperscript{20} The Guidelines 2005 strongly recommend that compressions be done with adequate depth, frequency and appropriate force on a firm surface.\textsuperscript{18} Since we cannot provide the non-compliant surface, the focus must be shifted to getting the depth right anyway and making the surface as firm as possible.

Our study has a number of limitations. Our preliminary telephone questionnaire demonstrated that mattresses available are strongly varied. Our choice of three distinct mattresses with different elastic and viscoelastic properties was intended to address this, but as local situations may differ,\textsuperscript{17} the characteristics of available mattresses should be understood by resuscitation officers as well as intervention teams. The appendix (VI) offers a mathematical approach to evaluating this limitation. Our study also does not specifically address effects that different patient weights, BMI or differing backboard sizes and shapes might have on hand movement. These effects, based on expectations and our observations with the backboard, might be complex and sensitive to body mass distribution, body shape and even compression technique. This subject deserves further study and perhaps modelling.

Our goal was to investigate the characteristics of the summation of chest and mattress and the impact of a backboard, not the ability of caregivers to perform chest compressions per se. We, therefore, cannot comment on quality, nor on the individual perception experts may use to perform ‘good’ compressions under complex conditions as described earlier, only on the system involved and its potential clinical relevance and to teaching implications.\textsuperscript{14,15} Further study is warranted and may directly improve in-hospital resuscitation outcomes.

### 14.5 Conclusions

Our study shows that hand movement is significantly larger (up to 110\% at 50 mm target sternum-to-spine compression depth) when CPR is performed on a compliant mattress. The amount of extra movement is directly related to the type of mattress and may be confusing during both compression and relaxation phases. The extraneous movement can be reduced by at least 50\%, but not eliminated, when a backboard is applied. The increased travel of the caregiver’s hands and proportionally increased workload are significant risks for unnoticed inappropriately shallow compressions during in-hospital CPR. We propose that these phenomena should be taken
into account by in-hospital caregivers and introduced into training sessions. Each EMS and hospital setting with patients on mattresses should evaluate these effects and ensure familiarity with the impact of these mattresses on quality.

### 14.6 Acknowledgements

The authors would like to thank Ms. Y. Koeken, MSc, Ms. A. Henderson, RN and Mr. H. Schoonen, RN for their technical assistance, willingness to participate, and evaluation of the manuscript.
14.7 References

4 Clement F. Does the time of day and where the event takes place have any influence on survival from in-hospital cardiac arrest. Resuscitation. 2006; 70(2): 304.
9 Price MF, Stevens H. The effect of pressure reducing mattresses in CPR. Nursing Stand. 1998; Dec 9-15: 13(12); 64.
12 Beghe C. Review: foam-based, low-pressure mattresses are better than standard hospital mattresses for reducing pressure ulcers. ACP J Club. 2005 Jan-Feb; 142(1): 8.


The accelerometer as a device in CPR

Accelerometer feedback during chest compressions: characteristics and value or just a must-have device.

Abstract

Feedback to improve quality in chest compressions is gaining acceptance with lay- and professional caregivers. This information about compression depth, rate, and no-compression time may be offered by totally different technologies. Both pressure-sensing and accelerometer-based technologies are in use. Some devices require additional work by the caregiver, while others are sensitive to the properties of the surface that supports the patient. In all cases, a clear understanding by the provider of how the device works is required to avoid inadvertent sub-optimal treatment.

The PocketCPR is the first autonomous (i.e., not incorporated in an AED or monitor) accelerometer based device for use in adult CPR. We demonstrate that it does not require any extra work by the caregiver and we describe this accelerometer technology so that users are aware of its potential and its limitations. If this technology is correctly used, it can make a major contribution to the quality of chest compressions.
Abstract

Feedback to improve quality in chest compressions is gaining acceptance with lay- and professional caregivers. This information about compression depth, rate, and no-compression time may be offered by totally different technologies. Both pressure-sensing and accelerometer-based technologies are in use. Some devices require additional work by the caregiver, while others are sensitive to the properties of the surface that supports the patient. In all cases, a clear understanding by the provider of how the device works is required to avoid inadvertent sub-optimal treatment.

The PocketCPR is the first autonomous (i.e., not incorporated in an AED or monitor) accelerometer based device for use in adult CPR. We demonstrate that it does not require any extra work by the caregiver and we describe this accelerometer technology so that users are aware of its potential and its limitations. If this technology is correctly used, it can make a major contribution to the quality of chest compressions.

Table of contents

15. THE ACCELEROMETER AS A DEVICE IN CPR ...................... 273
15.1 Introduction .............................................................................. 276
15.2 Materials and methods ............................................................. 276
15.3 Results .................................................................................. 278
15.4 Discussion .............................................................................. 279
15.5 Conclusions ............................................................................ 280
15.6 References .............................................................................. 281
15.1 Introduction

Performing good quality chest compression is the cornerstone of basic life support, but has repeatedly been shown to be difficult, even in expert hands. Various authors have stated that some form of feedback is essential if the desired quality is actually to be achieved.

Feedback devices are accepted during training and have a measurable, positive, effect on skills. These devices, as intelligent help, are being accepted for both out-of-hospital and in-hospital CPR. Their relevance is underlined by a consensus based on their documentation for quality assurance. Techniques used for feedback (i.e., about the depth of compression) range from force sensitive to piezoelectric accelerometer technology. The effective, safe use of feedback devices hinges on the ability of end users to evaluate the value of feedback offered. Caregivers should be aware of the potential as well as any limitations of the device, such as motion artefacts, sensory accuracy or extra exertion. This involves an understanding of force and caregiver work, as well as the physical reaction of the thorax to compressions, which has been demonstrated to be a potential challenge.

This study focuses on an accelerometer-based feedback device, the PocketCPR™ from a clinical and technological standpoint, with specific attention for caregiver work. We present information of which every caregiver using such a feedback device should be aware and contrasts the accelerometer with other, commonly available, feedback entities.

15.2 Materials and methods

For this study a highly precise (cumulative measurement error of ± 0.6 mm) manikin was used, based on a Resusci Anne (Laerdal Medical, Stavanger N), including a linear potentiometer (Type S13FLP100A, Sakae Tsushin Kogyo Co., Ltd, Japan) to measure the displacement of the anterior chest wall relative to the back (sternum-to-spine distance). This replaced the less accurate system...
Chapter 15: Getting feedback right

15.1 Introduction

Performing good quality chest compression is the cornerstone of basic life support, but has repeatedly been shown to be difficult, even in expert hands. Various authors have stated that some form of feedback is essential if the desired quality is actually to be achieved. Feedback devices are accepted during training and have a measurable, positive, effect on skills. These devices, as intelligent help, are being accepted for both out-of-hospital and in-hospital CPR. Their relevance is underlined by a consensus based on their documentation for quality assurance. Techniques used for feedback (i.e., about the depth of compression) range from force sensitive to piezoelectric accelerometer technology. The effective, safe use of feedback devices hinges on the ability of end users to evaluate the value of feedback offered. Caregivers should be aware of the potential as well as any limitations of the device, such as motion artefacts, sensory accuracy or extra exertion. This involves an understanding of force and caregiver work, as well as the physical reaction of the thorax to compressions, which has been demonstrated to be a potential challenge.

This study focuses on an accelerometer-based feedback device, the PocketCPR™ (Figure 1) from a clinical and technological standpoint, with specific attention for caregiver work. We present information of which every caregiver using such a feedback device should be aware and contrasts the accelerometer with other, commonly available, feedback entities.

15.2 Materials and methods

For this study a highly precise (cumulative measurement error of ± 0.6 mm) manikin was used, based on a Resusci Anne (Laerdal Medical, Stavanger N), including a linear potentiometer (Type S13FLP100A, Sakae Tsushin Kogyo Co., Ltd, Japan) to measure the displacement of the anterior chest wall relative to the back (sternum-to-spine distance). This replaced the less accurate system designed for training with the SkillReporter™ or VAM™. A linear voltage differential transformer (LVDT, Type SM100, WayCon, Taufkirchen, DE.) was attached to the firm underlying surface to avoid reference bias (Figure 2). This LVDT measures the total movement of the compression piston on the manikin, including any movement not detected by the potentiometer such as compression of the feedback device. The spring system simulating the elasticity of the manikin’s chest wall was the standard system available in all Laerdal SkillRecorder Resusci Anne (Laerdal, Stavanger, N).

The manikin was placed under an external, pressure adjustable, compression generator connected to the hospital wall compressed air source (72.5 ± 2.5 PSI). This device uses a piston (compression ring 45 mm diameter) to generate compressions with an error of 1.5 mm. (Figure 2) Calibration of the pressure device was confirmed beforehand (Seca Model 708, Hamburg, D). The piston was adjusted so that it just made contact with the manikin/device prior to every series of tests.

Figure 1: The PocketCPR™. The on-off button (arrow) allows for audio instructions on rhythm, frequency (metronome) and ventilation, pause information, as well as visual (4 LEDS along the rounded top edge). It offers information in terms of Guidelines directive (40-50 mm) compression depth. It is sized to fit under the caregiver’s hand.

Figure 2: Experimental setup. Note that the compression device has its own lower support avoiding any effect of underlying surfaces. The piston compresses straight down, and can be used for single or series of compressions. The LVDT and potentiometer are drawn in to clarify the difference in what they measure. See the text for further details.

The PocketCPR™ (Zoll Medical Systems, Chelmsford, MA, USA: <www.pocketcpr.com>; Figure 1) was used as an example for accelerometer-oriented devices. This reusable, battery-powered device incorporates the sensor technique, measurement algorithms and prompting messages already in general use as the Real CPR Help™ feedback technology found in ZOLL AEDs. It is the first “stand alone” accelerometer based feedback system in clinical use, weighing 80 grams, and including both audio and visual feedback in the form of four LEDs.

Forty-eight sets of compression series were analyzed using pressures for actual sternum-to-spine movements of 30 to 60 mm in 10 mm increments. After calibration, a series of 60 compressions was performed with the force shown to achieve that desired compression depth. After the first 30 compressions, a five-second pause was observed as an external check for instrumentation drift. In a random order, direct compressions were performed by the piston on the sternum, compression sets on the PocketCPR or on the block of wood lying on the sternum. The block of wood, with exactly the same surface area as the PocketCPR, was used to simulate a control situation, allowing caregiver work to be calculated. All sets were repeated after repositioning of the
manikin (20 mm caudally and 10 mm cranially) with respect to the piston. These series shifted the pressure point on the sternum to introduce other force vectors and any cantilever motion of the sternum.

Data was collected in real time using a laptop computer with analysis post hoc. All data was entered into an SPSS v16 database (SPSS Inc. Chicago, IL, USA). The one-way ANOVA (repeated measures) was used to compare mean depths. The independent measures t-test was used to investigate the workloads with and without the PocketCPR.

15.3 Results

Compression pressures of 25-60 pounds per square inch (PSI) were sufficient for the range of 30–60 mm of potentiometer recorded sternum-to-spine compression. Table 1 and Figure 3 demonstrate that there are no important changes in total downward movement of the piston with or without extra devices on the sternum (p > 0.9), no additional work (p > 0.8), or variation of feedback attributable to the accelerometer was observed under the different positioning conditions. Small and inconsistent differences in actual sternum-to-spine compressions were documented in all three conditions. Movement of the wood block, the compression piston, or the accelerometer slightly cranially or caudally on the sternum did not change the work done, the quality of the feedback or the actually achieved compression depths.

Table 1: Results by series and compression depth. Data presented as mean ± standard deviation. (SD)

<table>
<thead>
<tr>
<th>Target depth (mm)</th>
<th>Pressure (PSI)</th>
<th>Central (mm)</th>
<th>External LVDT</th>
<th>10mm Cranial LVDT</th>
<th>20mm Distal LVDT</th>
<th>Statistics</th>
</tr>
</thead>
<tbody>
<tr>
<td>30 mm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Piston directly</td>
<td>27</td>
<td>30.3 ± 0.2</td>
<td>30.7 ± 0.2</td>
<td>30.8 ± 0.3</td>
<td>29.8 ± 0.5</td>
<td>NS</td>
</tr>
<tr>
<td>- Via wood block</td>
<td></td>
<td>30.2 ± 0.5</td>
<td>30.6 ± 0.2</td>
<td>29.9 ± 0.3</td>
<td>31.0 ± 0.6</td>
<td></td>
</tr>
<tr>
<td>- Via PocketCPR</td>
<td></td>
<td>30.0 ± 0.2</td>
<td>29.4 ± 0.3</td>
<td>29.8 ± 0.4</td>
<td>30.4 ± 0.5</td>
<td></td>
</tr>
<tr>
<td>40 mm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Piston directly</td>
<td>33</td>
<td>39.4 ± 0.2</td>
<td>39.2 ± 0.3</td>
<td>39.7 ± 0.6</td>
<td>40.2 ± 0.3</td>
<td>NS</td>
</tr>
<tr>
<td>- Via wood block</td>
<td></td>
<td>40.4 ± 0.2</td>
<td>41.0 ± 0.3</td>
<td>39.9 ± 0.6</td>
<td>40.7 ± 0.4</td>
<td></td>
</tr>
<tr>
<td>- Via PocketCPR</td>
<td></td>
<td>39.9 ± 0.2</td>
<td>39.2 ± 0.5</td>
<td>39.6 ± 0.3</td>
<td>40.2 ± 0.4</td>
<td></td>
</tr>
<tr>
<td>50 mm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Piston directly</td>
<td>40</td>
<td>49.0 ± 0.2</td>
<td>48.5 ± 0.3</td>
<td>48.2 ± 0.6</td>
<td>49.6 ± 0.4</td>
<td>NS</td>
</tr>
<tr>
<td>- Via wood block</td>
<td></td>
<td>49.2 ± 0.6</td>
<td>49.4 ± 0.3</td>
<td>49.8 ± 0.5</td>
<td>50.2 ± 0.3</td>
<td></td>
</tr>
<tr>
<td>- Via PocketCPR</td>
<td></td>
<td>48.2 ± 0.4</td>
<td>48.3 ± 1.0</td>
<td>48.0 ± 0.7</td>
<td>49.8 ± 0.3</td>
<td></td>
</tr>
<tr>
<td>60 mm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Piston directly</td>
<td>49</td>
<td>60.3 ± 0.1</td>
<td>59.9 ± 0.3</td>
<td>59.5 ± 0.6</td>
<td>60.2 ± 0.4</td>
<td>NS</td>
</tr>
<tr>
<td>- Via wood block</td>
<td></td>
<td>59.0 ± 0.3</td>
<td>59.2 ± 0.3</td>
<td>58.9 ± 0.6</td>
<td>59.0 ± 0.9</td>
<td></td>
</tr>
<tr>
<td>- Via PocketCPR</td>
<td></td>
<td>59.0 ± 0.2</td>
<td>58.2 ± 0.3</td>
<td>58.0 ± 0.6</td>
<td>59.2 ± 0.4</td>
<td></td>
</tr>
</tbody>
</table>

Statistics: NS; NS; NS; NS

Notes: Central position refers to placement of the PocketCPR exactly in the middle of the inter-nipple line. LVDT = measures the total movement from piston end to back support of the manikin. Potentiometer = measures the sternum-to-spine compression. A larger LVDT number means that clinically extra hand travel or caregiver work might be performed. The differences in the table are very small. Note that the small size of the piston end has no effect on the force-distance relationship.
distance relationship. means that clinically extra hand travel or caregiver work might be performed. The differences in manikin. Potentiometer = measures the sternum-to-spine compression. A larger LVDT number nipple line. LVDT = measures the total movement from piston end to back support of the † = Central position refers to placement of the PocketCPR exactly in the middle of the inter-
small inconsistent differences, with no clinical impact on caregiver work (p > 0.8, all directions).

Table 1: Summary of differences between LVDT and potentiometer measurements. Note

Table 1: Results by series and compression depth. Data presented as mean ± standard deviation.

<table>
<thead>
<tr>
<th>Target depth (mm)</th>
<th>LVDT</th>
<th>Potentiometer</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Central† (mm)</td>
<td>Central† (mm)</td>
</tr>
<tr>
<td>27 (30mm)</td>
<td>58.9 ± 0.6</td>
<td>58.0 ± 0.6</td>
</tr>
<tr>
<td>33 (40mm)</td>
<td>59.2 ± 0.3</td>
<td>59.9 ± 0.3</td>
</tr>
<tr>
<td>40 (50mm)</td>
<td>59.9 ± 0.3</td>
<td>60.3 ± 0.1</td>
</tr>
<tr>
<td>49 (60mm)</td>
<td>49.2 ± 0.3</td>
<td>49.0 ± 0.2</td>
</tr>
</tbody>
</table>

Figure 3: Calculated caregiver work as a function of force and depth. Work (force x distance x area [with force as measured by LVDT]) by target compression depth, with normalization of area, shown as mean with standard deviation bars. There is no relevant or statistically significant extra work (p > 0.8) involved in the use of the PocketCPR. The small visual differences are due to minor compression of the PocketCPR due to the small (45 mm diameter) piston instead of the width of the hand, and measurement error. Note that the forces applied were standardized even if the exact target depth was not reached (potentiometer measurement). The work done with only the piston is not shown as its surface area is much smaller than that of the wood block/PocketCPR.

15.4 Discussion

The principal results of this study are that feedback given by the PocketCPR, an accelerometer-based, handheld feedback device for adult basic life support is empirical, reproducible, and does not involve extra compression work for the caregiver.

These findings complement a recent editorial by Leary and Abella, who focused on the difficulty in getting effective and constantly good compressions into practice. They point out that feedback devices and rescuer fatigue may be two pivotal qualities in the real world of improving patient care. However, the usefulness of feedback is directly effected by its cost, its quality and the ability of the caregivers to understand what they are shown.

Work in good chest compressions is calculated from the vertical movement of the caregivers hands, and increases if the feedback device must also be compressed. However, work also involves factors such as compression technique (in particular the compression-relaxation ratio), the caregiver’s weight and their position. Physical ability to perform good compressions may not, as previously thought, be a core issue.

Two types of feedback devices are in common use. One is pressure-sensing oriented, such as the CPR-Ezy™ or the CPRplus™. These devices correlate measured compression force, applied onto the sternum through the device, with the depth of compression and typically require some extra work by the caregiver. These devices are hand held, mechanically oriented, and most typically
found in the lay environment giving visual and auditory feedback. They are not sensitive to compliance of an underlying surface.

In contrast to force sensing, accelerometer-based systems, such as those ZOLL has placed in AED’s and monitors, and the Laerdal Q-CPR system, have been introduced into professional practice since 2002. Accelerometer refers to their measurement technique. This technology is not new, with accelerometers used to record subtle eye movement in the 1960’s. In one conceptual form, it consists of a piece of paddle-shaped silicon or a weighted cantilever beam. The base of the paddle or cantilever beam is attached to a frame. As the frame moves up and down with respect to the paddle, strain is translated into an electrical signal. Sensitive to gravity (with units of m/s^2) distance (units of m) is calculated by a mathematical algorithm in the device, based on a number of assumptions such as that gravity is not changing. By limiting calculations to frequencies within a designated range (e.g., between 1 and 2.5 Hz), movements such as that during transportation by ambulance can be filtered out. The PocketCPR measures from compression to compression using its own zero orientation, making placement for the patient on a rigid underlying surface a requirement if the reported total compression distance is to be exactly the same as the clinically relevant sternum-to-spine movement.

The study has a number of technical limitations. Our testing of the PocketCPR, as an example of current accelerometer technology, focused on the issue of movement, work and indirectly its reproducibility under carefully controlled mechanical conditions. The perception of work by a caregiver was not tested. We also sought to influence the force vectors on the manikin’s chest. This effect could only be moderately simulated using our bench test arrangement since the manikin’s chest plate is very firm.

15.5 Conclusions

Our study demonstrates that accelerometer-based systems, and specifically the PocketCPR, do not require extra work. Accelerometer-based systems are highly accurate and not sensitive to chest stiffness or depth since they support the Guideline directive and thereby avoid caregiver interpretation. The proper use of this device requires a rigid underlying surface.

The use of feedback devices should be strongly encouraged and the PocketCPR seems a good example of developing technology suitable for general, professional and lay use since it complements the technology in AEDs.
15.6 References


Conclusions and perspectives
Chapter 16: Conclusions and perspectives

16.1 Introduction

Cardiopulmonary resuscitation has been compared to 'end game' medicine: the results are poor, the emotional and physical aspects demanding, and the expense seemly prohibitive though no one has been able to calculate the cost of a good quality year of life saved. The costs in training, preparedness and implementation of adequate systems in growing daily, while an increased impact on outcome has only begun to be demonstrated.

This Chapter discusses, briefly, the results of the studies in this dissertation and ties them to current thinking. It also projects this line of thinking to issues perhaps not directly discussed in the dissertation but relevant to the issue at hand. In the end, the core issue, its fundamental challenges and their practical translation in terms of the possibilities of 'two hands' should have gained clarity.

16.2 Chest compressions: this dissertation

Revitalization, better known as resuscitation, has been a subject of human interest during all of recorded history as it touches on the core of divine determination, the time of death. From fascinating incidental reports in ancient religious texts, it has developed into an occurrence of epic proportions as sudden cardiac death accounts for ± 5.6% of annual mortality. Initial, pragmatic and non-invasive actions to deter premature death, such as placing the victim crosswise on a horse induced to trot, has given way to more aggressive interventions, such as opening the lateral chest wall and compressing the heart manually. Current treatment, initiated by Kouwenhoven and coworkers in the 1960's, returned action to a non-invasive technique by introducing closed chest cardiac resuscitation, involving compression of the chest wall as its principal mechanism, although without understanding the complex underlying events. This abrupt switch, which at the same time was overwhelmingly embraced by the general population, led to strong individual opinions as to how and why the technique works. The facts show, however, that dismal survival rates have now persisted for almost half a century duration in the face of widely popularized teaching using manikins as models, with only relatively small changes in policy. Retention and application of these so called discontinuous skills is not nearly as good as those imprinted when learning how to ride a bicycle.

Kouwenhoven's arguments that the (left) ventricle is compressed between the sternum and the vertebral column was shown to be incorrect. The 4 – 5 cm impression depth is insufficient to squeeze the ventricles even if their location were correct, since interior sternum-to-backbone distance is some 12.5 centimeters. The complexity of the underlying mechanisms reappears when ultrasound investigations showed that left ventricular cavity size may change by ± 20 ml. In addition, technical flaws and user limitations seem to abound, including the abject lack of both skill and the recognition of this lack by caregivers, boundary conditions such as the effects of supporting surfaces, the methods used for training, and the absence of focused treatment opportunities in those without ventricular fibrillation. Adjuvant techniques have been surprisingly consistent in their inability to demonstrate improvements outside the controlled environs of the laboratory. The most readily applicable, single, exception seems to be the use of feedback devices by both lay- and professional caregivers.

The approach in this dissertation, is the development of a mathematical model, based in physiology, to search for an understanding of the complex internal events, as well as to accept that Kouwenhoven's conviction that "two hands are enough" may hold an initial truth, but that the initial truth is not a goal unto itself. The use of analytical methods such as models to investigate single components will, after some time, allow integration of individual confounders into larger chains and gradually fulfill all requirements for success. Early data suggests that the human
16.1 Introduction

Cardiopulmonary resuscitation has been compared to ‘end game’ medicine: the results are poor, the emotional and physical aspects demanding, and the expense seemingly prohibitive though no one has been able to calculate the cost of a good quality year of life saved. The costs in training, preparedness and implementation of adequate systems in growing daily, while an increased impact on outcome has only begun to be demonstrated.

This Chapter discusses, briefly, the results of the studies in this dissertation and ties them to current thinking. It also projects this line of thinking to issues perhaps not directly discussed in the dissertation but relevant to the issue at hand. In the end, the core issue, its fundamental challenges and their practical translation in terms of the possibilities of ‘two hands’ should have gained clarity.

16.2 Chest compressions: this dissertation

Revitalization, better known as resuscitation, has been a subject of human interest during all of recorded history as it touches on the core of divine determination, the time of death. From fascinating incidental reports in ancient religious texts, it has developed into an occurrence of epic proportions as sudden cardiac death accounts for ± 5.6% of annual mortality. Initial, pragmatic and non-invasive actions to deter premature death, such as placing the victim crosswise on a horse induced to trot, has given way to more aggressive interventions, such as opening the lateral chest wall and compressing the heart manually. Current treatment, initiated by Kouwenhoven and coworkers in the 1960’s, returned action to a non-invasive technique by introducing closed chest cardiac resuscitation, involving compression of the chest wall as its principal mechanism, although without understanding the complex underlying events. This abrupt switch, which at the same time was overwhelmingly embraced by the general population, led to strong individual opinions as to how and why the technique works. The facts show, however, that dismal survival rates have now persisted for almost half a century duration in the face of widely popularized teaching using manikins as models, with only relatively small changes in policy. Retention and application of these so called discontinuous skills is not nearly as good as those imprinted when learning how to ride a bicycle.

Kouwenhoven’s arguments that the (left) ventricle is compressed between the sternum and the vertebral column was shown to be incorrect. The 4 – 5 cm impression depth is insufficient to squeeze the ventricles even if their location were correct, since interior sternum-to-backbone distance is some 12.5 centimeters. The complexity of the underlying mechanisms reappears when ultrasound investigations showed that left ventricular cavity size may change by ± 20 ml. In addition, technical flaws and user limitations seem to abound, including the abject lack of both skill and the recognition of this lack by caregivers, boundary conditions such as the effects of supporting surfaces, the methods used for training, and the absence of focused treatment opportunities in those without ventricular fibrillation. Adjuvant techniques have been surprisingly consistent in their inability to demonstrate improvements outside the controlled environs of the laboratory. The most readily applicable, single, exception seems to be the use of feedback devices by both lay- and professional caregivers.

The approach in this dissertation, is the development of a mathematical model, based in physiology, to search for an understanding of the complex internal events, as well as to accept that Kouwenhoven’s conviction that “two hands are enough” may hold an initial truth, but that the initial truth is not a goal unto itself. The use of analytical methods such as models to investigate single components will, after some time, allow integration of individual confounders into larger chains and gradually fulfill all requirements for success. Early data suggests that the human
cardiovascular system and thoracic cavity are intrinsically poorly configured for resuscitation activities. The impedance defined flow principle predicts CPR to shift fluid out of the thoracic cavity and into the abdomen and also that focus on the arteries may be void without consideration of the veins. Small differences in volume in the arterial system, minor alteration of the stiffness of the myocardium or vascular resistance can, independently, decrease effective output by as much as 90%. The (central) veins, indeed as shown by the model, are a key to functionality in CPR. The modeling artifact of “negative” volumes thereby gains the clinical relevance that chest compressions generate a transmural pressure sufficiently large to lock down venous volumes over a large portion of the compression and relaxation cycle.

This dissertation addresses two fundamental issues and has one goal: what is actually known about the ability to use chest compressions, or more specifically a cyclic pressure applied to the vasculature in the torso, to make blood move effectively. Succinctly stated, the dissertation set out to search for theoretical and practical support of the premise that “two hands are enough” by looking for fundamental critical factors in chest compressions. That cardiopulmonary resuscitation saves lives is not, intrinsically, at issue. The general ‘chain of survival’ points out goals and processes keynotes. It did not set out to usurp or undermine, but to find methods to understand, judge and improve on chest compression techniques. We recognized at the outset that this may violate the KISS principle.

Its principal outcomes are that if two hands are enough to save a life, the physiology underlying this survival may be attributable more to the unique characteristics of the patient, their situation, and their circulatory system at that specific moment in time than to the supposed simplicity of the activity involved. The model demonstrates that there is an absolute requirement of getting at least one cardiac valve to close during compression. This dissertation suggests that while two hands have the potential to be enough (to start), the impact of equal chest compressions may not be equal all of the time.

These conclusions are not completely unexpected and other authors have alluded to them. We seem to be the first to query the ‘initial conditions’ guiding, but at the same time limiting, how resuscitation is performed, and stimulating renewed interest in the professional resuscitation medicine community.

16.3 An adjunct to developing thinking and practice

The results and thinking put forward in this dissertation may seem progressive, perhaps even radical. However, there are clear indications that they are more path finding than they seem.

It is clear, and there is general support in the literature for this, that chest compressions remain the core functional tool in resuscitation medicine. At the same time, this “basic” skill has been determined to be poorly performed. Regrettably, the term “basic” has become confused with synonym “simple”; and the task is relegated to those unable to perform “advanced” techniques. No one has been willing to suggest a useful definition for adequacy, such as 95% good compressions, and the use of feedback devices continue to open hearts and minds to improvable quality. Where ventricular fibrillation has always been the focus of treatable, its decreasing incidence mandates that AEDs should all be equipped with interactive real-time feedback support, geared primarily to the compression and then to the process indicators. This will allow skilled professional care to arrive to a more viable patient, allowing rapid, patient specific, and useful application of mechanical compression devices, followed immediately by (non-ROSC) transportation for

8 “Keep it simple, stupid.” A modern variation of Da Vinci’s “Simplicity is the ultimate sophistication.”

286 Chapter 16: Conclusions and perspectives
processes keynotes. It did not set out to usurp or undermine, but to find methods to understand, and the use of feedback devices continue to open hearts and minds to improvable quality. Where one has been willing to suggest a useful definition for adequacy, such as 95% good compressions, synonym “simple”, and the task is relegated to those unable to perform “advanced” techniques. No determined to be poorly performed. Regrettably, the term “basic” has become confused with

The results and thinking put forward in this dissertation may seem progressive, perhaps even resuscitation is performed, and stimulating renewed interest in the professional resuscitation to be the first to query the ‘initial conditions’ guiding, but at the same time limiting, how These conclusions are not completely unexpected and other authors have alluded to them. We seem looking for fundamental critical factors in chest compressions. That cardiopulmonary resuscitation

This moves CPR a step further to a full circle: where Kouwenhoven moved the technique both out of-hospital and to the lay community, such a philosophy returns it to a mainstream medical procedure.

16.4 Future perspectives

A change in philosophy is becoming visible on the horizon. Kouwenhoven’s dangerous oversimplification of CPR treatment appears to be causing concern. This will create openings to take into account that CPR activities result in time-varying responses, requiring feedback to the caregiver to allow gearing to the patient’s needs.

In turn, this will demand control over the modalities of chest compression. It is difficult to imagine accommodation of such variations during manual compressions, hence they should be automated by mechanical devices, which can be adjusted by the caregiver or on the basis of direct biofeedback.

The Donders model will be utilized to help identify the origin of time-varying responses and aid in designing schedules for the feedback loops. This will require further adaption of the model to properties and behavior of the venous circulation. The latter is significantly different from the arterial circulation, successfully analyzed with the aid of earlier versions of the model. Preliminary studies have exposed a number of features of the venous system, such as the effect of compression force on its thin walls causing collapse, volume trapping and sloshing. Their easy expansion offer a unique ability for blood storage. CPR administration to the victim places a strong emphasis on the non-linear behavior of the veins, in particular the larger ones, resulting from the magnitude of the compression signal (force). Another unusual phenomenon is the ‘locking’ of a (small) volume of blood in a compartment as a consequence of other nonlinearities. Conceptually, by placing a catheter with a parachuting-inflatable balloon (PIB) at the inlets to the right heart and allowing its deployment to be timed synchronically with mechanical compressions may eliminate this issue completely, by reducing sloshing from up to 80% of volume to a meager 10-15%. This lack of valves seemed a surprising oversight and a cynical person might query whether this functional oversight should not be seen as a hint that transthoracic resuscitation of the human heart was not divinely intended. The development of this PIB is one of the clinical products of this dissertation project, and has been embraced by a large medical research firm and is progressing from ‘intellectual property’ to initial mock-up stages.

Recent examples of large, well organized, RCTs have been unable to find evidence that differences in terms of survival can be achieved, be it by placing AEDs in the home of high-risk patients, or with the use of thrombolytic treatment, can be explained: the complexity of CPR effectively precludes large effects when only one aspect is adapted and other prerequisites are not met. A more functional, analytic approach will offer opportunities: if evaluation of mechanical CPR is being performed the ability of the device to produce and maintain a predefined circulation should be the principal endpoint, not hospital discharge. This requires both scientists and clinicians to work together to define functional endpoints, measurement techniques and strategies. Once mechanical devices have been ‘released’ to optimize compressions - without the limitations set because of

** Submitted and accepted as Intellectual Property patent (IP ref. num. 22273)
caregiver constraints and with the help of bio-feedback modalities, patient-directed (PDR) resuscitation becomes a realistic option. The first, careful steps have been taken in this direction.

While modeling will offer insight into mechanisms available for influence, clinicians must produce the time-sensitive, patient-specific, feedback and monitoring devices. This new approach may encounter some, small, resistance, not because it is fundamentally a new discipline or approach, but because it encroaches on historical mindset that “two hands are enough” until there is an adequate perfusion rhythm, and only after which, further invasive interventions are opportune. Succinctly put, it moves a portion of resuscitation which Kouwenhoven donated to the layman caregiver, back into the prerogative of resuscitation medicine practitioners, who may or may not be physicians.

16.5 Conclusions

Cardiopulmonary resuscitation is not ‘end game’ medicine. The contradiction of how is it possible that with unpredictable chest compressions we expect that CPR can have a real impact on survival remains, as actual evidence on what is exactly right is sparse and the material complex. Since we are not willing to accept that those surviving do so because they are destined to do so, or because their specific condition is amiable to small interventions, focused and critical work will continue to be needed to finally improve outcome.
Chapter 16: Conclusions and perspectives

16.5 Conclusions

Cardiopulmonary resuscitation is not 'end game' medicine. The contradiction of how is it possible that with unpredictable chest compressions we expect that CPR can have a real impact on survival remains, as actual evidence on what is exactly right is sparse and the material complex. Since we are not willing to accept that those surviving do so because they are destined to do so, or because their specific condition is amiable to small interventions, focused and critical work will continue to be needed to finally improve outcome.

16.6 References


17 ATLS Advanced Trauma Life Support Program for Doctors (7th Ed.) Committee on Traumatology, American College of Surgeons.


Summary
17.1 Introduction

This chapter offers a brief summary of each of the chapters in this dissertation, highlighting the points of discussion they bring forward and identifying the conclusions in them. Few references are used, but where necessary, the reader is referred back to the full text.

This dissertation sets out to look into resuscitation medicine, and more specifically the premise advanced by Kouwenhoven et al. that “two hands are enough (… to save a life)”. 1 Focusing on chest compressions, it approached this task from a sectarian, non-conformist, principle of ‘can we understand and validate’ that which we do. It did not look into outcomes, medication, or even the specific patient involved.

17.2 Summaries of the individual chapters

Part I

Chapter 1 describes the challenge and the mission statement. Cardiopulmonary resuscitation (CPR) changed in the 1960s. From a physician driven, in-hospital, invasive procedure it moved to a lay-driven, out-of-hospital activity, involving training, psycho-motor skills, and persisting poor outcomes. Despite the results, it as an area of medicine which has generated enthusiasm, commitment and belief that the effort is useful. Understanding, is a potential contrast for belief, has, however, remained limited in this utterly complex material. CPR sets out to restore order in a framework of minutes, following a cardiac event leading to acute cardiac death, endocrinological chaos and hemodynamic disaster.

The chapter briefly names the theory of how blood is moved during resuscitation, its historical reference and relevance, that the action of bystanders has been reported to improve chances of (good) survival by 50%, and that, most interestingly, the 2005 Guidelines broke with tradition and looked for a window of opportunity instead of strictly for available evidence. 2 The focus of the dissertation, chest compressions and their significance, is laid out for the reader.

This chapter sets out to wet the appetite of the reader and offers a positive-critical questioning frame of mind for the remainder of the dissertation.

Chapter 2 sets out to prepare the reader for CPR. It offers a brief course in CPR as currently performed by both lay- and professional caregivers, placing this in the context of the chain of survival. It describes in some detail the psycho-motor skills needed by a rescuer and the technique involved in their use. Following the more general information, the manikin, a practical teaching tool, is introduced.

As one of the few places in the dissertation, chapter 2 presents and briefly discusses outcome definitions and statistics. It introduces the reader to the concepts of pump theory and its place in current teaching, and closes with a section on the (in)possibilities of monitoring in CPR, the step towards the core of the dissertation. Units, measures and some equations are supplied to lay groundwork for Part II of the dissertation.

Following chapter 2, the reader has accumulated the required tools to place the positive-critical questioning into current practice.

Chapter 3, with a provocative title, approaches the aspect of chest compressions from a historical, mechanistic, point of view. A great many researchers have been intrigued by the movement of blood during CPR. The number of publications directly addressing or linked to this topic is, therefore, extensive.
17.1 Introduction

This chapter offers a brief summary of each of the chapters in this dissertation, highlighting the points of discussion they bring forward and identifying the conclusions in them. Few references are used, but where necessary, the reader is referred back to the full text.

This dissertation sets out to look into resuscitation medicine, and more specifically the premise advanced by Kouwenhoven et al. that “two hands are enough (… to save a life)". Focusing on chest compressions, it approached this task from a sectarian, non-conformist, principle of ‘can we understand and validate’ that which we do. It did not look into outcomes, medication, or even the specific patient involved.

17.2 Summaries of the individual chapters

Part I
Chapter 1 describes the challenge and the mission statement. Cardiopulmonary resuscitation (CPR) changed in the 1960s. From a physician driven, in-hospital, invasive procedure it moved to a lay-driven, out-of-hospital activity, involving training, psycho-motor skills, and persisting poor outcomes. Despite the results, it as an area of medicine which has generated enthusiasm, commitment and belief that the effort is useful. Understanding, is a potential contrast for belief, has, however, remained limited in this utterly complex material. CPR sets out to restore order in a framework of minutes, following a cardiac event leading to acute cardiac death, endocrinological chaos and hemodynamic disaster.

The chapter briefly names the theory of how blood is moved during resuscitation, its historical reference and relevance, that the action of bystanders has been reported to improve chances of (good) survival by 50%, and that, most interestingly, the 2005 Guidelines broke with tradition and looked for a window of opportunity instead of strictly for available evidence. The focus of the dissertation, chest compressions and their significance, is laid out for the reader.

This chapter sets out to wet the appetite of the reader and offers a positive-critical questioning frame of mind for the remainder of the dissertation.

Chapter 2 sets out to prepare the reader for CPR. It offers a brief course in CPR as currently performed by both lay- and professional caregivers, placing this in the context of the chain of survival. It describes in some detail the psycho-motor skills needed by a rescuer and the technique involved in their use. Following the more general information, the manikin, a practical teaching tool, is introduced.

As one of the few places in the dissertation, chapter 2 presents and briefly discusses outcome definitions and statistics. It introduces the reader to the concepts of pump theory and its place in current teaching, and closes with a section on the (in)possibilities of monitoring in CPR, the step towards the core of the dissertation. Units, measures and some equations are supplied to lay ground work for Part II of the dissertation.

Following chapter 2, the reader has accumulated the required tools to place the positive-critical questioning into current practice.

Chapter 3, with a provocative title, approaches the aspect of chest compressions from a historical, mechanistic, point of view. A great many researchers have been intrigued by the movement of blood during CPR. The number of publications directly addressing or linked to this topic is, therefore, extensive.
In this chapter, using a chronological approach, tables summarize the model, the purpose of the study and highlight results and conclusions. The chronological approach exposes the gradual alteration in thinking within resuscitation medicine, such as the migration from small animals to large dogs and finally to swine. The compelling complexity of resuscitation becomes clearer under this limited approach, which excludes analysis of airway, ventilation, and medication aspects in CPR where they are not directly relevant to pump mechanisms. Where possible, adjunct techniques, for example the use of airway pressure to pump blood are presented and discussed, as well as monitoring opportunities, such as ultrasound, finally concluding that while there is a great deal of data available, but surprisingly little is specifically focused on the hemodynamics in CPR from a flow view-point. Analysis of the data available, remains difficult because of limitations in modeling capabilities (e.g. compliant chest, successive experimentation on one animal, difficulty in investigation in humans for both practical and ethical reasons.

After reading chapter 3, the reader may be convinced that resuscitation medicine is a very serious, highly specialized and compelling branch in medicine. Research into resuscitation effects has moved forward extensively over the years though fundamental issues in CPR remain to be addressed.

Chapter 4 is the first chapter referring to a specific aspect in the effect of chest compressions during CPR. Kouwenhoven et al. claimed that the differences between the classically addressed open chest cardiac resuscitation (OCCR) and closed chest cardiac resuscitation (CCCR) were minor, since in both cases the heart (i.e. the ventricles) was compressed.

Using a cohort of 50 patients thoracic CT-scans show that the left ventricle is not in-line between the sternum and the vertebral column, but that, the right and left in- and outflow tracts are. This work has been repeated confirming the earlier results for adults, as well as performed on children, more recently. We also demonstrate that the linear distances suggest that direct compression, causing effective decrease in ventricular size is unlikely.

Additionally, this chapter describes the forces involved in (experimental) CPR, and using the same technique as in chapter 3, describes the diversity of pressures and forces used, with the results ranging from \(0.1 \times 10^{-2}\) to \(2.9 \times 10^{-2}\) cm N\(^{-1}\), as a first step towards reconciliation of contradictory findings.

Ending Part I, chapter 4 rounds out the foundations of the dissertation.

Part II

Chapter 5 introduces the concepts of modeling and simulation as teaching and clinical tools to analyze complex relationships. Models but perhaps more so simulations, are well known tools for teaching purposes, and help to reduce complexity by limiting the number of variables to those defined by the user. The differences between simulation and model making are presented.

Different levels of modeling complexity are described. This chapter discusses the usefulness of models, including some of the limitations in animal models and the ability to translate fundamental research from the laboratory to clinical medicine. CPR became notorious for producing strongly divergent fundamental or laboratory versus clinical results. The reader is prepared for the Donders model as a specific example of an RLC model, unique in its functional heart, suitable for research into the circulation.

\* Published work
Chapter 6 continues where chapter 5 left off, by bringing general modeling concepts to the specific situation of research into CPR. Chapter 6 describes the initial conditions and philosophy in the Donders model as well its evolutionary states. The reader is introduced to the active and passive components in the model as well as the validity of parameter values and assumptions. Each of the components is briefly presented.

Chapter 7 * returns to CPR, using the model in a simple form as vehicle. Since cardiac pump theory has remained the most popular version of pump theory, the chapter describes a series of experiments into the potential of the cardiac pump to move blood forward from an asystolic heart. This chapter described the importance of valves for the cardiac pump theory and, using a range of compression values demonstrates the relationships described in physiology.

Important findings are that preload has a more than proportional effect of left ventricular output, that compression pressure is incompletely related to cardiac output under CPR conditions raising flows from 17 to 88 ml compression⁻¹, and that, since the experiments were done under 1 Hz, current practice of 1.8 Hz seems logical. The most fundamental outcome is that impedance distribution in the system may be the largest, single, important parameter.

The chapter concludes by suggesting that if valves are competent -incompetent valves are not compatible with any effective forward flow in this set up- the cardiac pump model seems a viable if not very effective source of cardiac output. It also identifies the cause of negative volumes, a viable mathematical, but not clinical, aspect caused by transmural pressure gradients outside of physiology.

Chapter 8 * continues further into CPR and modeling. The circulation is now modeled as a closed loop. Experiments with the Donders model are used to extend it into impedance defined flow and control theory. Using opportunities from chapter 7, pump theory is extended to include five different compressions forms as variations on the cardiac pump and four different modalities using the thoracic pump theory as base. The effects of external compressions (p_e(t)) on the vascular structures, such as the large veins is shown to be a major component in effective pumping, and the concept of sloshing (i.e. regurgitant flow of up to a fraction of 0.8) is introduced.

The chapter demonstrates that if impedance defined flow is assumed as forward flow without valves in a closed system, the human circulation as a whole is relatively resistant to this due to the varying impedances within the system.

Chapter 9 * proceeds to use the Donders model to investigate and describe a number of central issues with clinical CPR relevance. The coronary flow, as more than just the CPP, is explored and the effects of compressions, interruption of compressions and the addition of ventilations are modeled, as is the effects of compression forces on stroke volumes. Intravascular volume and its impact on resuscibility is reintroduced (chapter 7) as well as a new concept in resuscitation medicine called volume sequestration.

While preliminary in its scope, this chapter brings the very theoretical modeling more into clinical and applied medicine, setting the stage for Part III, in which clinical resuscitation science furthers the line of thinking that doing chest compressions well is the core in resuscitation medicine.

* Published work
Part III

Chapter 10 * refocuses on clinical CPR: where the Donders model has produced suggestions on how the circulation may be moved, chapter 10 looks towards the manikin as the model in its classical sense. Early in the 1960’s manikins became an essential part of resuscitation training, but getting the chest compression right (as per guidelines: hand position, depth, frequency, c:r ratio and non-leaning) has proven to be difficult. Even just after training scores are poor, begging the question of how we lay- and professional caregivers perform.

This chapter, using two different manuscripts, looks at the issue in functional simulation: does the manikin contribute or is it the quality required in the course which determines outcome. Both extremely simple as well as the disadvantages of complex simulators without the basic option of chest compressions are discussed with their advantages and potential for improvement.

A careful suggestion is made that not quantity in training but quality in training may have to be further extended to compression technique and perhaps less on process measures. But enticing remains the suggested impact of bystanders chest compressions and the improvement suggested to be attributed to this, even if the quality of compressions is poor (chapter 12).

Chapter 11 * continues on this line of thinking and places not the model, but the caregiver in the central role. Using a cohort of interns and residents in anesthesiology, knowledge, skills and self reflection were mirrored to 1992 guideline standards.

Using a cohort of 55 anesthesia trainees, an unannounced prospective blind analysis was performed. A recording manikin, 35 multiple choice questions and a questionnaire were used to inventory these three components. A validated scoring system, developed by Berden and incorporating both scoring of individual psycho-motor skills as well as process scores, was used to assign values to skills. Of those taking part only 7 (13%) were able to perform adequately. No correlation was found between an individual’s self assessment and their actual skill levels, with or without confounders such as ICU, ER or length of time in training.

The chapter suggests that many experts may, unjustly, not be given the opportunity to evaluate their skills but develop a skewed perspective based on the assumption that frequent exposure is correlated with skill development.

Chapter 12 * approaches the topic of quality in chest compressions from a functional model and feedback viewpoint. 224 hospital staff were entered into this study, and asked to perform 270 seconds of optimal quality of chest compressions with or without a feedback device (CPREzy™). Using a non-cross over design, and looking at the effectively done compressions on a per-compressions basis, 36% ± 41 without feedback versus 6% ± 13 of compression in the feedback group showed strongly significant differences.

As in chapters 10 and 11, the ability of a caregiver to decide what is ‘adequate’ in chest compressions is at issue. The Belgian Resuscitation Database uses the opinion of EMS personnel and emergency physicians to judge quality on bystander resuscitation though this may not be valid. In training situations the opinion of instructors or peers has been used as a training tool. If effective compressions are to remain the cornerstone in resuscitation medicine, objectifying this quality seems essential.

Chapter 13 * looks further into the device used in chapter 12: the CPREzy. As a spring based system, and in the face of concerns about caregiver work, this chapter addresses the mechanics of

---

* Published work
chest compressions and analyzes the pressures and work involved. The study, using a manikin model, demonstrates that while circa 20% extra work is involved in using the CPREzy for feedback, clinical complaints by caregivers are more likely due to the increased force they are applying to CPR. The chapter also goes into the question of whether downward hand movement (i.e. 4-5 cm) or the amount of force needed for suitable chest compression (e.g. 50 kg) is the message being taught during courses.

**Chapter 14** addresses a specific in-hospital aspect in quality of chest compressions. Historically, a firm surface has been desired beneath a patient. Out-of-hospital resuscitations move patients off beds and couches, while in-hospital resuscitation is performed in bed with or even without a backboard. This study evaluated the influence of a backboard as well as the type of mattress on the efficacy of chest compressions.

The study demonstrates that large, extraneous, movements of ± 100% can be measured using a manikin on typical hospital mattresses. The largest positive factor seems to be the width of the backboard, as it increases (doubles) the supporting surface and thereby decreases extraneous movements from as much as 11 cm for a 5 cm effective compression depth.

In conjunction with chapters 10-13, chapter 14 suggests that getting chest compressions right may be more complex than previously thought and that feedback devices should be available to all resuscitations certainly no later than the arrival of professional care.

**Chapter 15** describes the other of the common feedback modalities. Where the CPREzy is a force-sensing modality (chapter 4), accelerometer based technology is distance-sensing and may be sensitive to confounders such as mattresses and other extraneous movements. This chapter described the technology and lays out the mechanics for lay- and professional caregivers.

**Chapter 16** sets out to tie all three sections of the dissertation together in a discussion and conclusions format. It also offers a number of aspects within resuscitation medicine, and more specifically chest compressions, which may be suitable for further research, both with the Donders model as in animal- and human models. It also weighs the opportunities currently available in mechanical CPR. Where in Chapter 2 PDR is introduced, this chapter binds together the opportunities and potential horizon in resuscitation medicine for the coming decennia, and offers a succinct comment on the need to shift resuscitation from the prerogative of the lay community back towards resuscitation professionals.

Finally in closure, the reader is returned to chapter 1, the relevance and potential of CPR as an important and growing field within critical care medicine.

* Published work
17.3 References

Samenvatting
Chapter 18: Samenvatting

18.1 Inleiding
Dit hoofdstuk biedt een korte samenvatting van elk van de afzonderlijke hoofdstukken in deze dissertatie. Hierin worden de punten van discussie naar voren gebracht en de conclusies gepresenteerd. Een klein aantal referenties wordt gebruikt en waar noodzakelijk, wordt de lezer terug verwezen naar de volledige tekst.

Deze dissertatie rapporteert over onderzoek naar reanimatie binnen de geneeskunde, en meer specifiek naar de ideeën die door Kouwenhoven et al. zijn geformuleerd dat “twee handen voldoende zijn (…..om een leven te redden)”. 1 De dissertatie benadert deze taak vanuit een sektarisch, non-conformistisch, principe van “kunnen we begrijpen en bevestigen” wat we doen en concentreert zich op het enkelvoudige aspect van thorax compressie. Er is niet gekeken naar de resultaten van reanimaties, andere leeftijdsgroepen dan volwassenen, medicatie gebruik of zelfs de specifieke patiënt die hierbij betrokken was.

18.2 Samenvatting van elk van de afzonderlijke hoofdstukken
Deel I
Hoofdstuk 1
beschrijft de uitdaging en de doelstellingen van deze dissertatie. Cardiopulmonale reanimatie (CPR) veranderde rond de 60 er jaren. Van een door een medicus gestuurde, invasieve, ziekenhuis procedure, veranderde het in een door leken gestuurde pre-hospitale activiteit die training, psychomotorische vaardigheden en aanhoudende slechte overleving met zich meebracht. Ondanks de matige resultaten na reanimatie, is het een domein van de geneeskunde dat enthousiasme, betrokkenheid en de overtuiging heeft gegeven dat de inspanningen verdienstelijk zijn. Het begrijpen, als een mogelijk contrast ten opzicht van de overtuiging (geloof of dogma), blijft desalniettemin zeer beperkt in deze extreem gecompliceerde materie. Er is geen discussie over deze complexiteit: CPR is het vertrekpunt om orde te herstellen, in een periode van minuten, waarin cardiale problemen leiden tot cardiale dood, endocrinologische chaos en hemodynamische catastrofe.

Dit hoofdstuk noemt beknopt de theorie over de bloedstroom gedurende reanimatie, de historische referenties en relevantie daarvan. Tevens wordt het goede effect van de aanwezigheid van omstanders gerapporteerd die een (goede) overlevingskans met 50% groter maken. Tot slot wordt aangegeven dat de Richtlijnen van 2005 braken met traditie en belangstelling toonden voor mogelijkheden en niet strikt alleen keken naar beschikbare bewijzen uit onderzoek. 2 De focus van de dissertatie, thorax compressie, en de betekenis daarvan, wordt beschikbaar gesteld voor de lezer.

Dit hoofdstuk probeert de lezer te interesseren in een positief-kritische opbouw qua gedachtengang als benaderingswijze in de dissertatie. De dissertatie is onderverdeeld in drie delen, namelijk: de fundamentele aspecten, modellen voor CPR en verschillende praktische aspecten van CPR rondom thorax compressies.

Hoofdstuk 2
bereidt de lezer voor op het toepassen van CPR. Het biedt een beknopte CPR cursus zoals die tegenwoordig wordt gegeven aan leken, zowel als aan professionele hulpverleners en plaatst dit tegen de achtergrond van de “keten van overleving”. Het beschrijft psycho-motorische vaardigheden die vereist worden van de hulpverlener en de daarbij behorende techniek. Het wordt gevolgd door meer algemene informatie over het model dat als een praktisch hulpmiddel ten behoeve van het onderwijs wordt geïntroduceerd.

Dit hoofdstuk gaat op een beknopte wijze in op definities omtrent overleving en statistieken. Het introduceert de lezer tot de denkbeelden betreffende de pomp theorie en de plaats daarvan in het huidige onderwijs en het sluit af met een beschrijving van de (on)mogelijkheden van het bewaken en objectief volgen van wat CPR doet, de stap naar de kern van deze dissertatie. Tabellen, metingen en vergelijkingen worden toegevoegd als basis voor Deel II van de dissertatie.

In hoofdstuk 2 ziet de lezer de aanpak om de positief kritische vraagstelling toe te passen in de huidige praktijk.
18.1 Inleiding

Dit hoofdstuk biedt een korte samenvatting van elk van de afzonderlijke hoofdstukken in deze dissertatie. Hierin worden de punten van discussie naar voren gebracht en de conclusies gepresenteerd. Een klein aantal referenties wordt gebruikt en waar noodzakelijk, wordt de lezer terug verwezen naar de volledige tekst.

Deze dissertatie rapporteert over een onderzoek naar reanimatie binnen de geneeskunde, en meer specifiek naar de ideeën die door Kouwenhoven et al. zijn geformuleerd dat “twee handen voldoende zijn (…..om een leven te redden)’. De dissertatie benadert deze taak vanuit een sektarisch, non-conformistisch, principe van “kunnen we begrijpen en bevestigen” wat we doen en concentreert zich op het enkelvoudige aspect van thorax compressie. Er is niet gekeken naar de resultaten van reanimaties, andere leeftijdgroepen dan volwassenen, medicatie gebruik of zelfs de specifieke patiënt die hierbij betrokken was.

18.2 Samenvatting van elk van de afzonderlijke hoofdstukken

Deel I
Hoofdstuk 1 beschrijft de uitdaging en de doelstellingen van deze dissertatie. Cardiopulmonale reanimatie (CPR) veranderde rond de 60e jaren. Van een door een medicus gestuurde, invasieve, ziekenhuis procedure, veranderde het in een door leken gestuurde pre-hospitale activiteit die training, psychomotorische vaardigheden en aanhoudende slechte overleving met zich meebreacht. Ondanks de matige resultaten na reanimatie, is het een domein van de geneeskunde dat enthousiasme, betrokkenheid en de overtuiging heeft gegeven dat de inspanningen verdienstelijk zijn. Het begrijpen, als een mogelijk contrast ten opzichte van de overtuiging (geloof of dogma), blijft desalniettemin zeer beperkt in deze extreem gecompliceerde materie. Er is geen discussie over deze complexiteit: CPR is het vertrekpunt om orde te herstellen, in een periode van minuten, waarin cardiale problemen leiden tot cardiale dood, endocrinologische chaos en hemodynamische catastrofe.

Dit hoofdstuk noemt beknopt de theorie over de bloedstroom gedurende reanimatie, de historische referenties en relevantie daarvan. Tevens wordt het goede effect van de aanwezigheid van omstanders gerapporteerd die een (goede) overlevingskans met 50% groter maken. Tot slot wordt aangegeven dat de Richtlijnen van 2005 braken met traditie en belangstelling toonden voor mogelijkheden en niet strikt alleen kelen naar beschikbare bewijzen uit onderzoek. De focus van de dissertatie, thorax compressie, en de betekenis daarvan, wordt beschikbaar gesteld voor de lezer.

Dit hoofdstuk probeert de lezer te interesseren in een positief-kritische opbouw qua gedachtengang als benaderingswijze in de dissertatie. De dissertatie is onderverdeeld in drie delen, namelijk: de fundamentele aspecten, modellen voor CPR en verschillende praktische aspecten van CPR rondom thorax compressies.

Hoofdstuk 2 bereidt de lezer voor op het toepassen van CPR. Het biedt een beknopte CPR cursus zoals die tegenwoordig wordt gegeven aan leken, zowel als aan professionele hulpverleners en plaatst dit tegen de achtergrond van de “keten van overleving”. Het beschrijft psycho-motorische vaardigheden die vereist worden van de hulpverlener en de daarbij behorende techniek. Het wordt gevolgd door meer algemene informatie over het model dat als een praktisch hulpmiddel ten behoeve van het onderwijs wordt geïntroduceerd. Dit hoofdstuk gaat op een beknopte wijze in op definities omtrent overleving en statistieken. Het introduceert de lezer tot de denkbeelden betreffende de pomp theorie en de plaats daarvan in het huidige onderwijs en het sluit af met een beschrijving van de (on)mogelijkheden van het bewaken en objectief volgen van wat CPR doet, de stap naar de kern van deze dissertatie. Tabellen, metingen en vergelijkingen worden toegevoegd als basis voor Deel II van de dissertatie.

In hoofdstuk 2 ziet de lezer de aanpak om de positief kritische vraagstelling toe te passen in de huidige praktijk.
**Hoofdstuk 3** heeft een uitdagerende titel. Het benadert thorax compressies vanuit een historisch en mechanisch oogpunt. Een groot aantal auteurs zijn geboeid geraakt door de bloedstroom gedurende CPR. Het aantal publicaties dat direct betrekking heeft op dit onderwerp, of dat er nauw mee verbonden is, is dan ook groot.

In dit hoofdstuk worden, in chronologische volgorde, een overzicht van het model, het doel van de studie, als ook de hoogtepunten van de resultaten en conclusies van verschillende studies weergegeven. Door gebruik te maken van een chronologische benadering, wordt de geleidelijke verandering in denken betreffende reanimatie verduidelijkt. De ontwikkeling in termen van theoretische en experimentele modellen, dat wil zeggen de verschuiving van het onderzoek in kleine dieren naar grote honden en uiteindelijk naar varkens wordt opgesomd. De fascinerende complexiteit van reanimatie wordt duidelijker door deze beperkte benadering. De ontgonnen gebeurtenissen belangrijke analyse van de luchtweg, ventilatie en medicatie aspecten bij CPR worden genegeerd omdat deze niet direct relevantie hebben voor het pomp mechanisme.

Aanvullende technieken worden, daar waar mogelijk beschreven en besproken, zoals dat het toepassen van druk in de luchtweg om bloeddoorstroming te veroorzaken, evenals de mogelijkheden om effecten met ultrasound te volgen. Uiteindelijk wordt geconcludeerd dat er een groot aantal gegevens beschikbaar zijn, maar dat er verbazingwekkend weinig gegevens zijn die specifiek focussen op CPR vanuit een hemodynamisch oogpunt. De gegevens die beschikbaar zijn, zijn moeilijk te analyseren vanwege de beperkingen van het model, zoals de compliantie van de thorax, opeenvolgende onderzoeken op één dier, de moeilijkheid om onderzoek op mensen te doen voordat reguliere, ongecontroleerde acties zijn ondernomen.

De bedoeling is dat de lezer, na het lezen van hoofdstuk 3, overtuigd is dat reanimatie een serieuze, zeer gespecialiseerde en fascinerende tak binnen de geneeskunde is die het onderzoek naar de effecten van reanimatie geweldig heeft uitgebreid maar dat fundamentele vragen omtrent CPR nog steeds onbeantwoord blijven.

**Hoofdstuk 4** is het eerste hoofdstuk dat zich richt op de specifieke aspecten van het effect van thorax compressie gedurende CPR. Kouwenhoven et al. argumenteerden dat het verschil tussen klassieke chirurgische “open chest cardiac reanimatie” (OCCR) en de noninvasieve “closed chest cardiac reanimatie” (CCCR) klein was, omdat in beide procedures het hart (d.w.z. de ventrikel) samengedrukt worden.1

Door middel van een serie van 50 CT-scans van de thorax van patiënten zien we dat de linker ventrikel niet in lijn ligt tussen het sternum en de wervelkolom, maar dat de rechter aan en afvoerende bloedvaten daar wel liggen. Dit is herhaald met dezelfde resultaten voor zowel volwassenen, als meer recent ook voor kinderen.34,5 We laten ook zien dat de lineaire afstand suggereert dat directe compressie, die een effectieve vermindering veroorzaakt van de grootte van de ventrikel onwaarschijnlijk is. Bovendien bespruit dit hoofdstuk de krachten die in (experimentele) CPR worden toegepast. Het beschrijft de diversiteit van de drukken en de krachten die gebruikt worden, met resultaten voor thorax compliantie die variëren van $0.1 \times 10^{-5}$ to $2.9 \times 10^{-5}$ cm N$^{-1}$, als een eerste stap naar het in overeenstemming brengen van tegenstrijdige resultaten.

Hoofdstuk 4 aan het eind van Deel I, rond de basis van de dissertatie af.

**Deel II**

**Hoofdstuk 5** introduceert de denkbeelden omtrent modellen en simulatie in het onderwijs en de klinische instrumenten om complexe verhoudingen te analyseren. Modellen, maar wellicht meer simulaties zijn bekende instrumenten voor lesdoeleinden en helpen de complexiteit te verminderen door het aantal variabelen te beperken tot degene die door de gebruiker geanalyseerd worden. De verschillen tussen simulatie en het maken van een model worden voorgedragen en de verschillende niveaus van complexiteit van de modellen wordt beschreven.
Hoofdstuk 6 gaat verder waar hoofdstuk 5 ophoudt en brengt de algemene eisen van het maken van modellen naar voren in de specifieke omgeving van onderzoek binnen CPR. Hoofdstuk 6 beschrijft de eerste voorwaarden en de filosofie van het Donders model, als ook de status van de ontwikkeling hiervan. De lezer maakt kennis met de actieve en passieve componenten in het model en ook met de geldigheid van de waarden van de parameters en de veronderstellingen. Elk van deze componenten wordt beknopt gepresenteerd.

Hoofdstuk 7 keert terug naar CPR met gebruikmaking van het model in een eenvoudige vorm als middel. Aangezien de cardiale pomp theorie de meest bekende pomp theorie is gebleven, beschrijft het een serie van experimenten vanuit de mogelijkheid van de cardiale pomp, waarbij bloed voorwaarts beweegt vanuit een hart in asystolie. Dit hoofdstuk beschrijft de betekenis van kleppen voor de cardiale pomp theorie en het gebruik maken van compressie waarden geeft de relatie aan die wordt beschreven in de fysiologie.

Een belangrijke conclusie is dat de preload verhoudingsgewijs het grootste effect heeft op de linker ventriculaire output, dat de compressie druk incompiete gerealiseerd kan zijn aan cardiac output en onder CPR omstandigheden de flow doet stijgen met 17 naar 88 ml per compressie en dat, omdat de experimenten zijn gedaan met 1Hz, in de huidige praktijk 1.8 Hz een logisch vervolg kan zijn. Het meest fundamentele resultaat is dat de impedantie verdeling in het systeem de grootste, enige, belangrijke parameter zou kunnen zijn.

Het hoofdstuk eindigt met het idee dat als kleppen competent zijn- incompetente kleppen beletten de mogelijkheid tot effectieve voorwaartse flow- het cardiale pomp model dan een bruikbare, zo niet een erg effectieve bron is van cardiac output. Het stelt ook vast dat de oorzaak van negatieve volumes, hetgeen wiskundig mogelijk is, maar niet klinisch hoewel de transmurale drukken de fysiologische grens overschrijden.

Hoofdstuk 8 borduurt verder voort op CPR en modelbouw. De circulatie wordt nu voorgesteld als een gesloten lus en experimenten met het Donders model worden gebruikt om het uit te breiden met impendante gedefinieerde stroom en control theorie. Met gebruikmaking van de mogelijkheden van hoofdstuk 7, wordt de pomp theorie uitgebreid tot vijf verschillende compressie vormen als variatie op de cardiale pomp en vier verschillende vormen met gebruikmaking van de thoracale pomp theorie als basis. Het effect van externe compressie (p(t)) op de vasculaire structuren, zoals op de grote venen, toont aan dat dit een belangrijk onderdeel is voor effectieve compressie. Het verschijnsel van ‘sloshing’ (d.w.z. terugstromende flow tot een fractie van 0.8) wordt geïntroduceerd.

Dit hoofdstuk toont aan dat als impendante gedefinieerde flow wordt gezien als voorwaartse flow zonder kleppen in een gesloten systeem, de menselijke circulatie als geheel, hier weerstand tegen biedt door de verschillende impedanties binnen het systeem.

Hoofdstuk 9 gaat verder met het Donders model en onderzoekt en omschrijft een aantal centrale onderwerpen die klinisch relevant zijn voor CPR. De coronare bloedstroom, meer dan alleen de druk gradiant, en meer in het bijzonder de CPP (coronary perfusion pressure), wordt onderzocht en de effecten van compressie, interruptie van compressie en de toevoeging van de ventilatie worden gemodelleerd, even als de effecten van compressie grootte op het slagvolume. Intravasculair volume en de invloed daarvan op het reanimeren worden opnieuw geïntroduceerd (hoofdstuk 7) als ook een nieuw begrip binnen reanimatie in de geneeskunde dat volume sequestratie heet.
Als voorbereiding brengt dit hoofdstuk het theoretische modeleren dichter bij het niveau van klinisch toegepaste geneeskunde, om een overgang te maken naar Deel III, waarin klinische wetenschap met betrekking tot reanimatie, verder gaat in de lijn van denken dan het juist uitvoeren van thorax compressie, de kern waarom het draait in reanimatie.

Deel III  
**Hoofdstuk 10** focuseert opnieuw op klinische CPR: terwijl het Donders model suggesties heeft gegeven over hoe de circulatie zich gedraagt, kijkt hoofdstuk 10 naar de manikin als model in de klassieke betekenis. Vroeg in de 60-iger jaren werden manikins een essentieel onderdeel van de reanimatie training, maar het op juiste wijze uitvoeren van de thorax compressie (zoals in de Richtlijnen: hand positie, diepte, frequentie, compressie: relaxatie ratio en het niet leunen) is gebleken moeilijk te zijn. Zelfs net na een training periode zijn de resultaten slecht, waardoor men zich moet afvragen hoe goed we als leken - en professionele hulpverleners - compressies uitvoeren. Dit hoofdstuk maakt gebruik van twee verschillende manuscripten en kijkt naar de functionele simulatie: voegt de manikin daadwerkelijk iets toe, of is het de vereiste kwaliteit van de cursus die het resultaat bepaalt. Zowel de extreem eenvoudige manikin, als de nadelen van complexe simulators zonder de basis optie voor thorax compressie worden besproken, en er wordt gekeken naar de voordelen en de mogelijkheden ter verbetering.

Een voorzichtige suggestie wordt gedaan dat niet de kwantiteit van de training, maar de kwaliteit van de training meer moet worden uitgebreid in de richting van compressie techniek en wellicht minder naar procesmatige beoordelingen. Maar het blijft verleidelijk dit niet te doen als de Richtlijnen de suggestie geven dat aan het effect van thorax compressie uitgevoerd door standaard de hulpverlener een verbetering wordt toegerekend, zelfs als de kwaliteit van de compressie beneden peil is (hoofdstuk 12).


Een voorzichtige suggestie wordt gedaan dat niet de kwantiteit van de training, maar de kwaliteit van de training meer moet worden uitgebreid in de richting van compressie techniek en wellicht minder naar procesmatige beoordelingen. Maar het blijft verleidelijk dit niet te doen als de Richtlijnen de suggestie geven dat aan het effect van thorax compressie uitgevoerd door standaard de hulpverlener een verbetering wordt toegerekend, zelfs als de kwaliteit van de compressie beneden peil is (hoofdstuk 12).

**Hoofdstuk 12** onderzoekt het onderwerp van de kwaliteit van thorax compressie vanuit het oogpunt van een functioneel model en feedback. Er namen 224 ziekenhuis medewerkers deel aan deze studie en aan hen werd gevraagd om 270 seconden optimale kwaliteit thorax compressie te doen, met of zonder een hulpmiddel voor feedback (CPREzy™). Via een non-cross over studie en met behulp van de beoordeling van elke enkelvoudige compressie, zien we dat 64% ± 41% in de groep zonder feedback versus 94% ± 13% van de compressies in de feedback groep, adequaat waren. Zowel in absolute getallen als in de spreiding zijn grote significante verschillen te zien.

Zoals in hoofdstuk 10 en 11 al wordt beschreven, is het vermogen van een hulpverlener om te bepalen wat een “adequate” thorax compressie is, een punt van kritiek. De Belgische Reanimatie Database gebruikt de opinie van ambulance personeel en Urgentisten om de kwaliteit van reanimatie door omstanders te beoordelen, maar de vraag is of dit betrouwbaar is. 

---

304 Chapter 18: Samenvatting
Gedurende training sessies werd de mening van instructeurs of collega’s gebruikt als instrumenten bij de training. Als effectieve compressie de hoeksteen moet blijven in de reanimatie geneeskunde, dan lijkt het objectiveren van de kwaliteit van de compressies een essentieel onderwerp.

**Hoofdstuk 13** bestudeert het apparaat dat gebruikt wordt in hoofdstuk 12, de CPREzy. Als een systeem gebaseerd op veerkracht, en vanwege bezorgdheid voor de arbeid die de hulpverlener moet verrichten, behandelt dit hoofdstuk het mechanisme van thorax compressie en analyseert de kracht en de arbeid die dit met zich meebrengt. Deze studie, waarbij gebruik wordt gemaakt van een speciaal ontwikkelde oefenfantoom, laat zien dat hoewel het gebruik van de CPREzy circa 20% extra werk met zich meebrengt, de klinische bezwaren van de hulpverlener meer waarschijnlijk toegeschreven moeten worden aan de schijnbare toename van de kracht die nu wordt uitgeoefend tijdens CPR om de gewenste compressie diepte te bereiken. Het hoofdstuk gaat ook in op de vraag of de neerwaartse beweging van de hand (4-5 cm), of de kracht die nodig is voor een geschikte thorax compressie (50 kg) de boodschap moet zijn die wordt uitgedragen gedurende de cursussen.

**Hoofdstuk 14** behandelt een specifiek aspect van de kwaliteit van thorax compressie binnen het ziekenhuis. Historisch gezien is een stevige harde ondergrond onder de patiënt gewenst. Bij reanimaties buiten het ziekenhuis wordt de patiënt verplaatst van het bed of de bank, terwijl in het ziekenhuis de reanimatie wordt uitgevoerd in bed, met of zonder gebruikmaking van een bedplank die tevens de oppervlakte van de thorax vergroot. Deze studie beoordeelt de invloed van een harde bedplank, als ook van het type matras in relatie tot de effectiviteit van de thorax compressie. De studie toont aan dat grote, externe bewegingen gemeten kunnen worden bij het gebruik van een manikin op typische ziekenhuis matrassen. De grootste positieve factor blijkt de breedte van de harde bedplank te zijn, omdat die de ondersteunende oppervlakte vergroot (verdubbelt) en daardoor het zakken van de patiënt in de matras verminderd van ongeveer 11 cm voor een 5 cm effectieve compressie diepte.

In combinatie met hoofdstukken 10-13, suggereert hoofdstuk 14 dat het juist uitvoeren van thorax compressie een complexer geheel is dan men voorheen dacht en dat feedback middelen beschikbaar zouden moeten zijn bij alle reanimaties zeker niet later dan de aankomst van de professionele hulpverlening.

**Hoofdstuk 15** beschrijft de andere feedback mogelijkheden. De CPREzy is een krachtgestuurd instrument (hoofdstuk 4) terwijl de op de accelerometer gebaseerde technologie een verplaatsingsgestuurd instrument is dat derhalve gevoelig kan zijn voor de invloed van de matras en andere externe bewegingen. Dit hoofdstuk beschrijft de technologie en de mechanica voor leken en professionele hulpverleners.

**Hoofdstuk 16** verbindt de drie delen van de dissertatie in de vorm van discussie en conclusies. Het biedt tevens een aantal aspecten die binnen de reanimatie geneeskunde, en meer specifiek de thorax compressie, geschikt zijn voor verder onderzoek, zowel met behulp van het Donders model, als in dier en fysieke modellen. Het weegt ook de mogelijkheden af van de huidig beschikbare mechanische CPR instrumenten. Waar in hoofdstuk 2 PDR wordt geïntroduceerd, brengt dit hoofdstuk de onderdelen bij elkaar: professionele hulpverleners -niet noodzakelijk artsen- moeten gebruik maken van de nieuwe mogelijkheden en de horizon van reanimatie en meer in het bijzonder basic life support weer verbreden.

Ten slotte wordt de lezer aan het eind terug verwezen naar hoofdstuk 1, waarin de relevantie en de mogelijkheden van CPR als een belangrijk en groeiend onderdeel wordt gezien van reguliere geneeskunde.

**Hoofdstuk 17** geeft een samenvatting per hoofdstuk, die in **Hoofdstuk 18** in het Nederlands vertaald is.

Chapter 18: Samenvatting 305
18.3 References

Acknowledgements
Acknowledgements

Working on, and finally writing, a dissertation is a unique experience, involving many people, ideas, challenges and some little time. It would be impossible to name all those who have contributed to and assisted in this work, but naming just a few would be in order.

Professor Scheffer, Gert Jan. Combining the arts of general clinical and academic medicine might, for some, be a challenge. However, to refine these qualities even further with statesmanship makes for a remarkable person. As mentor and promoter you focused and carefully steered this project to its culmination: not an endpoint but a stepping stone forward. Many thanks for making that step real.

This dissertation also reflects to a degree on the compatibility and cohesion of fundamental and applied science. Few sons have had the pleasure and distinction of having a father who is also coach, motivator and finally, promoter. Bram, thanks for all the unremitting, the consistent, even unrelenting, ability to focus ideas, put forward new ones, as well as project them towards a finite goal. Now that the writing is done, and a project incorporating both abstract and clinical medicine is drawing to its first mile post, we’ll need to do this discussion over again.

I would like to thank Professor Van Swieten, Professor Van der Hoeve and Professor Roukema and the members of the defense committee for their reading of the dissertation and enthusiasm for resuscitation medicine.

For my colleagues in the Department of Anesthesia & Resuscitation: Cathy, Frank, Frederik, Herman, Jan, Joëlle, Robbert, Teun, and Ysabel. When I joined the department the dissertation project was well under way, its completion inevitable and seemingly imminent. Thanks for the support, the allowances over the years to focus on and develop the departmental role in our Quick Response Team system and to pick up any slack I may have left. My thanks too for keeping me firmly rooted in the relevance of clinical medicine by pointing out the importance of ‘getting it done’ as well as allowing time to explore the ‘why’, the ‘how’ and the ‘teaching’ of the thing.

For the members of the Elisabeth Hospital Quick Response Team, or Outreach team, or SIT, or what you would like to call it, my thanks and compliments. Your consistent willingness to excel in patient care under typically difficult conditions, to strive for more and for better care, and to achieve your goals even if this involves scaling walls, performing a cricothyroidectomy a windy 30 m up, or just getting the “tower” right, makes me proud to be one of your team members.

My special thanks to the Instructor corps past and , Alyssa, Angelique, Ger, Hans v H, Hans S, Jelle, Lenneke and Paul: getting the message out, being convincing and always ready to be there has offered great support and underlined your enthusiasm over the years. My gratitude and thanks.

I also owe a debt to the members of Staff, both Medical and Nursing, who lived with and embrace an intention for excellence, and allowed me the room to use up some of my energy in working towards that goal.

I acknowledge a debt and as well as great deal of recognition is due to Ms. Godelieve Engbersen and Sjors Clemens, Librarians extraordinaire. They rivaled the skills, speed and accuracy of Ivy League institutions in acquiring references even if the available data was sketchy at best.

To D.A. Goulden, to whom no comma was insignificant and not dot could be left unturned. Your contributions were timely and most welcome. For all your contributions and thoughts my thanks.

To all those not named, and you are legion, my gratitude and thanks for your acceptance of my project and prize myself lucky with such friends.

This dissertation also reflects to a degree on the compatibility and cohesion of fundamental and applied science. Few sons have had the pleasure and distinction of having a father who is also coach, motivator and finally, promoter. Bram, thanks for all the unremitting, the consistent, even unrelenting, ability to focus ideas, put forward new ones, as well as project them towards a finite goal. Now that the writing is done, and a project incorporating both abstract and clinical medicine is drawing to its first mile post, we’ll need to do this discussion over again.

I would like to thank Professor Van Swieten, Professor Van der Hoeve and Professor Roukema and the members of the defense committee for their reading of the dissertation and enthusiasm for resuscitation medicine.

For my colleagues in the Department of Anesthesia & Resuscitation: Cathy, Frank, Frederik, Herman, Jan, Joëlle, Robbert, Teun, and Ysabel. When I joined the department the dissertation project was well under way, its completion inevitable and seemingly imminent. Thanks for the support, the allowances over the years to focus on and develop the departmental role in our Quick Response Team system and to pick up any slack I may have left. My thanks too for keeping me firmly rooted in the relevance of clinical medicine by pointing out the importance of ‘getting it done’ as well as allowing time to explore the ‘why’, the ‘how’ and the ‘teaching’ of the thing.

For the members of the Elisabeth Hospital Quick Response Team, or Outreach team, or SIT, or what you would like to call it, my thanks and compliments. Your consistent willingness to excel in patient care under typically difficult conditions, to strive for more and for better care, and to achieve your goals even if this involves scaling walls, performing a cricothyroidectomy a windy 30 m up, or just getting the “tower” right, makes me proud to be one of your team members.

My special thanks to the Instructor corps past and , Alyssa, Angelique, Ger, Hans v H, Hans S, Jelle, Lenneke and Paul: getting the message out, being convincing and always ready to be there has offered great support and underlined your enthusiasm over the years. My gratitude and thanks.

I also owe a debt to the members of Staff, both Medical and Nursing, who lived with and embrace an intention for excellence, and allowed me the room to use up some of my energy in working towards that goal.

I acknowledge a debt and as well as great deal of recognition is due to Ms. Godelieve Engbersen and Sjors Clemens, Librarians extraordinaire. They rivaled the skills, speed and accuracy of Ivy League institutions in acquiring references even if the available data was sketchy at best.

To D.A. Goulden, to whom no comma was insignificant and not dot could be left unturned. Your contributions were timely and most welcome. For all your contributions and thoughts my thanks.
Professor Schilders, Will Sr., Tammo Jan, and more recently Professor Woerlee, Igor, Yvette and Paul: finishing one project has led to new and enticing collaborative challenges, my thanks for all you have contributed to date and remember, we are not done (yet).

For my family, Cathy, Jeske, Tristan and Cyril. Saddled with my “idée fix” of more than 15 years, they were willing to let me drop (literally) what was in my hands to go off to the hospital to resuscitate, to teach, to withdraw into my study to write and to debate the writing, even when this was less than convenient. Cathy, who finally limited herself to “drive carefully”; Jeske who, together with her good friend Amber, showed themselves tenacious database wizards; Tristan, who would sometimes accompany me and developed a special relationship with the nurses on the dialysis unit (they have really neat electric chairs); and finally Cyril, who created unique, reflective moments as we cruised Brabant by night when he could not sleep. For their love, understanding and support in keeping the ship of family on course, I am truly in their debt.

For Truus, I am indebted to you for your support and understanding, even when you didn’t.

To Paul and Alyssa, paranymphs. A diversity of roles, a generosity of mind and a great ability to support, think along and critique. I am most grateful for your help and your company in this project and prize myself lucky with such friends.

Dr. Watermuller,† Emeritus Pastor, who judged my interests at an early phase, deciding that those who had an inclination to look for an answer in the here and now, were perhaps less suited to contemplate the influences of eternity: well, you were almost right!

To all those not named, and you are legion, my gratitude and thanks for your acceptance of my interest in resuscitation medicine in its many facets and your willingness to be part of this project.
Curriculum Vitae
Curriculum Vitae

Gerrit Jan Noordergraaf was born at Zeist, The Netherlands, in 1959. Moving to Haverford, on the Presbyterian Main Line in Pennsylvania at an early age laid the basis for his formative years. Immersed in the East Coast approach to learning, as well as to fundamental science, his interest in ‘why’ developed. Attending a Quaker Elementary and Middle school followed by a public High School, he graduated in 1978. During this period he trained and worked as paramedic in the nearby City of Philadelphia and as a Research fellow in the Cardiovascular Studies Unit at Temple University. He continued on to Gannon University, a Catholic Pre-Med oriented University on the shore of Lake Erie, where he also spent a year as a resident in the Anglican monastery St. Barnabas, graduating in 1982. During this period he was twice recognized for outstanding services to the public and academic communities. Discussion as to further formal education in theology was closed by the conclusion that ‘those who seek answers in brief order, (i.e. now would be good) are less suited to ponder eternity’.1

Matriculating to the Medical School at the University Utrecht in 1982, improved his Dutch language skills while he obtained his Masters in 1989 and his Medical Degree in 1991. In close cooperation with two other medical students Reinier Hoff and Henk Parmentier, and guided by Prof.dr. J.M.C. Douze1, Dr. R. van Kesteren, and Dr R.G. Beerens1, they developed courses in emergency medicine for first year medical students, residents and dentists and wrote a textbook as basis for the course. This course is still being taught and the textbook has been reprinted three times.

During his medical school training, he worked as a paramedic in CPA Stadsgewest Breda, as well as for the Regional Ambulance service in Utrecht, was at the forefront of active patient management by ambulance nurses, and structured care (SOSA) participated in the Oranje Kruis both as instructor and as a member of the advisory board on Resuscitation, was active in the Red Cross, as well as many other social and professional functions. His teaching at the University of Utrecht Medical School resulted in an appointment as a lecturer, while still a medical student.

Following completion of his degree he stayed at the University Hospital and Medical School in Utrecht, combining teaching with clinical work in the Intensive Care Unit, and in management tasks for the National Organization of Trauma Teams, the Emergency Hospital – a cooperative project between the Hospital, the Dept. of Defense and the Dept. of Health- and a position at the National Institute of Public Health and Environmental protection, Bilthoven, NL.

To further his training, he became a resident in Anesthesiology and Resuscitation at the Catholic University Medical School in Leuven, Belgium. Initially, under the leadership of Prof.dr. H. van Aken en later by Prof.dr. E. Vandermeersch, who allowed him to focus on cardio-vascular anesthesia and clinical research, he rotate through the emergency medicine and intensive care departments. Involved with the Emergency Medical System, he became medical director for the ambulance company in Aarschot (B), where he introduced protocol-driven quality management.

Finishing his training at the University Hospital Utrecht in 1999, he accepted a position in the Dept of Anesthesiology at the St. Elisabeth Hospital in Tilburg. He has remained active as a teacher, both as visiting lecturer at the University of Pennsylvania, in the St. Elisabeth Hospital, as well as outside the hospital. Currently he is involved as instructor with no less than three ‘4-letter’ courses in some aspect of emergency medicine and resuscitation. He is the secretary of the Department and coordinator for residents within the department. He has, initially with Dr. M. van

1 Dr. D. Watermulder, Senior pastor Bryn Mawr Presbyterian Church
Curriculum Vitae

Gerrit Jan Noordergraaf was born at Zeist, The Netherlands, in 1959. Moving to Haverford, on the Presbyterian Main Line in Pennsylvania at an early age laid the basis for his formative years. Immersed in the East Coast approach to learning, as well as to fundamental science, his interest in 'why' developed. Attending a Quaker Elementary and Middle school followed by a public High School, he graduated in 1978. During this period he trained and worked as paramedic in the nearby City of Philadelphia and as a Research fellow in the Cardiovascular Studies Unit at Temple University. He continued on to Gannon University, a Catholic Pre-Med oriented University on the shore of Lake Erie, where he also spent a year as a resident in the Anglican monastery St. Barnabas, graduating in 1982. During this period he was twice recognized for outstanding services to the public and academic communities.

Discussion as to further formal education in theology was closed by the conclusion that 'those who seek answers in brief order, (i.e. now would be good) are less suited to ponder eternity'.

Matriculating to the Medical School at the University Utrecht in 1982, improved his Dutch language skills while he obtained his Masters in 1989 and his Medical Degree in 1991. In close cooperation with two other medical students Reinier Hoff and Henk Parmentier, and guided by Prof.dr. J.M.C. Douze †, Dr. R. van Kesteren, and Dr R.G. Beerens †, they developed courses in emergency medicine for first year medical students, residents and dentists and wrote a textbook as basis for the course. This course is still being taught and the textbook has been reprinted three times.

During his medical school training, he worked as a paramedic in CPA Stadsgewest Breda, as well as for the Regional Ambulance service in Utrecht, was at the forefront of active patient management by ambulance nurses, and structured care (SOSA) participated in the Oranje Kruis both as instructor and as a member of the advisory board on Resuscitation, was active in the Red Cross, as well as many other social and professional functions. His teaching at the University of Utrecht Medical School resulted in an appointment as a lecturer, while still a medical student.

Following completion of his degree he stayed at the University Hospital and Medical School in Utrecht, combining teaching with clinical work in the Intensive Care Unit, and in management tasks for the National Organization of Trauma Teams, the Emergency Hospital – a cooperative project between the Hospital, the Dept. of Defense and the Dept. of Health- and a position at the National Institute of Public Health and Environmental protection, Bilthoven, NL.

To further his training, he became a resident in Anesthesiology and Resuscitation at the Catholic University Medical School in Leuven, Belgium. Initially, under the leadership of Prof.dr. H. van Aken en later by Prof.dr. E. Vandermeersch, who allowed him to focus on cardio-vascular anesthesia and clinical research, he rotate through the emergency medicine and intensive care departments. Involved with the Emergency Medical System, he became medical director for the ambulance company in Aarschot (B), where he introduced protocol-driven quality management. Finishing his training at the University Hospital Utrecht in 1999, he accepted a position in the Dept of Anesthesiology at the St. Elisabeth Hospital in Tilburg. He has remained active as a teacher, both as visiting lecturer at the University of Pennsylvania, in the St. Elisabeth Hospital, as well as outside the hospital. Currently he is involved as instructor with no less than three '4-letter' courses in some aspect of emergency medicine and resuscitation. He is the secretary of the Department and coordinator for residents within the department. He has, initially with Dr. M. van Puyenbroek, set up one of the first ‘quick response team (SIT team)’ in the hospital in Tilburg, he is chair of the steering committee, and has been instrumental in the organization and the leadership of a number of conferences on resuscitation.

He has accepted an appointment as regional coordinator for emergency medicine (ROAZ), and is a member of the Board for the Dutch Society for coordinators of resuscitations (NVCR), as well as editor for to Dutch Society of Anesthesia Nurses.

As a Lt. Colonel (Res) he is involved in a cooperative project with the Dept. of Defense and has been active in Iraq and Gabon.

His long term interest in resuscitation continued and finally resulted in this formal Ph.D. project under Prof.dr. G.J. Scheffer at the Dept. of Anesthesiology, University Hospital Nijmegen, St Radboud.
List of Publications, presentation and posters incorporated in this dissertation
### Peer reviewed publications:

<table>
<thead>
<tr>
<th>Authors</th>
<th>Title</th>
<th>In</th>
<th>Reference</th>
<th>Location(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dijkstra T, Kortsmit WJPM, Schilders WHA, Scheffer GJ, Noordergraaf A</td>
<td>Modeling in cardiopulmonary resuscitation: pumping the heart</td>
<td>Cardiovascular Engineering</td>
<td>2005; 5(3): 105-118</td>
<td>Chap 8</td>
</tr>
<tr>
<td>Noordergraaf GJ, Drinkwaard BWPM, Berkom PFJ, Hemert HP van, Venema A, Scheffer GJ, Noordergraaf A</td>
<td>The quality of chest compressions by trained personnel: the effect of feedback, via the CPREzy, in a randomized controlled trial using a manikin model</td>
<td>Resuscitation</td>
<td>2006; 69: 241-252</td>
<td>Chap 11</td>
</tr>
<tr>
<td>Berkom P van, Noordergraaf GJ</td>
<td>Integrated resuscitation simulators should retain “basic” options.</td>
<td>Resuscitation</td>
<td>2008 Mar; 76(3): 485-486</td>
<td>Chap 2, 10</td>
</tr>
<tr>
<td>Berkom P van, Noordergraaf GJ, Scheffer GJ, Noordergraaf A</td>
<td>Does feedback by the CPREzy involve extra work?</td>
<td>Resuscitation</td>
<td>2008 Mar 26 (PMID 18374466)</td>
<td>Chap 10</td>
</tr>
<tr>
<td>Noordergraaf GJ, Nabuurs J, Smits G, Berkom P van, Venema A, Scheffer GJ, Noordergraaf A</td>
<td>Accelerometer feedback during chest compressions: characteristics and value or just a must-have device.</td>
<td>Acad Emerg Med</td>
<td>2009</td>
<td>Chap 14</td>
</tr>
</tbody>
</table>
### Scientific presentations, Posters

<table>
<thead>
<tr>
<th>Author</th>
<th>Presentations, Abstracts &amp; posters</th>
<th>Presented at</th>
<th>Date</th>
<th>Location(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Noordergraaf GJ</td>
<td>Model making and compression depth [presentation]</td>
<td>2003, July 23-25</td>
<td>Chap 4</td>
<td></td>
</tr>
<tr>
<td>Noordergraaf GJ</td>
<td>Essential factors in CPR (stroke volume) [poster]</td>
<td>NVA Annual Scientific meeting (A'dam, NL)</td>
<td>2005, Sept. 30</td>
<td>Chap 10</td>
</tr>
<tr>
<td>Noordergraaf GJ</td>
<td>Coronary perfusion and no compression time: a modeling approach [poster]</td>
<td>ERC-UK Annual meeting (London UK)</td>
<td>2006, Jan. 19</td>
<td>Chap 10</td>
</tr>
<tr>
<td>Noordergraaf GJ</td>
<td>Modeling the effects of interrupted compressions: less is better. [presentation]</td>
<td>ERC-Uk Annual meeting (London, UK)</td>
<td>2006, Jan. 19</td>
<td>Chap 5, 13</td>
</tr>
<tr>
<td>Noordergraaf GJ, Scheffer GJ, Noordergraaf A</td>
<td>Stroke volumes as a window to improving CPR (using the Donders Model) [presentation + poster]</td>
<td>NVA Annual Scientific meeting (A'dam, NL)</td>
<td>2006, Sept. 29</td>
<td>Chap 6</td>
</tr>
<tr>
<td>Noordergraaf GJ</td>
<td>Force in compression [presentation]</td>
<td>Invitational symposium on CPR. (Utrecht, NL)</td>
<td>2006, Sept. 19</td>
<td>Chap 5, 13</td>
</tr>
<tr>
<td>Author</td>
<td>Presentations, Abstracts &amp; posters</td>
<td>Presented at</td>
<td>Date</td>
<td>Location(s)</td>
</tr>
<tr>
<td>------------------------------</td>
<td>---------------------------------------------------------------------------------------------------</td>
<td>------------------------------------------------</td>
<td>------------</td>
<td>-------------</td>
</tr>
<tr>
<td>Noordergraaf GJ, Janssen GCP, Noordergraaf A</td>
<td>The CPRezy’s and caregiver safety under stringent defibrillation conditions. [poster]</td>
<td>BVAR, Annual meeting (Antwerpen, B)</td>
<td>2006, Nov. 5</td>
<td>Chap 13</td>
</tr>
<tr>
<td>Berkom P van, Venema A, Schoonen H, Noordergraaf GJ</td>
<td>Electronic learning complements skills and drills training in CPR. [poster, Abstract in Resuscitation]</td>
<td>European Resuscitation Council 9th Scientific Congress. (Ghent , B)</td>
<td>2008, May 22-24</td>
<td>Chap 9, 10</td>
</tr>
<tr>
<td>Noordergraaf GJ</td>
<td>Science in CPR [presentation]</td>
<td>ESA, (Copenhagen, S)</td>
<td>2008, May 31 – June 3</td>
<td>Chap 16</td>
</tr>
<tr>
<td>Aelen P, Paulussen P, Noordergraaf GJ, Woerlee P, Henderson A, Koeken Y</td>
<td>More compression depth: only compensation or real increase? [poster]</td>
<td>ERC, 10th meeting (Keulen G)</td>
<td>2009, Oct 2-4</td>
<td>Chap 4</td>
</tr>
</tbody>
</table>
Publications, presentations & posters in this dissertation

Author Presentations, Abstracts & posters
Presented at Date Location(s)

Noordergraaf GJ, Janssen GCP, Noordergraaf A
The CPREzy's and caregiver safety under stringent defibrillation conditions.
[poster]
BVAR, Annual meeting (Antwerpen, B) 2006, Nov.

Chap 13
Noordergraaf GJ, Lampe JW, Scheffer GJ, Noordergraaf A
Inappropriate force or compression to relaxation ratio may cause volume trapping in CPR [poster, abstract in Resuscitation]
European Resuscitation Council 9th Scientific Congress. (Ghent, B) 2008, May 22-24

Chap 9
Berkom P van, Venema A, Schoonen H, Noordergraaf GJ
Electronic learning complements skills and drills training in CPR. [poster, abstract in Resuscitation]
European Resuscitation Council 9th Scientific Congress. (Ghent, B) 2008, May 22-24

Chap 9, 10
Paulussen I, Noordergraaf GJ, Woerlee PH.
Hand position: a factor in compression efficiency.
9th Scientific Congress ERC, (Ghent, Belgium) 2008, May 22-24

Chap 10, 15

Chap 16
More compression depth: only compensation or real increase? [poster]
ERC, 10th meeting (Keulen G) 2009, oct

Chap 4

Appendices
Table of contents

APPENDICES ................................................................................................................................. 1
OVERVIEW ........................................................................................................................................ 4

1 APPENDIX I: DONDER MODEL (DI, CHAPTER 7) ................................................................... 4
1.1 Equations (a and b) for the open model DI ............................................................................. 4
1.2 The complete Donders model (DI, chapter 7) ..................................................................... 5
1.3 Equations for the complete model DI .................................................................................... 6
1.3.1 Systemic veins and their valves ..................................................................................... 6
1.3.2 The right heart ................................................................................................ .......... 7
1.3.3 Pulmonary arteries ....................................................................................................... 7
1.3.4 Pulmonary periphery .................................................................................................. 8
1.3.5 Pulmonary veins ......................................................................................................... 8
1.3.6 The left heart .............................................................................................................. 8
1.3.7 The systemic arteries ................................................................................................ 9
1.3.8 reduced (practical) .................................................................................................... 9
1.3.9 reduced (practical). In addition to the equations for the coronary circulation .......... 9
1.3.10 Valves ..................................................................................................................... 9
1.4 Volumes and pressures (including locations) DI ............................................................... 10

2 THE DONDERS MODEL (DII, CHAPTER 8) ....................................................................... 11
2.1 Representation of The Donders model (II) with annotations ............................................. 11
2.2 Equations for DII .............................................................................................................. 12
2.2.1 Systemic veins and their valves ................................................................................ 12
2.2.2 The right heart ........................................................................................................ 13
2.2.3 Pulmonary arteries .................................................................................................. 13
2.2.4 Pulmonary periphery .............................................................................................. 13
2.2.5 Pulmonary veins ..................................................................................................... 14
2.2.6 The left heart .......................................................................................................... 14
2.2.7 The systemic arteries ............................................................................................. 15
2.2.8 Coronary circulation ............................................................................................... 15
2.2.9 Equations of state .................................................................................................. 15
2.2.10 Valves .................................................................................................................. 15
2.2.11 Parameter values and variables DII .................................................................. 16

3 THE DONDERS MODEL (DIII, CHAPTER 9) ...................................................................... 17
3.1 Representation of The Donders Model (DIII) with annotations ....................................... 17
3.2 Equations for DIII ............................................................................................................. 17
3.2.1 The Right Heart ...................................................................................................... 17
3.2.2 Pulmonary arteries ................................................................................................. 18
3.2.3 Pulmonary periphery ............................................................................................ 19
3.2.4 Pulmonary veins ................................................................................................... 19
3.2.5 The left heart ......................................................................................................... 19
3.2.6 The systemic arteries ........................................................................................... 20
3.2.7 Legs ....................................................................................................................... 20
3.2.8 Abdomen and Kidneys ......................................................................................... 22
3.2.9 VCI ...................................................................................................................... 22
3.2.10 Head and upper extremities ............................................................................... 23
3.2.11 Valves ................................................................................................................ 25
3.2.12 Parameter and variable values DIII ................................................................ 26
3.2.13 Overview of values in DIII .............................................................................. 27

Appendices
OVERVIEW.................................................................................................................................................. 4

1.3 Equations for the complete model DI.......................................................................................... 6

1.1 Equations (a and b) for the open model DI.................................................................................. 4

2.2 Equations for DII....................................................................................................................... 12

3.2 Equations for DIII ..................................................................................................................... 17

3.1 Representation of The Donders Model (DIII) with annotations................................................ 17

APPENDIX I: DONDER MODEL (DI, CHAPTER 7) ................................................................... 4

APPENDIX II: DONDER MODEL (DII, CHAPTER 8).............................................................................. 11

The left heart................................................................................................................................... 8

Pulmonary periphery.............................................................................................................. 8

Pulmonary arteries ................................................................................................................. 7

The right heart........................................................................................................................ 7

The systemic arteries .............................................................................................................. 9

The left heart......................................................................................................................... 14

Pulmonary periphery............................................................................................................ 13

The right heart...................................................................................................................... 13

Systemic veins and their valves............................................................................................. 12

Coronary circulation ............................................................................................................ 15

Equatiosn of state ................................................................................................................. 15

Pulmonary arteries ............................................................................................................... 18

The Right Heart .................................................................................................................... 17

Pulmonary veins ................................................................................................................... 19

Pulmonary periphery............................................................................................................ 19

Abdomen and Kidneys .......................................................................................................... 22

Legs....................................................................................................................................... 20

VCI........................................................................................................................................ 22

Parameter values and variables DII................................................................................ 16

Parameter and variable values DIII ................................................................................ 26

Valves............................................................................................................................... 25

Head and upper extremities ............................................................................................. 23

QUESTIONNAIRES USED IN CHAPTER 11 ................................................................................. 29

4.1 Overview of subjects addressed .............................................................................................. 29

4.2 Demographic and subjective information Questionnaire .......................................................... 29

4.3 Knowledge Questionnaire (translated from Dutch and validated)........................................ 30

QUESTIONNAIRES USED IN CHAPTER 12 ................................................................................. 35

5.1 The pre-assessment questionnaire .......................................................................................... 35

5.2 The post assessment questionnaire ......................................................................................... 35

5.3 The effects of combining springs with different properties as applies to CPR (appendix to the
manuscript). .................................................................................................................................. 36

APPENDIX VI TO CHAPTER 14 MODELLING THE CHARACTERISTICS FOR ANY
LOCAL SITUATION. .................................................................................................................. 38

6.1 Captions for the figures. ......................................................................................................... 39

APPENDIX VII (CHAPTER 15): MATHEMATICS INVOLVED IN FEEDBACK WITH
ACCELEROMETERS IN CHEST COMPRESSIONS' ........................................................................ 40
Overview

This section contains figures, tables and other information referred to in different places and as such moved here for the readers convenience.

1 Appendix I: Donder model (DI, Chapter 7)

1.1 Equations (a and b) for the open model DI

\[ \begin{align*}
Q_{H3}(t) &= Q_{H3}(t) \\
p_{\text{om}} - p(t) &= (R_{LX} + R_3)Q_{H13}(t) \\
p_{\text{om}}^*(t) &= \begin{cases} 
p_{\text{om}}(t) & \text{if the mitral value is open} \\
p_{\text{om}}(t) & \text{if the mitral value is shut} 
\end{cases} \\
Q_{H3}(t) - Q_{H3}(t) &= V_1(t) \\
Q_{H3}(t) &= Q_{H3}(t) \\
p_1(t) &= \begin{cases} 
p_1(t) & \text{if the aortic value is open} \\
p_1(t) & \text{if the aortic value is shut} 
\end{cases} \\
p_1(t) - p_1(t) &= (Z_{\text{om}} + R_3)Q_{H13}(t) \\
Q_{H13}(t) - Q_1(t) &= Q_{V1}(t) \\
Q_{V1} &= C_{\text{H13}} \frac{d}{dt}p_1(t) \\
V_1(t) &= V_1(0) + \int_0^t Q_{V1}(t) \, dt \\
p_1(t) - p_1(t) &= a_{\text{V1}}(V_1(t) - h_{\text{V1}})^2 + (e_{\text{V1}}V_1(t)) \\
&- d_{\text{V1}}F_{V1}(t)
\end{align*} \]

with

\[ \begin{align*}
F_{V1}(t) &= f_{V1}(t) - k_t Q_{H13}(t) \\
&+ k_2(Q_{H13}(t) - \tau_{V1}(t))^2 \\
\tau_{V1}(t) &= \tau_{V1}^t
\end{align*} \]

stroke volume = \int_0^t Q_{H13}(t) \, dt

antegrade stroke volume = \int_0^t p_{\text{om}} Q_{H13}(t) \, dt

retrograde stroke volume = \int_0^t neg Q_{H13}(t) \, dt
1.2 The complete Donders model (DI, chapter 7)
1.3 Equations for the complete model DI.

1.3.1 Systemic veins and their valves

\[ Q_{V1} = Q_{H605} + Q_{H606} - Q_{H250} \]  

(1)

\[ Q_{V1} = C_1 \frac{d}{dt} p_3 \]  

(2)

\[ Q_{V250} = Q_{H250} - Q_{H251} \]  

(3)

\[ Q_{V250} = C_{250} \frac{d}{dt} (p_{250} - p_r) \]  

(4)

\[ Q_{H251} = \frac{(p_{250} - p_{300})}{R_{250}} \]  

(5)

\[ p_3 - p_{250} = L_{250} \frac{d}{dt} Q_{H250} \]  

(6)

\[ p_{51} - p_{52} = L_{51} \frac{d}{dt} Q_{H51} + R_5 Q_{H51} \]  

(7)

\[ p_2 - p_3 = L_2 \frac{d}{dt} Q_{H606} \]  

(8)

\[ Q_{V40} = C_{40} \frac{d}{dt} (p_{50} - p_r) \]  

(9)

\[ Q_{V40} = Q_{H403} - Q_{H50} \]  

(10)

\[ Q_{V51} = C_{51} \frac{d}{dt} (p_{51} - p_r) \]  

(11)

\[ Q_{V51} = Q_{H604} - Q_{H51} \]  

(12)

\[ Q_{V200} = C_{200} \frac{d}{dt} (p_{200} - p_r) \]  

(13)

\[ Q_{V200} = Q_{H200} - Q_{H201} \]  

(14)

\[ Q_{H200} = Q_{H50} + Q_{H51} \]  

(15)

\[ p_{50} - p_{51} = L_{50} \frac{d}{dt} Q_{H50} + R_5 Q_{H50} \]  

(16)

\[ p_{53} - p_{201} = R_{200} Q_{H201} \]  

(17)

\[ p_{300} - p_{300} = L_{300} \frac{d}{dt} Q_{H201} + R_{201} Q_{H201} \]  

(18)

\[ V_{200}(t) = V_{200}(t = 0) + \int Q_{V200} dt \]  

(19)

\[ V_{250}(t) = V_{250}(t = 0) + \int Q_{V250} dt \]  

(20)

\[ V_{51}(t) = V_{51}(t = 0) + \int Q_{V51} dt \]  

(21)

\[ V_{50}(t) = V_{50}(t = 0) + \int Q_{V50} dt \]  

(22)

\[ V_{r}(t) = V_{r}(t = 0) + \int Q_{r} dt \]  

(23)
1.3.2 The right heart

\[ P_{300} - P_{301} = R_{300}Q_{H300} + L_{300} \frac{d}{dt} Q_{H300} \] (24)

\[ P_{301} - P_{310} = R_{311}Q_{H301} + L_{310} \frac{d}{dt} Q_{H301} \] (25)

\[ Q_{V300} = Q_{H302} - Q_{H301} \] (26)

\[ Q_{V350} = Q_{H301} - Q_{H400} \] (27)

\[ P_{350} - P_{352} = R_{350}Q_{H301} \] (28)

\[ p_{301} - p_{e} = \pm a_{RA}(V_{300} - b_{RA})^2 + (c_{RA}V_{300} - d_{RA})f_{RA} \] (29)

\[ p_{352}(V_{350}, t, Q_{H400}) - p_{e}(t) = \pm a_{RV}(V_{350} - B_{RV})^2 + \ldots \] (30)

\[ ...(c_{RV}V_{350} - d_{RV})F_{RV}(t, Q_{400}) \]

\[ F_{RV} = f_{RV}(t) - k_{1RV}Q_{H400}(t) + k_{2RV}Q_{H400}(t - \tau_{RV}) \] (31)

\[ P_{352} - P_{351} = R_{351}Q_{H400} + L_{350} \frac{d}{dt} Q_{H400} \] (32)

\[ Q_{H300} = Q_{H251} + Q_{H201} \] (33)

\[ V_{300}(t) = V_{300}(t = 0) + \int (Q_{V300})dt \] (34)

\[ V_{350}(t) = V_{350}(t = 0) + \int (Q_{V350})dt \] (35)

\[ V_{450} = \int Q_{H400}dt \] (36)

\[ Q_{H302} = Q_{H300} + Q_{H700} \] (37)

1.3.3 Pulmonary arteries

\[ Q_{V400} = Q_{H400} - Q_{H430} \] (38)

\[ Q_{V400} = c_{400} \frac{d}{dt}(p_{401} - p_{e}) \] (39)

\[ p_{400} - p_{401} = Q_{H400}Z_{400} \] (40)

\[ p_{401} - p_{430} = L_{410} \frac{d}{dt} Q_{H430} + R_{411}Q_{H430} \] (41)

\[ V_{400}(t) = V_{400}(t = 0) + \int Q_{V400}dt \] (42)
1.3.4 Pulmonary periphery

\[ Q_{v,430} = Q_{H,430} - Q_{H,460} \]  
(43)

\[ Q_{v,430} = \frac{d}{dt} (p_{431} - p_e) \]  
(44)

\[ p_{431} - p_{461} = L_{440} \frac{d}{dt} Q_{H,460} + R_{441} Q_{H,460} \]  
(45)

\[ p_{430} - p_{431} = R_{430} Q_{H,430} \]  
(46)

\[ V_{430}(t) = V_{430}(t = 0) + \int Q_{v,430} \, dt \]  
(47)

1.3.5 Pulmonary veins

\[ Q_{v,460} = Q_{H,460} - Q_{H,500} \]  
(48)

\[ Q_{v,460} = \frac{d}{dt} (p_{462} - p_e) \]  
(49)

\[ p_{462} - p_{502} = L_{460} \frac{d}{dt} Q_{H,500} + R_{461} Q_{H,500} \]  
(50)

\[ p_{462} - p_{462} = R_{460} Q_{H,460} \]  
(51)

\[ V_{460}(t) = V_{460}(t = 0) + \int Q_{v,460} \, dt \]  
(52)

1.3.6 The left heart

\[ p_{501} - p_e = \pm a_{LA} (V_{500} - b_{LA})^2 + (c_{LA} V_{500} - d_{LA}) f_{LA} \]  
(53)

\[ Q_{v,500} = Q_{H,500} - Q_{H,501} \]  
(54)

\[ p_{502} - p_{501} = R_{500} Q_{H,500} \]  
(55)

\[ p_{501} - p_{510} = R_{511} Q_{H,501} + L_{510} \frac{d}{dt} Q_{H,501} \]  
(56)

\[ V_{500}(t) = V_{500}(t = 0) + \int Q_{v,500} \, dt \]  
(57)

\[ p_{551}(V_{501}, t, Q_{H,600}) - p_e = \pm a_{LV} (V_{501} - b_{LV})^2 + \ldots \]  
(58)

\[ ... (c_{LV} V_{501} - d_{LV}) F_{LV}(t, Q_{H,14}) \]  

\[ F_{LV} = f_{LV} (t) - k_{LV} Q_{H,600}(t) + k_{LV} Q_{H,600}(t - \tau_{LV}) \]  
(59)

\[ p_{530} - p_{551} = R_{550} Q_{H,501} \]  
(60)

\[ p_{551} - p_{552} = R_{502} Q_{H,600} + L_{501} \frac{d}{dt} Q_{H,600} \]  
(61)

\[ Q_{v,501} = Q_{H,501} - Q_{H,600} \]  
(62)

\[ V_{501}(t) = V_{501}(t = 0) + \int Q_{v,501} \, dt \]  
(63)

\[ V_{d,LV} = \int Q_{H,600} \, dt \]  
(64)
1.3.7 The systemic arteries

\[ \begin{align*}
    p_{553} - p_{600} &= Z_{600}Q_{H600} \\
    p_{600} - p_{601} &= Z_{601}Q_{H601} \\
    Q_{H600} &= Q_{H700} + Q_{H601} \\
    Q_{V600} &= Q_{H601} - Q_{H602} \\
    Q_{V600} &= C_{600}\frac{d}{dt}(p_{601} - p_s) \\
    V_{600}(t) &= V_{600}(t = 0) + \int Q_{600}dt
\end{align*} \]

1.3.8 reduced (practical)

\[ Q_{H700}(t) = \frac{(p_{600} - p_{501})}{(R_{501} + R_{501}(t))} \] (71)

1.3.9 reduced (practical). In addition to the equations for the coronary circulation

\[ \begin{align*}
    Q_{H602} &= Q_{H605} + Q_{H606} + Q_{H603} + Q_{H604} \\
    Q_{H605} &= \frac{(p_{601} - p_1)}{R_{601}} \\
    Q_{H606} &= \frac{(p_{601} - p_2)}{R_{602}} \\
    Q_{H603} &= \frac{(p_{601} - p_{50})}{R_{600}} \\
    Q_{H604} &= \frac{(p_{601} - p_{51})}{R_{603}}
\end{align*} \]

1.3.10 Valves

\[ \begin{align*}
    \text{Tricuspid valve open: } p_{350} = p_{310} \text{ closed: } p_{350} = p_{352} \\
    \text{Pulmonary valve open: } p_{400} = p_{55} \text{ closed: } p_{400} = p_{401} \\
    \text{Mitra valve open: } p_{550} = p_{310} \text{ closed: } p_{550} = p_{551} \\
    \text{Aortic valve open: } p_{553} = p_{552} \text{ closed: } p_{553} = p_{600} \\
    \text{N valve open: } p_1 = p_3 \text{ closed: } p_1 = p_{601} \\
    \text{L valve open: } p_{52} = p_{55} \text{ closed: } p_{52} = p_{51}
\end{align*} \]
### 1.4 Volumes and pressures (including locations) DI

<table>
<thead>
<tr>
<th>#</th>
<th>Location</th>
<th>Ved</th>
<th>Ves</th>
<th>ped</th>
<th>pes</th>
</tr>
</thead>
<tbody>
<tr>
<td>250</td>
<td>Vena cava superior</td>
<td>598.2</td>
<td>598.7</td>
<td>-4.2179</td>
<td>-9.2675</td>
</tr>
<tr>
<td>50</td>
<td>Abdomen</td>
<td>937.5</td>
<td>909.8</td>
<td>11.7187</td>
<td>11.3729</td>
</tr>
<tr>
<td>200</td>
<td>intra-abdominal vena cava inferior</td>
<td>1580.9</td>
<td>1642.2</td>
<td>-2.8955</td>
<td>-7.515</td>
</tr>
<tr>
<td>300</td>
<td>Right atrium</td>
<td>44.5</td>
<td>54.8</td>
<td>-6.2831</td>
<td>-6.821</td>
</tr>
<tr>
<td>350</td>
<td>Right ventricle</td>
<td>128</td>
<td>54.1</td>
<td>-3.5923</td>
<td>3.5959</td>
</tr>
<tr>
<td>350</td>
<td>Pulmonary arteries</td>
<td>246.4</td>
<td>294.5</td>
<td>21.0055</td>
<td>22.8193</td>
</tr>
<tr>
<td>400</td>
<td>Pulmonary veins</td>
<td>449.8</td>
<td>460</td>
<td>-7.7617</td>
<td>-12.6735</td>
</tr>
<tr>
<td>500</td>
<td>Left Atrium</td>
<td>25.4</td>
<td>41.1</td>
<td>11.8924</td>
<td>-12.7175</td>
</tr>
<tr>
<td>501</td>
<td>Left Ventricle</td>
<td>98.2</td>
<td>23.9</td>
<td>-8.1102</td>
<td>34.7936</td>
</tr>
<tr>
<td>600</td>
<td>Aorta</td>
<td>203.5</td>
<td>255.8</td>
<td>87.559</td>
<td>108.6639</td>
</tr>
<tr>
<td>460</td>
<td>pulmonary periphery</td>
<td>254.1</td>
<td>254.1</td>
<td>-5.7167</td>
<td>-10.7759</td>
</tr>
</tbody>
</table>

#### 2 The Donders model (DII, chapter 8)

2.1 Representation of The Donders model (II) with annotations
2 The Donders model (DII, chapter 8)

2.1 Representation of The Donders model (II) with annotations
2.2 Equations for DII.

2.2.1 Systemic veins and their valves

\[ Q_{v1} = Q_{H605} + Q_{H606} - Q_{H250} \]  \hspace{1cm} (1)

\[ Q_{v1} = C_1 \frac{d}{dt} p_3 \]  \hspace{1cm} (2)

\[ Q_{V250} = Q_{H250} - Q_{H251} \]  \hspace{1cm} (3)

\[ Q_{V250} = C_{250} \frac{d}{dt} (p_{250} - p_v) \]  \hspace{1cm} (4)

\[ p_3 - p_{250} = \frac{1}{2} (L_{250} + L_2) \frac{d}{dt} Q_{H250} + \frac{1}{2} (R_2 + R_{250}) \]  \hspace{1cm} (5)

\[ p_{250} - p_{300} = \frac{1}{2} L_{250} Q_{H251} + \frac{1}{2} R_{250} Q_{H251} \]  \hspace{1cm} (6)

\[ p_{601} - p_3 = R_{601} Q_{H605} + \frac{1}{2} L_2 \frac{d}{dt} Q_{H605} \]  \hspace{1cm} (7)

\[ p_{601} - p_3 = R_{602} Q_{H606} + L_3 \frac{d}{dt} Q_{H606} + \frac{1}{2} L_2 \frac{d}{dt} Q_{H606} \]  \hspace{1cm} (8)

\[ p_{51} - p_{52} = L_{51} \frac{d}{dt} Q_{H51} + R_{51} Q_{H51} \]  \hspace{1cm} (9)

\[ Q_{V50} = C_{50} \frac{d}{dt} (p_{50} - p_v) \]  \hspace{1cm} (10)

\[ Q_{V50} = Q_{H605} - Q_{H50} \]  \hspace{1cm} (11)

\[ Q_{V51} = C_{51} \frac{d}{dt} (p_{51} - p_v) \]  \hspace{1cm} (12)

\[ Q_{V51} = Q_{H604} - Q_{H51} \]  \hspace{1cm} (13)

\[ p_{50} - p_{51} = L_{50} \frac{d}{dt} Q_{H50} + R_{50} Q_{H50} \]  \hspace{1cm} (14)

\[ p_{51} - p_{202} = R_{200} Q_{H200} + \frac{1}{2} L_{200} \frac{d}{dt} Q_{H200} + \frac{1}{2} R_{201} Q_{H200} \]  \hspace{1cm} (15)

\[ Q_{V200} = C_{200} \frac{d}{dt} (p_{202} - p_v) \]  \hspace{1cm} (16)

\[ Q_{V200} = Q_{H200} - Q_{H201} \]  \hspace{1cm} (17)

\[ Q_{H200} = Q_{H50} + Q_{H51} \]  \hspace{1cm} (18)

\[ p_{202} - p_{300} = \frac{1}{2} L_{200} \frac{d}{dt} Q_{H201} + \frac{1}{2} R_{201} Q_{H201} \]  \hspace{1cm} (19)

\[ V_{200}(t) = V_{200}(t = 0) + \int Q_{200} dt \]  \hspace{1cm} (20)

\[ V_{250}(t) = V_{250}(t = 0) + \int Q_{250} dt \]  \hspace{1cm} (21)

\[ V_{51}(t) = V_{51}(t = 0) + \int Q_{51} dt \]  \hspace{1cm} (22)
2.2.1 Systemic veins and their valves

\[ V_{50}(t) = V_{50}(t = 0) + \int Q_{V 50} dt \]
\[ V_i(t) = V_i(t = 0) + \int Q_{V i} dt \]

\[ p_{300} - p_{301} = R_{300} Q_{H 300} + L_{300} \frac{d}{dt} Q_{H 300} \]
\[ p_{301} - p_{310} = R_{310} Q_{H 301} + L_{310} \frac{d}{dt} Q_{H 301} \]
\[ Q_{V 300} = Q_{H 302} - Q_{H 301} \]
\[ Q_{V 350} = Q_{H 301} - Q_{H 400} \]
\[ p_{350} - p_{352} = R_{350} Q_{H 301} \]
\[ p_{301} - p_r = \pm d_R (V_{300} - b_R) + (c_{RA} V_{300} - d_{RA}) f_{RA} \]
\[ p_{352} (V_{350} - p_r) - p_r (t) = \pm d_{RV} (V_{350} - b_{RV}) + \ldots \]
\[ F_{RV} = f_{RV} (t) - k_{1RV} Q_{H 400} (t) + k_{2RV} Q_{H 400} (t - \tau_{RV}) \]
\[ p_{352} - p_{351} = R_{351} Q_{H 400} + L_{350} \frac{d}{dt} Q_{H 400} \]
\[ Q_{H 300} = Q_{H 251} + Q_{H 201} \]
\[ V_{300}(t) = V_{300}(t = 0) + \int (Q_{V 300}) dt \]
\[ V_{350}(t) = V_{350}(t = 0) + \int (Q_{V 350}) dt \]
\[ V_{350} = \int Q_{H 400} dt \]
\[ Q_{H 302} = Q_{H 300} + Q_{H 300} \]

2.2.2 The right heart

\[ p_{300} - p_{301} = R_{300} Q_{H 300} + L_{300} \frac{d}{dt} Q_{H 300} \]
\[ p_{301} - p_{310} = R_{310} Q_{H 301} + L_{310} \frac{d}{dt} Q_{H 301} \]
\[ Q_{V 300} = Q_{H 302} - Q_{H 301} \]
\[ Q_{V 350} = Q_{H 301} - Q_{H 400} \]
\[ p_{350} - p_{352} = R_{350} Q_{H 301} \]
\[ p_{301} - p_r = \pm d_R (V_{300} - b_R) + (c_{RA} V_{300} - d_{RA}) f_{RA} \]
\[ p_{352} (V_{350} - p_r) - p_r (t) = \pm d_{RV} (V_{350} - b_{RV}) + \ldots \]
\[ F_{RV} = f_{RV} (t) - k_{1RV} Q_{H 400} (t) + k_{2RV} Q_{H 400} (t - \tau_{RV}) \]
\[ p_{352} - p_{351} = R_{351} Q_{H 400} + L_{350} \frac{d}{dt} Q_{H 400} \]
\[ Q_{H 300} = Q_{H 251} + Q_{H 201} \]
\[ V_{300}(t) = V_{300}(t = 0) + \int (Q_{V 300}) dt \]
\[ V_{350}(t) = V_{350}(t = 0) + \int (Q_{V 350}) dt \]
\[ V_{350} = \int Q_{H 400} dt \]
\[ Q_{H 302} = Q_{H 300} + Q_{H 700} \]

2.2.3 Pulmonary arteries

\[ Q_{V 400} = Q_{H 400} - Q_{H 430} \]
\[ Q_{V 400} = C_{400} \frac{d}{dt} (p_{401} - p_r) \]
\[ p_{400} - p_{401} = Q_{H 400} Z_{400} + \frac{1}{2} R_{411} Q_{H 400} + \frac{1}{2} L_{410} \frac{d}{dt} Q_{H 400} \]
\[ p_{401} - p_{430} = \frac{1}{2} R_{411} Q_{H 430} + \frac{1}{2} L_{410} \frac{d}{dt} Q_{H 430} \]
\[ V_{400}(t) = V_{400}(t = 0) + \int Q_{V 400} dt \]

2.2.4 Pulmonary periphery

\[ Q_{V 430} = Q_{H 430} - Q_{H 460} \]
\[ Q_{V,430} = C_{401} \frac{d}{dt} (p_{431} - p_r) \]  
\[ p_{430} - p_{441} = R_{430} Q_{H,430} + \frac{1}{2} L_{440} \frac{d}{dt} Q_{H,430} + \frac{1}{2} R_{441} Q_{H,430} \]  
\[ p_{441} - p_{460} = \frac{1}{2} L_{440} \frac{d}{dt} Q_{H,430} + \frac{1}{2} R_{441} Q_{H,430} \]  
\[ V_{430}(t) = V_{430}(t = 0) + \int Q_{V,430} dt \]  

2.2.5 Pulmonary veins

\[ Q_{V,460} = Q_{H,460} - Q_{H,500} \]  
\[ Q_{V,460} = C_{460} \frac{d}{dt} (p_{462} - p_r) \]  
\[ p_{460} - p_{461} = R_{460} Q_{H,460} + \frac{1}{2} L_{460} \frac{d}{dt} Q_{H,460} + \frac{1}{2} R_{461} Q_{H,460} \]  
\[ p_{463} - p_{502} = \frac{1}{2} L_{460} \frac{d}{dt} Q_{H,500} + \frac{1}{2} R_{461} Q_{H,500} \]  
\[ V_{460}(t) = V_{460}(t = 0) + \int Q_{V,460} dt \]  

2.2.6 The left heart

\[ p_{501} - p_e = \pm a_{LA} (V_{500} - b_{LA})^2 + (c_{LA} V_{500} - d_{LA}) f_{LA} \]  
\[ Q_{V,500} = Q_{H,500} - Q_{H,501} \]  
\[ p_{502} - p_{501} = R_{500} Q_{H,500} \]  
\[ p_{501} - p_{510} = R_{510} Q_{H,501} + L_{510} \frac{d}{dt} Q_{H,501} \]  
\[ V_{500}(t) = V_{500}(t = 0) + \int Q_{V,500} dt \]  
\[ p_{551}(V_{501}, t, Q_{H,600}) - p_e(t) = \pm a_{LV} (V_{501} - b_{LV})^2 + ... \]  
\[ ... (c_{LV} V_{501} - d_{LV}) F_{LV} (t, Q_{H,51}) \]  
\[ F_{LV} = f_{LV} (t) - k_{1LV} Q_{H,600} (t) + k_{2LV} Q_{H,600} (t - \tau_{LV}) \]  
\[ p_{550} - p_{551} = R_{550} Q_{H,501} \]  
\[ p_{551} - p_{552} = R_{552} Q_{H,600} + L_{501} \frac{d}{dt} Q_{H,600} \]  
\[ Q_{V,501} = Q_{H,501} - Q_{H,600} \]  
\[ V_{501}(t) = V_{501}(t = 0) + \int Q_{V,501} dt \]  
\[ V_{LV} = \int Q_{H,600} dt \]
2.2.7 The systemic arteries

\[ p_{553} - p_{600} = Z_{600}Q_{H600} \]  
(66)

\[ p_{600} - p_{601} = Z_{601}Q_{H601} \]  
(67)

\[ Q_{H600} = Q_{H700} + Q_{H601} \]  
(68)

\[ Q_{V600} = Q_{H601} - Q_{H602} \]  
(69)

\[ Q_{V600} = C_{600} \frac{d}{dt}(p_{601} - p_{61}) \]  
(70)

\[ V_{600}(t) = V_{600}(t = 0) + \int Q_{V600} dt \]  
(71)

2.2.8 Coronary circulation

\[ Q_{H700}(t) = \frac{(p_{600} - p_{500})}{(R_{301} + R_{302}(t))} \]  
(72)

2.2.9 Equations of state

\[ Q_{H602} = Q_{H605} + Q_{H606} + Q_{H603} + Q_{H604} \]  
(73)

\[ Q_{H605} = \frac{(p_{601} - p_1)}{R_{601}} \]  
(74)

\[ Q_{H606} = \frac{(p_{601} - p_2)}{R_{602}} \]  
(75)

\[ Q_{H603} = \frac{(p_{601} - p_3)}{R_{600}} \]  
(76)

\[ Q_{H604} = \frac{(p_{601} - p_4)}{R_{603}} \]  
(77)

2.2.10 Valves

Tricuspid valve open: \( p_{550} = p_{310} \) closed: \( p_{550} = p_{552} \)  
(78)

Pulmonary valve open: \( p_{400} = p_{551} \) closed: \( p_{400} = p_{401} \)  
(79)

Mitrvalvalve open: \( p_{550} = p_{551} \) closed: \( p_{550} = p_{551} \)  
(80)

Aortic valve open: \( p_{553} = p_{552} \) closed: \( p_{553} = p_{600} \)  
(81)

Nvalve open: \( p_1 = p_3 \) closed: \( p_1 = p_{601} \)  
(82)

Lvalve open: \( p_{52} = p_{51} \) closed: \( p_{52} = p_{51} \)  
(83)
### Parameter values and variables DII

<table>
<thead>
<tr>
<th>#</th>
<th>Description</th>
<th>Ved</th>
<th>Ves</th>
<th>ped</th>
<th>pes</th>
</tr>
</thead>
<tbody>
<tr>
<td>250</td>
<td>Vena cava superior</td>
<td>687.4</td>
<td>689.4</td>
<td>-2.73</td>
<td>-8.50</td>
</tr>
<tr>
<td>50</td>
<td>Abdomen</td>
<td>466.8</td>
<td>450.8</td>
<td>5.83</td>
<td>5.63</td>
</tr>
<tr>
<td>200</td>
<td>intra-abdominal vena cava inferior</td>
<td>1066.7</td>
<td>1092.7</td>
<td>-6.57</td>
<td>-12.19</td>
</tr>
<tr>
<td>300</td>
<td>Right atrium</td>
<td>37.5</td>
<td>47.4</td>
<td>-8.81</td>
<td>-11.04</td>
</tr>
<tr>
<td>350</td>
<td>Right ventricle</td>
<td>111</td>
<td>94.7</td>
<td>-6.32</td>
<td>77.92</td>
</tr>
<tr>
<td>350</td>
<td>Pulmonary arteries</td>
<td>243.7</td>
<td>245.3</td>
<td>20.63</td>
<td>15.05</td>
</tr>
<tr>
<td>400</td>
<td>Pulmonary veins</td>
<td>404</td>
<td>400.7</td>
<td>-8.42</td>
<td>-14.27</td>
</tr>
<tr>
<td>500</td>
<td>Left Atrium</td>
<td>14.7</td>
<td>34</td>
<td>-13.60</td>
<td>-15.81</td>
</tr>
<tr>
<td>501</td>
<td>Left Ventricle</td>
<td>74.2</td>
<td>12.7</td>
<td>-10.84</td>
<td>59.35</td>
</tr>
<tr>
<td>600</td>
<td>Aorta</td>
<td>91.4</td>
<td>145.5</td>
<td>31.49</td>
<td>52.73</td>
</tr>
<tr>
<td>51</td>
<td>Upper systemic return</td>
<td>1503.7</td>
<td>1495.1</td>
<td>30.07</td>
<td>29.90</td>
</tr>
<tr>
<td>51</td>
<td>Legs</td>
<td>266.2</td>
<td>260.2</td>
<td>5.32</td>
<td>5.20</td>
</tr>
<tr>
<td>460</td>
<td>pulmonary periphery</td>
<td>271.3</td>
<td>270.1</td>
<td>-5.15</td>
<td>-10.99</td>
</tr>
</tbody>
</table>
3 The Donders model (DIII, chapter 9)

3.1 Representation of The Donders Model (DIII) with annotations

3.2 Equations for DIII

3.2.1 The Right Heart

\[ P_{300} - P_e = \pm a_{RA}(V_{300} - B_{RA})^2 + (c_{RA}V_{300} - d_{RA})f_{RA} \]  
\[ B_{RA} = b_{RA} + \frac{1}{3} \int_0^t Q_{H350} dt \]  
\[ p_{350}(V_{350}, t, Q_{H350}) - p_e(t) = \pm a_{RV}(V_{350} - b_{RV})^2 + \ldots \]  
\[ (c_{RV}V_{350} - d_{RV})F_{RV}(t, Q_{H350}) \]  
\[ F_{RV} = f_{RV}(t) - k_{1RV}Q_{H350}(t) + k_{2RV}Q_{H350}(t - \tau_{RV}) \]  
\[ P_{300} - P_{302} = R_{300}Q_{H300} + L_{300} \frac{d}{dt} Q_{H300} \]  
\[ P_{302} - P_{350} = R_{302}Q_{H300} \]
\[ P_{350} - P_{352} = R_{350}Q_{H350} + L_{350} \frac{d}{dt} Q_{H350} \]  \hspace{1cm} (7)

\[ P_{352} - P_{400} = R_{352}Q_{H350} + \frac{1}{2} L_{400} \frac{d}{dt} Q_{H350} + \frac{1}{2} R_{400}Q_{H350} \]  \hspace{1cm} (8)

\[ Q_{V300} = Q_{H299} - Q_{H300} \]  \hspace{1cm} (9)

\[ Q_{V350} = Q_{H300} - Q_{H350} \]  \hspace{1cm} (10)

\[ Q_{H299} = Q_{H700} + Q_{H250} \]  \hspace{1cm} (11)

\[ V_{300}(t) = V_{300}(0) + \int_{0}^{t} (Q_{V300}) dt \]  \hspace{1cm} (12)

\[ V_{350}(t) = V_{350}(0) + \int_{0}^{t} (Q_{V350}) dt \]  \hspace{1cm} (13)

3.2.2 Pulmonary arteries

\[ Q_{V400} = Q_{H350} - Q_{H400} \]  \hspace{1cm} (14)

\[ Q_{V400} = C_{400} \frac{d}{dt} (P_{400} - P_{v}) \]  \hspace{1cm} (15)

\[ P_{400} - P_{401} = \frac{1}{2} R_{400}Q_{H400} + \frac{1}{2} L_{400} \frac{d}{dt} Q_{H400} \]  \hspace{1cm} (16)

\[ P_{401} - P_{430} = R_{401}Q_{H400} + \frac{1}{2} R_{430}Q_{H400} + \frac{1}{2} L_{430} \frac{d}{dt} Q_{H400} \]  \hspace{1cm} (17)

\[ V_{400}(t) = V_{400}(0) + \int_{0}^{t} (Q_{V400}) dt \]  \hspace{1cm} (18)
3.2.3 Pulmonary periphery

\[ \begin{align*}
Q_{V_{430}} &= Q_{H_{400}} - Q_{H_{430}} \\
Q_{V_{430}} &= C_{430} \frac{d}{dt} (p_{430} - p_r) \\
p_{430} - p_{431} &= \frac{1}{2} R_{430} Q_{H_{430}} + \frac{1}{2} L_{430} \frac{d}{dt} Q_{H_{430}} \\
p_{431} - p_{460} &= R_{431} Q_{H_{430}} + \frac{1}{2} R_{460} Q_{H_{430}} + \frac{1}{2} L_{460} \frac{d}{dt} Q_{H_{430}} \\
V_{430}(t) &= V_{430}(0) + \int_0^t Q_{V_{430}} dt 
\end{align*} \]

(19) (20) (21) (22) (23)

3.2.4 Pulmonary veins

\[ \begin{align*}
Q_{V_{460}} &= Q_{H_{430}} - Q_{H_{460}} \\
Q_{V_{460}} &= C_{460} \frac{d}{dt} (p_{460} - p_r) \\
p_{460} - p_{461} &= \frac{1}{2} R_{460} Q_{H_{460}} + \frac{1}{2} L_{460} \frac{d}{dt} Q_{H_{460}} \\
p_{461} - p_{500} &= R_{461} Q_{H_{460}} \\
V_{460}(t) &= V_{460}(0) + \int_0^t Q_{V_{460}} dt 
\end{align*} \]

(24) (25) (26) (27) (28)

3.2.5 The left heart

\[ \begin{align*}
p_{500} - p_r &= \pm a_{LA} (V_{500} - B_{LA})^2 + (c_{LA} V_{500} - d_{LA}) f_{LA} \\
B_{LA} &= b_{LA} + \frac{1}{3} \int_0^t Q_{H_{550}} dt \\
p_{550} (V_{501}, t, Q_{H_{500}}) - p_r(t) &= \pm a_{LV} (V_{500} - b_{LV})^2 + \ldots \\
&\ldots (c_{LV} V_{500} - d_{LV}) F_{LV}(t, Q_{H_{500}}) \\
F_{LV} &= f_{LV} (t) - k_{1LV} Q_{H_{500}} (t) + k_{2LV} Q_{H_{500}} (t - \tau_{LV}) \\
p_{500} - p_{502} &= R_{500} Q_{H_{500}} + L_{500} \frac{d}{dt} Q_{H_{500}} \\
p_{502} - p_{550} &= R_{502} Q_{H_{500}} \\
p_{550} - p_{552} &= R_{550} Q_{H_{550}} + L_{550} \frac{d}{dt} Q_{H_{550}} \\
Q_{H_{500}} &= Q_{H_{460}} - Q_{H_{500}} \\
V_{500}(t) &= V_{500}(0) + \int_0^t Q_{V_{500}} dt 
\end{align*} \]

(29) (30) (31) (32) (33) (34) (35) (36) (37)
\[ Q_{V550} = Q_{H500} - Q_{V550} \]  \hspace{1cm} (38)

\[ V_{550}(t) = V_{550}(0) + \int_0^t Q_{V550} \, dt \] \hspace{1cm} (39)

### 3.2.6 The systemic arteries

\[ p_{552} - p_{599} = Z_{552}Q_{H550} \] \hspace{1cm} (40)

\[ p_{599} - p_{600} = Z_{600}Q_{H599} + \frac{1}{2} L_{600} \frac{d}{dt} Q_{H599} \] \hspace{1cm} (41)

\[ Q_{V600} = Q_{H599} - Q_{H600} \] \hspace{1cm} (42)

\[ Q_{V600} = C_{600} \frac{d}{dt} (p_{600} - p_v) \] \hspace{1cm} (43)

\[ V_{600}(t) = V_{600}(0) + \int_0^t Q_{V600} \, dt \] \hspace{1cm} (44)

\[ Q_{H550} = Q_{H700} + Q_{H599} \] \hspace{1cm} (45)

\[ Q_{H600} = Q_{H744} + Q_{H745} + Q_{H746} + Q_{H747} + Q_{H748} + Q_{H749} + Q_{H799} + Q_{H899} \] \hspace{1cm} (46)

\[ Q_{H700}(t) = \frac{(p_{599} - p_{501})}{(R_{700} + R_{701}(t))} \] \hspace{1cm} (47)

\[ (p_{600} - p_{601}) = R_{6001}Q_{H744} + \frac{1}{2} R_{6001}Q_{H745} + \frac{1}{2} L_{601} \frac{d}{dt} Q_{H744} \] \hspace{1cm} (48)

\[ (p_{600} - p_{602}) = R_{6002}Q_{H745} + \frac{1}{2} R_{6002}Q_{H746} + \frac{1}{2} L_{602} \frac{d}{dt} Q_{H745} \] \hspace{1cm} (49)

\[ (p_{600} - p_{603}) = R_{6003}Q_{H746} + \frac{1}{2} R_{6003}Q_{H747} + \frac{1}{2} L_{603} \frac{d}{dt} Q_{H746} \] \hspace{1cm} (50)

\[ (p_{600} - p_{604}) = R_{6004}Q_{H747} + \frac{1}{2} R_{6004}Q_{H748} + \frac{1}{2} L_{604} \frac{d}{dt} Q_{H747} \] \hspace{1cm} (51)

### 3.2.7 Legs

\[ p_{800} - p_{800} = (R_{799} + \frac{1}{2} R_{800})Q_{H799} + \frac{1}{2} L_{800} \frac{d}{dt} Q_{H799} \] \hspace{1cm} (52)

\[ Q_{V800} = Q_{H799} - Q_{H800} \] \hspace{1cm} (53)

\[ Q_{V800} = C_{800} \frac{d}{dt} (p_{800} - p_v) \] \hspace{1cm} (54)

\[ V_{800}(t) = V_{800}(0) + \int_0^t Q_{V800} \, dt \] \hspace{1cm} (55)

\[ (p_{800} - p_{830}) = \frac{1}{2} L_{800} \frac{d}{dt} Q_{H800} + \frac{1}{2} R_{800}Q_{H800} + \frac{1}{2} L_{830} \frac{d}{dt} Q_{H800} + \frac{1}{2} R_{830}Q_{H800} \] \hspace{1cm} (56)

\[ Q_{V830} = Q_{H800} - Q_{H830} \] \hspace{1cm} (57)
\[ Q_{V30} = C_{V30} \frac{d}{dt} (p_{30} - p_s) \]  
(58)

\[ V_{30}(t) = V_{30}(0) + \int_{0}^{t} Q_{V30} dt \]  
(59)

\[ p_{30} - p_{960} = \frac{1}{2} L_{30} \frac{d}{dt} Q_{H30} + \frac{1}{2} R_{30} Q_{H30} + \frac{1}{2} L_{960} \frac{d}{dt} Q_{H960} + \frac{1}{2} R_{960} Q_{H960} \]  
(60)

\[ Q_{V30} = Q_{H30} - Q_{H60} \]  
(61)

\[ Q_{V30} = C_{V30} \frac{d}{dt} (p_{30} - p_s) \]  
(62)

\[ V_{960}(t) = V_{960}(0) + \int_{0}^{t} Q_{V960} dt \]  
(63)

\[ p_{960} - p_{970} = \frac{1}{2} L_{960} \frac{d}{dt} Q_{H960} + \frac{1}{2} R_{960} Q_{H960} + \frac{1}{2} L_{970} \frac{d}{dt} Q_{H970} + \frac{1}{2} R_{970} Q_{H970} \]  
(64)

\[ Q_{V900} = Q_{H999} - Q_{H900} \]  
(65)

\[ Q_{V900} = C_{900} \frac{d}{dt} (p_{900} - p_s) \]  
(66)

\[ V_{990}(t) = V_{990}(0) + \int_{0}^{t} Q_{V990} dt \]  
(67)

\[ p_{900} - p_{930} = \frac{1}{2} L_{900} \frac{d}{dt} Q_{H900} + \frac{1}{2} R_{900} Q_{H900} + \frac{1}{2} L_{930} \frac{d}{dt} Q_{H930} + \frac{1}{2} R_{930} Q_{H930} \]  
(68)

\[ Q_{V930} = Q_{H930} - Q_{H900} \]  
(69)

\[ Q_{V930} = C_{930} \frac{d}{dt} (p_{930} - p_s) \]  
(70)

\[ V_{930}(t) = V_{930}(0) + \int_{0}^{t} Q_{V930} dt \]  
(71)

\[ p_{930} - p_{960} = \frac{1}{2} L_{930} \frac{d}{dt} Q_{H930} + \frac{1}{2} R_{930} Q_{H930} + \frac{1}{2} L_{960} \frac{d}{dt} Q_{H960} + \frac{1}{2} R_{960} Q_{H960} \]  
(72)

\[ Q_{V960} = Q_{H960} - Q_{H930} \]  
(73)

\[ Q_{V960} = C_{960} \frac{d}{dt} (p_{960} - p_s) \]  
(74)

\[ V_{960}(t) = V_{960}(0) + \int_{0}^{t} Q_{V960} dt \]  
(75)

\[ p_{960} - p_{970} = \frac{1}{2} L_{960} \frac{d}{dt} Q_{H960} + \frac{1}{2} R_{960} Q_{H960} + \frac{1}{2} L_{970} \frac{d}{dt} Q_{H970} + \frac{1}{2} R_{970} Q_{H970} \]  
(76)

\[ Q_{V970} = Q_{H970} + Q_{H960} \]  
(77)

\[ Q_{V970} = C_{970} \frac{d}{dt} (p_{970} - p_s) \]  
(78)
3.2.8 Abdomen and Kidneys

\[ P_{650} - P_{650} = (R_{649} + \frac{1}{2} R_{650}) Q_{H649} + \frac{1}{2} L_{650} \frac{d}{dt} Q_{H649} \]  (79)

\[ Q_{V650} = Q_{H649} - Q_{H650} \]  (80)

\[ Q_{V650} = C_{650} \frac{d}{dt} (P_{650} - P_e) \]  (81)

\[ V_{650}(t) = V_{650}(0) + \int_0^t Q_{V650} dt \]  (82)

\[ P_{750} - P_{750} = (R_{749} + \frac{1}{2} R_{750}) Q_{H749} + \frac{1}{2} L_{750} \frac{d}{dt} Q_{H749} \]  (83)

\[ Q_{V750} = Q_{H749} - Q_{H750} \]  (84)

\[ Q_{V750} = C_{750} \frac{d}{dt} (P_{750} - P_e) \]  (85)

\[ V_{750}(t) = V_{750}(0) + \int_0^t Q_{V750} dt \]  (86)

\[ Q_{H989} = Q_{H750} + Q_{H980} \]  (87)

\[ Q_{V990} = Q_{H750} + Q_{H980} - Q_{H990} \]  (88)

\[ Q_{V990} = C_{990} \frac{d}{dt} (P_{990} - P_e) \]  (89)

\[ V_{990}(t) = V_{990}(0) + \int_0^t Q_{V990} dt \]  (90)

\[ P_{990} - P_{1000} = \frac{1}{2} L_{990} \frac{d}{dt} Q_{H990} + \frac{1}{2} R_{990} Q_{H990} + \frac{1}{2} L_{1000} \frac{d}{dt} Q_{H1000} + \frac{1}{2} R_{1000} Q_{H1000} \]  (91)

3.2.9 VCI

\[ Q_{V970} = Q_{H969} - Q_{H970} \]  (92)

\[ Q_{V970} = C_{970} \frac{d}{dt} (P_{970} - P_e) \]  (93)

\[ V_{970}(t) = V_{970}(0) + \int_0^t Q_{V970} dt \]  (94)

\[ P_{970} - P_{980} = \frac{1}{2} L_{970} \frac{d}{dt} Q_{H970} + \frac{1}{2} R_{970} Q_{H970} + \frac{1}{2} L_{980} \frac{d}{dt} Q_{H970} + \frac{1}{2} R_{980} Q_{H970} \]  (95)

\[ Q_{V980} = Q_{H970} - Q_{H980} \]  (96)

\[ Q_{V980} = C_{980} \frac{d}{dt} (P_{980} - P_e) \]  (97)
\[ V_{990}(t) = V_{990}(0) + \int_0^t Q_{990} \, dt \]  
(98)

\[ Q_{v1000} = Q_{H950} + Q_{H1000} - Q_{H1000} \]  
(99)

\[ Q_{v1000} = C_{1000} \frac{d}{dt} (p_{1000} - p_e) \]  
(100)

\[ V_{1000}(t) = V_{1000}(0) + \int_0^t Q_{1000} \, dt \]  
(101)

\[ p_{1000} - p_{250} = \frac{1}{2} L_{1000} \frac{d}{dt} Q_{H1000} + \frac{1}{2} R_{1000} Q_{H1000} \]  
(102)

\[ Q_{H250} = Q_{H1000} + Q_{H200} \]  
(103)

\[ p_{250} - p_{300} = R_{250} Q_{H250} + L_{250} \frac{d}{dt} Q_{H250} \]  
(104)

### 3.2.9 VCI

\[ Q_{v001} = Q_{H744} - Q_{H001} \]  
(105)

\[ Q_{v001} = C_{001} \frac{d}{dt} (p_{001} - p_e) \]  
(106)

\[ V_{001}(t) = V_{001}(0) + \int_0^t Q_{001} \, dt \]  
(107)

\[ p_{001} - p_{011} = \frac{1}{2} L_{001} \frac{d}{dt} Q_{H001} + \frac{1}{2} R_{001} Q_{H001} + \frac{1}{2} L_{011} \frac{d}{dt} Q_{H001} + \frac{1}{2} R_{011} Q_{H001} \]  
(108)

\[ Q_{v011} = Q_{H001} - Q_{H001} \]  
(109)

\[ Q_{v011} = C_{011} \frac{d}{dt} (p_{011} - p_e) \]  
(110)

\[ V_{011}(t) = V_{011}(0) + \int_0^t Q_{011} \, dt \]  
(111)

\[ p_{011} - p_{100} = \frac{1}{2} L_{011} \frac{d}{dt} Q_{H100} + \frac{1}{2} R_{011} Q_{H100} + \frac{1}{2} L_{100} \frac{d}{dt} Q_{H100} + \frac{1}{2} R_{100} Q_{H100} \]  
(112)

\[ Q_{v002} = Q_{H745} - Q_{H002} \]  
(113)

\[ Q_{v002} = C_{002} \frac{d}{dt} (p_{002} - p_e) \]  
(114)

\[ V_{002}(t) = V_{002}(0) + \int_0^t Q_{002} \, dt \]  
(115)

\[ p_{002} - p_{012} = \frac{1}{2} L_{002} \frac{d}{dt} Q_{H002} + \frac{1}{2} R_{002} Q_{H002} + \frac{1}{2} L_{012} \frac{d}{dt} Q_{H002} + \frac{1}{2} R_{012} Q_{H002} \]  
(116)

\[ Q_{v012} = Q_{H002} - Q_{H012} \]  
(117)

\[ Q_{v012} = C_{012} \frac{d}{dt} (p_{012} - p_e) \]  
(118)
\[ V_{012}(t) = V_{012}(0) + \int_0^t Q_{012} dt \] (119)

\[ P_{012} - P_{100} = \frac{1}{2} L_{012} \frac{d}{dt} Q_{H012} + \frac{1}{2} R_{012} Q_{H012} + \frac{1}{2} L_{100} \frac{d}{dt} Q_{H012} + \frac{1}{2} R_{100} Q_{H012} \] (120)

\[ Q_{H099} = Q_{H011} + Q_{H012} \] (121)

\[ Q_{V100} = Q_{H099} - Q_{H100} \] (122)

\[ Q_{V100} = C_{100} \frac{d}{dt} (p_{100} - p_r) \] (123)

\[ V_{100}(t) = V_{100}(0) + \int_0^t Q_{100} dt \] (124)

\[ Q_{V003} = Q_{H746} - Q_{H003} \] (125)

\[ Q_{V003} = C_{003} \frac{d}{dt} (p_{003} - p_r) \] (126)

\[ V_{003}(t) = V_{003}(0) + \int_0^t Q_{003} dt \] (127)

\[ P_{003} - P_{013} = \frac{1}{2} L_{003} \frac{d}{dt} Q_{H003} + \frac{1}{2} R_{003} Q_{H003} + \frac{1}{2} L_{013} \frac{d}{dt} Q_{H003} + \frac{1}{2} R_{013} Q_{H003} \] (128)

\[ Q_{V013} = Q_{H003} - Q_{H103} \] (129)

\[ Q_{V013} = C_{013} \frac{d}{dt} (p_{013} - p_r) \] (130)

\[ V_{013}(t) = V_{013}(0) + \int_0^t Q_{013} dt \] (131)

\[ P_{013} - P_{125} = \frac{1}{2} L_{013} \frac{d}{dt} Q_{H103} + \frac{1}{2} R_{013} Q_{H103} + \frac{1}{2} L_{125} \frac{d}{dt} Q_{H103} + \frac{1}{2} R_{125} Q_{H103} \] (132)

\[ Q_{V004} = Q_{H747} - Q_{H004} \] (133)

\[ Q_{V004} = C_{004} \frac{d}{dt} (p_{004} - p_r) \] (134)

\[ V_{004}(t) = V_{004}(0) + \int_0^t Q_{004} dt \] (135)

\[ P_{004} - P_{014} = \frac{1}{2} L_{004} \frac{d}{dt} Q_{H004} + \frac{1}{2} R_{004} Q_{H004} + \frac{1}{2} L_{014} \frac{d}{dt} Q_{H004} + \frac{1}{2} R_{014} Q_{H004} \] (136)

\[ Q_{V014} = Q_{H004} - Q_{H104} \] (137)

\[ Q_{V014} = C_{014} \frac{d}{dt} (p_{014} - p_r) \] (138)

\[ V_{014}(t) = V_{014}(0) + \int_0^t Q_{014} dt \] (139)

\[ P_{014} - P_{125} = \frac{1}{2} L_{014} \frac{d}{dt} Q_{H104} + \frac{1}{2} R_{014} Q_{H104} + \frac{1}{2} L_{125} \frac{d}{dt} Q_{H104} + \frac{1}{2} R_{125} Q_{H104} \] (140)

3.2.11 Valves

T valve open: 301302 = \text{if} T valve closed: 350302 = \text{p p}

P valve open: 351352 = \text{if} P valve closed: 400352 = \text{p p}

M valve open: 501502 = \text{if} M valve closed: 550502 = \text{p p}

A valve open: 551552 = \text{if} A valve closed: 559552 = \text{p p}

L valve open: 960960960960960969 = \text{if} L valve closed: 960960960960960969 = \text{p p}

N1 valve open: )\text{if} N1 valve closed: 011011011099 = \text{H Q dt dL p p +−}


\[ Q_{H124} = Q_{013} + Q_{H014} \]  
(141)

\[ Q_{V125} = Q_{H124} - Q_{H125} \]  
(142)

\[ Q_{V125} = C_{125} \frac{d}{dt} (p_{125} - p_e) \]  
(143)

\[ V_{125}(t) = V_{125}(0) + \int_{0}^{t} Q_{V125} dt \]  
(144)

\[ Q_{H149} = Q_{H100} + Q_{H125} \]  
(145)

\[ \frac{p_{100} - p_{150}}{2} = \frac{1}{2} L_{100} \frac{d}{dt} Q_{H100} + \frac{1}{2} R_{100} Q_{H100} + \frac{1}{2} L_{150} \frac{d}{dt} Q_{H150} + \frac{1}{2} R_{150} Q_{H150} + \frac{1}{2} R_{150} Q_{H125} \]  
(146)

\[ \frac{p_{125} - p_{150}}{2} = \frac{1}{2} L_{125} \frac{d}{dt} Q_{H125} + \frac{1}{2} R_{125} Q_{H125} + \frac{1}{2} L_{150} \frac{d}{dt} Q_{H150} + \frac{1}{2} R_{150} Q_{H150} + \frac{1}{2} R_{150} Q_{H125} \]  
(147)

\[ Q_{V150} = Q_{H149} - Q_{H150} \]  
(148)

\[ Q_{V150} = C_{150} \frac{d}{dt} (p_{150} - p_e) \]  
(149)

\[ V_{150}(t) = V_{150}(0) + \int_{0}^{t} Q_{V150} dt \]  
(150)

\[ \frac{p_{150} - p_{200}}{2} = \frac{1}{2} L_{150} \frac{d}{dt} Q_{H150} + \frac{1}{2} R_{150} Q_{H150} + \frac{1}{2} L_{200} \frac{d}{dt} Q_{H200} + \frac{1}{2} R_{200} Q_{H200} \]  
(151)

\[ Q_{V200} = Q_{H150} - Q_{H100} \]  
(152)

\[ Q_{V200} = C_{200} \frac{d}{dt} (p_{200} - p_e) \]  
(153)

\[ V_{200}(t) = V_{200}(0) + \int_{0}^{t} Q_{V200} dt \]  
(154)

\[ \frac{p_{200} - p_{250}}{2} = \frac{1}{2} L_{200} \frac{d}{dt} Q_{H200} + \frac{1}{2} R_{200} Q_{H200} \]  
(155)

### 3.2.11 Valves

T valve open: \( p_{302} = p_{301} \) if T valve closed: \( p_{302} = p_{350} \)

P valve open: \( p_{352} = p_{351} \) if P valve closed: \( p_{352} = p_{400} \)

M valve open: \( p_{502} = p_{501} \) if M valve closed: \( p_{502} = p_{550} \)

A valve open: \( p_{552} = p_{551} \) if A valve closed: \( p_{552} = p_{559} \)

L valve open: \( p_{969} = p_{960} - \frac{1}{2} L_{960} \frac{d}{dt} Q_{H960} - \frac{1}{2} R_{960} Q_{H960} \) if L valve closed:

\[ p_{960} = p_{960} \]

N1 valve open: \( p_{99} = p_{100} + \frac{1}{2} R_{100} (Q_{H011} + Q_{H012}) \) if N1 valve closed:

\[ p_{99} = p_{99} - \frac{1}{2} L_{991} \frac{d}{dt} Q_{H011} \]
N2 valve open:  
\[ p_{125} = p_{124} + \frac{1}{2} R_{125} (Q_{H012} + Q_{H014}) \]
if N2 valve closed:

\[ p_{124} = p_{013} - \frac{1}{2} L_{013} \frac{d}{dt} Q_{H013} \]

### 3.2.12 Parameter and variable values DIII

<table>
<thead>
<tr>
<th>comp</th>
<th>Ved [mL]</th>
<th>Ves [mL]</th>
<th>Ped[mmHg]</th>
<th>Pes[mmHg]</th>
</tr>
</thead>
<tbody>
<tr>
<td>001 R.jugular</td>
<td>232.18</td>
<td>232.25</td>
<td>46.44</td>
<td>46.45</td>
</tr>
<tr>
<td>002 R.subclavian</td>
<td>462.26</td>
<td>462.50</td>
<td>46.23</td>
<td>46.25</td>
</tr>
<tr>
<td>003 L.jugular</td>
<td>228.68</td>
<td>228.74</td>
<td>45.74</td>
<td>45.75</td>
</tr>
<tr>
<td>004 L.subclavian</td>
<td>481.30</td>
<td>481.52</td>
<td>48.13</td>
<td>48.15</td>
</tr>
<tr>
<td>011 R.jugular</td>
<td>26.32</td>
<td>26.33</td>
<td>30.21</td>
<td>30.22</td>
</tr>
<tr>
<td>012 R.subclavian</td>
<td>30.61</td>
<td>30.63</td>
<td>30.70</td>
<td>30.72</td>
</tr>
<tr>
<td>013 L.jugular</td>
<td>25.39</td>
<td>25.40</td>
<td>29.14</td>
<td>29.15</td>
</tr>
<tr>
<td>014 L.subclavian</td>
<td>30.59</td>
<td>30.60</td>
<td>30.68</td>
<td>30.69</td>
</tr>
<tr>
<td>100 R.innominate</td>
<td>26.58</td>
<td>26.58</td>
<td>18.64</td>
<td>18.65</td>
</tr>
<tr>
<td>125 L.innominate</td>
<td>25.52</td>
<td>25.52</td>
<td>17.90</td>
<td>17.90</td>
</tr>
<tr>
<td>150 VSC</td>
<td>310.23</td>
<td>309.99</td>
<td>10.34</td>
<td>10.33</td>
</tr>
<tr>
<td>200 VCS</td>
<td>266.19</td>
<td>272.48</td>
<td>8.87</td>
<td>9.08</td>
</tr>
<tr>
<td>300 RA</td>
<td>40.14</td>
<td>58.94</td>
<td>6.28</td>
<td>14.55</td>
</tr>
<tr>
<td>350 RV</td>
<td>112.17</td>
<td>47.31</td>
<td>8.04</td>
<td>19.73</td>
</tr>
<tr>
<td>400 pulm.arteries</td>
<td>164.10</td>
<td>206.52</td>
<td>23.44</td>
<td>29.50</td>
</tr>
<tr>
<td>430 pulm.periphery</td>
<td>216.04</td>
<td>215.20</td>
<td>7.20</td>
<td>7.17</td>
</tr>
<tr>
<td>460 pulm.veins</td>
<td>260.60</td>
<td>255.33</td>
<td>5.21</td>
<td>5.11</td>
</tr>
<tr>
<td>500 LA</td>
<td>16.59</td>
<td>45.15</td>
<td>0.81</td>
<td>8.06</td>
</tr>
<tr>
<td>550 LV</td>
<td>82.18</td>
<td>17.08</td>
<td>4.17</td>
<td>38.52</td>
</tr>
<tr>
<td>600 aorta</td>
<td>178.78</td>
<td>221.74</td>
<td>89.39</td>
<td>110.87</td>
</tr>
<tr>
<td>650 abdominal</td>
<td>749.06</td>
<td>749.00</td>
<td>8.49</td>
<td>8.49</td>
</tr>
<tr>
<td>750 renal</td>
<td>545.92</td>
<td>546.02</td>
<td>8.84</td>
<td>8.84</td>
</tr>
<tr>
<td>800 R.femoralis</td>
<td>91.07</td>
<td>91.10</td>
<td>9.11</td>
<td>9.11</td>
</tr>
<tr>
<td>830 R.femoralis</td>
<td>91.00</td>
<td>91.02</td>
<td>9.10</td>
<td>9.10</td>
</tr>
<tr>
<td>860 R.iliac</td>
<td>90.60</td>
<td>90.58</td>
<td>9.06</td>
<td>9.06</td>
</tr>
<tr>
<td>900 L.femoralis</td>
<td>91.07</td>
<td>91.10</td>
<td>9.11</td>
<td>9.11</td>
</tr>
<tr>
<td>930 L.femoralis</td>
<td>91.00</td>
<td>91.02</td>
<td>9.10</td>
<td>9.10</td>
</tr>
<tr>
<td>960 L.iliac</td>
<td>90.60</td>
<td>90.58</td>
<td>9.06</td>
<td>9.06</td>
</tr>
<tr>
<td>970 VCI</td>
<td>336.92</td>
<td>336.79</td>
<td>8.98</td>
<td>8.98</td>
</tr>
<tr>
<td>980 VCI</td>
<td>331.11</td>
<td>331.03</td>
<td>8.83</td>
<td>8.83</td>
</tr>
<tr>
<td>990 VCI</td>
<td>325.33</td>
<td>325.10</td>
<td>8.68</td>
<td>8.67</td>
</tr>
<tr>
<td>1000 VCI</td>
<td>315.87</td>
<td>312.82</td>
<td>8.42</td>
<td>8.34</td>
</tr>
</tbody>
</table>

Total Volume=6365.99mL
### 3.2.13 Overview of values in DIII

**Impedances:**

<table>
<thead>
<tr>
<th>Impedance</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>R.jugular L001</td>
<td>0.032 g/cm³</td>
</tr>
<tr>
<td>R.subclavian L002</td>
<td>0 g/cm³</td>
</tr>
<tr>
<td>L.jugular L003</td>
<td>0.032 g/cm³</td>
</tr>
<tr>
<td>L.subclavian L004</td>
<td>0 g/cm³</td>
</tr>
<tr>
<td>R.jugular L011</td>
<td>0.032 g/cm³</td>
</tr>
<tr>
<td>R.subclavian L012</td>
<td>0 g/cm³</td>
</tr>
<tr>
<td>L.jugular L013</td>
<td>0.032 g/cm³</td>
</tr>
<tr>
<td>L.subclavian L014</td>
<td>0 g/cm³</td>
</tr>
<tr>
<td>R.innominate L100</td>
<td>0.0005 g/cm³</td>
</tr>
<tr>
<td>L.innominate L125</td>
<td>0.0005 g/cm³</td>
</tr>
<tr>
<td>VCS L150</td>
<td>0.18712 g/cm³</td>
</tr>
<tr>
<td>RA L250</td>
<td>0 g/cm³</td>
</tr>
<tr>
<td>RA L300</td>
<td>0 g/cm³</td>
</tr>
<tr>
<td>RV L350</td>
<td>0.0005 g/cm³</td>
</tr>
<tr>
<td>pulm.arteries L400</td>
<td>0 g/cm³</td>
</tr>
<tr>
<td>pulm.periphery L430</td>
<td>0.0003 g/cm³</td>
</tr>
<tr>
<td>pulm.veins L460</td>
<td>0.0295 g/cm³</td>
</tr>
<tr>
<td>LA L500</td>
<td>0.001 g/cm³</td>
</tr>
<tr>
<td>LV L550</td>
<td>0.001 g/cm³</td>
</tr>
<tr>
<td>aorta L650</td>
<td>0.0003 g/cm³</td>
</tr>
<tr>
<td>renal L600</td>
<td>0.0003 g/cm³</td>
</tr>
<tr>
<td>R.femoralis L800</td>
<td>0 g/cm³</td>
</tr>
<tr>
<td>R.femoralis L830</td>
<td>0 g/cm³</td>
</tr>
<tr>
<td>R.iliac L860</td>
<td>0 g/cm³</td>
</tr>
<tr>
<td>L.femoralis L900</td>
<td>0 g/cm³</td>
</tr>
<tr>
<td>L.femoralis L930</td>
<td>0 g/cm³</td>
</tr>
<tr>
<td>L.iliac L960</td>
<td>0 g/cm³</td>
</tr>
<tr>
<td>VCI L970</td>
<td>0 g/cm³</td>
</tr>
<tr>
<td>VCI L980</td>
<td>0 g/cm³</td>
</tr>
<tr>
<td>VCI L990</td>
<td>0 g/cm³</td>
</tr>
<tr>
<td>VCI L1000</td>
<td>0.7822 g/cm³</td>
</tr>
</tbody>
</table>

**Other resistances:**

<table>
<thead>
<tr>
<th>Other resistance</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>R.jugular R001</td>
<td>11.172 mmHg s/mL</td>
</tr>
<tr>
<td>R.subclavian R002</td>
<td>8.531 mmHg s/mL</td>
</tr>
<tr>
<td>L.jugular R003</td>
<td>11.17 mmHg s/mL</td>
</tr>
<tr>
<td>L.subclavian R004</td>
<td>8.531 mmHg s/mL</td>
</tr>
<tr>
<td>R.jugular R011</td>
<td>11.1727 mmHg s/mL</td>
</tr>
<tr>
<td>R.subclavian R012</td>
<td>8.531 mmHg s/mL</td>
</tr>
<tr>
<td>L.jugular R013</td>
<td>11.1727 mmHg s/mL</td>
</tr>
<tr>
<td>L.subclavian R014</td>
<td>8.531 mmHg s/mL</td>
</tr>
</tbody>
</table>

**Compliances:**

<table>
<thead>
<tr>
<th>Compliance</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>R.jugular C001</td>
<td>5 cm³/mmHg</td>
</tr>
<tr>
<td>R.subclavian C002</td>
<td>10 cm³/mmHg</td>
</tr>
<tr>
<td>L.jugular C003</td>
<td>5 cm³/mmHg</td>
</tr>
<tr>
<td>L.subclavian C004</td>
<td>10 cm³/mmHg</td>
</tr>
<tr>
<td>R.jugular C011</td>
<td>0.8713 cm³/mmHg</td>
</tr>
<tr>
<td>R.subclavian C012</td>
<td>0.9971 cm³/mmHg</td>
</tr>
</tbody>
</table>

---

Appendices 27
<table>
<thead>
<tr>
<th>Artery</th>
<th>Code</th>
<th>Volume</th>
<th>Pressure</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>L.jugular</td>
<td>C013</td>
<td>0.8713 cm$^3$/mmHg</td>
<td>pulmonary Z352</td>
<td>0.04 mmHg s/mL</td>
</tr>
<tr>
<td>L.subclavian</td>
<td>C014</td>
<td>0.9971 cm$^3$/mmHg</td>
<td>aorta Z551</td>
<td>0.02 mmHg s/mL</td>
</tr>
<tr>
<td>R.innominate</td>
<td>C100</td>
<td>1.4255 cm$^3$/mmHg</td>
<td>aorta Z600</td>
<td>0.08 mmHg s/mL</td>
</tr>
<tr>
<td>L.innominate</td>
<td>C125</td>
<td>1.4255 cm$^3$/mmHg</td>
<td>Other:</td>
<td></td>
</tr>
<tr>
<td>VSC</td>
<td>C150</td>
<td>30 cm$^3$/mmHg</td>
<td>t_delay: 0.15 s</td>
<td></td>
</tr>
<tr>
<td>VCS</td>
<td>C200</td>
<td>30 cm$^3$/mmHg</td>
<td>a_RA: 0.005 mmHg/mL$^2$</td>
<td></td>
</tr>
<tr>
<td>RA</td>
<td>C300</td>
<td>1 cm$^3$/mmHg</td>
<td>b_RA: 5 mL</td>
<td></td>
</tr>
<tr>
<td>RV</td>
<td>C350</td>
<td>1 cm$^3$/mmHg</td>
<td>c_RA: 0.165 mmHg/ml</td>
<td></td>
</tr>
<tr>
<td>pulm.arteries</td>
<td>C400</td>
<td>7 cm$^3$/mmHg</td>
<td>d_RA: 0.2 mmHg</td>
<td></td>
</tr>
<tr>
<td>pulm.periphery</td>
<td>C430</td>
<td>30 cm$^3$/mmHg</td>
<td>a_RV: 0.0007 mmHg/ml$^2$</td>
<td></td>
</tr>
<tr>
<td>pulm.veins</td>
<td>C460</td>
<td>50 cm$^3$/mmHg</td>
<td>b_RV: 5 mL</td>
<td></td>
</tr>
<tr>
<td>LA</td>
<td>C500</td>
<td>1 cm$^3$/mmHg</td>
<td>c_RV: 1.4 mmHg/mL</td>
<td></td>
</tr>
<tr>
<td>LV</td>
<td>C550</td>
<td>1 cm$^3$/mmHg</td>
<td>d_RV: 1 mmHg</td>
<td></td>
</tr>
<tr>
<td>abdominal</td>
<td>C650</td>
<td>88.23529412 cm$^3$/mmHg</td>
<td>k_1RV: 0.001 s/mL</td>
<td></td>
</tr>
<tr>
<td>renal</td>
<td>C750</td>
<td>61.76470588 cm$^3$/mmHg</td>
<td>k_2RV: 0.000005 s/mL</td>
<td></td>
</tr>
<tr>
<td>R.femoralis</td>
<td>C800</td>
<td>10 cm$^3$/mmHg</td>
<td>a_LA: 0.005 mmHg/mL$^2$</td>
<td></td>
</tr>
<tr>
<td>R.femoralis</td>
<td>C830</td>
<td>10 cm$^3$/mmHg</td>
<td>b_LA: 5 mL</td>
<td></td>
</tr>
<tr>
<td>R.iliac</td>
<td>C860</td>
<td>10 cm$^3$/mmHg</td>
<td>c_LA: 0.55 mmHg/ml</td>
<td></td>
</tr>
<tr>
<td>L.femoralis</td>
<td>C900</td>
<td>10 cm$^3$/mmHg</td>
<td>d_LA: 1 mmHg</td>
<td></td>
</tr>
<tr>
<td>L.femoralis</td>
<td>C930</td>
<td>10 cm$^3$/mmHg</td>
<td>a_LV: 0.0007 mmHg/ml$^2$</td>
<td></td>
</tr>
<tr>
<td>L.iliac</td>
<td>C960</td>
<td>10 cm$^3$/mmHg</td>
<td>b_LV: 5 mL</td>
<td></td>
</tr>
<tr>
<td>VCI</td>
<td>C970</td>
<td>37.5 cm$^3$/mmHg</td>
<td>c_LV: 8 mmHg/mL</td>
<td></td>
</tr>
<tr>
<td>VCI</td>
<td>C980</td>
<td>37.5 cm$^3$/mmHg</td>
<td>d_LV: 1 mmHg</td>
<td></td>
</tr>
<tr>
<td>VCI</td>
<td>C990</td>
<td>37.5 cm$^3$/mmHg</td>
<td>k_1LV: 0.001 s/mL</td>
<td></td>
</tr>
<tr>
<td>VCI</td>
<td>C1000</td>
<td>37.5 cm$^3$/mmHg</td>
<td>k_2LV: 0.000005 s/mL</td>
<td></td>
</tr>
<tr>
<td>VCI</td>
<td>C1020</td>
<td>37.5 cm$^3$/mmHg</td>
<td>t_h: 1 s</td>
<td></td>
</tr>
<tr>
<td>VCI</td>
<td>C1050</td>
<td>37.5 cm$^3$/mmHg</td>
<td>t_inc: 0.001 s</td>
<td></td>
</tr>
<tr>
<td>VCI</td>
<td>C1070</td>
<td>37.5 cm$^3$/mmHg</td>
<td>p_int: 11 mmHg</td>
<td></td>
</tr>
</tbody>
</table>

**Systemic peripheral resistances:**

- R601: 2.574 mmHg s/mL
- R0001: 26.6-2.574 mmHg s/mL
- R0002: 17.2-2.574 mmHg s/mL
- R0003: 26.7-2.574 mmHg s/mL
- R0004: 15-2.574 mmHg s/mL
- R649: 4.29-2.574 mmHg s/mL
- R749: 5.46-2.574 mmHg s/mL
- R799: 12.2-2.574 mmHg s/mL
- R899: 12.2-2.574 mmHg s/mL
- R700: 18.3 mmHg s/mL

**Other:**

- RV_tau: 0.5*t_inc; s
- LV_tau: 0.5*t_inc; s
- t_pV: 0.22 s
- tau_clV: 3 - s
- tau_rLV: 0.17 s
- t_pA: 0.065 s
- alpha_A: 2.88
- tau_clLA: 0.06 s
- tau_rLA: 0.08 s
- tau_clRA: 0.06 s
- tau_rRA: 0.08 s
- tau_clRV: 0.15 s
- tau_rRV: 0.17 s

---

**Appendices 28**

**Questionnaires used in chapter 11**

**4.1 Overview of subjects addressed**

<table>
<thead>
<tr>
<th>General</th>
<th>Theory</th>
<th>Practical</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intermittent Abd. compression</td>
<td>Thoracic &amp; cardiac pump</td>
<td>Survival factors and ethics</td>
</tr>
<tr>
<td>Defibrillation</td>
<td>Diagnosis and ‘call for help’</td>
<td>Precordial thump</td>
</tr>
<tr>
<td>A-B-C &amp; C-A-B systems</td>
<td>Ventilation pressure</td>
<td>Trauma and CPR</td>
</tr>
<tr>
<td>Defibrillation</td>
<td>Airway management</td>
<td>Medication dosages</td>
</tr>
<tr>
<td></td>
<td>Diagnosis/recognition</td>
<td>Medication</td>
</tr>
<tr>
<td></td>
<td>Med. access routes</td>
<td>Intoxication’s and ACLS</td>
</tr>
<tr>
<td></td>
<td>Drug classes</td>
<td>Adenosine</td>
</tr>
<tr>
<td></td>
<td>Bretylium</td>
<td>Epinephrine</td>
</tr>
<tr>
<td></td>
<td>Lidocaine</td>
<td>Magnesium</td>
</tr>
<tr>
<td></td>
<td>Procainamide</td>
<td>Sodium Bicarbonate</td>
</tr>
</tbody>
</table>

**Key items in the MC questions are listed to give the scope. Categories are somewhat arbitrary. ACLS questions have not been listed separately. Topics were used based on “useful to know” and “knowledge should improve resuscitation” criteria as judged by authors and an outside adviser.**

**4.2 Demographic and subjective information Questionnaire**

**Instructions:** Check □ on right side of text as appropriate, Fill in ___ on _____, and place an “X” on scale from left (best) to right (10=worst) value in a fashion similar to a Visual analog scale (VAS).

<table>
<thead>
<tr>
<th>Part 1: Demographics</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Gender: Male □, Female □</td>
</tr>
<tr>
<td>2. Current year-in-training: Intern □, 1st year □, 2nd year □, 3rd year □, 4th year □, 5th year □</td>
</tr>
<tr>
<td>3. Experience since in-training in: Gasthuisberg Yes □, No □, Pellenberg Yes □, No □, St. Pieter Yes □, No □</td>
</tr>
<tr>
<td>4. The Intensive Care unit(s): Yes □, No □</td>
</tr>
<tr>
<td>5. The Emergency Dept.: Yes □, No □</td>
</tr>
<tr>
<td>6. Pre-hospital EMS: Yes □, No □</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Part 2:</th>
</tr>
</thead>
<tbody>
<tr>
<td>4. Did you receive BLS (Basic Life support) CPR instruction during Medical School?  Yes □, No □</td>
</tr>
<tr>
<td>5. If yes: how long ago was the last course: (fill in) ___ years ago.</td>
</tr>
<tr>
<td>6. How many hours did the course encompass: (fill in) ___ hours, or don’t remember □</td>
</tr>
<tr>
<td>7. When was the LAST BLS-CPR course you followed? In Medical School □, or (fill in) ___ years ago, or never followed a course □</td>
</tr>
<tr>
<td>8. Have you ever followed a CPR course organised for laypersons: Yes □, No □</td>
</tr>
<tr>
<td>9. Have you followed an ACLS (Advanced Cardiac Life Support) course? Yes □, No □</td>
</tr>
<tr>
<td>10. If yes: how long ago was the last one: 0 years □, 1 □, 2 □, 3 □, 4 □, 5 □, 6 years ago □</td>
</tr>
<tr>
<td>11. During the MOST recent CPR course you followed, which guidelines were used: Am. Heart Ass □, European Resuscitation Counc. □, unknown □</td>
</tr>
<tr>
<td>12. Do you know what the Utstein Methodology is: Yes □, No □</td>
</tr>
</tbody>
</table>
4 Questionnaires used in chapter 11

4.1 Overview of subjects addressed

<table>
<thead>
<tr>
<th>General</th>
<th>Theory</th>
<th>Practical</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Intermittent compression</td>
<td>Diagnosis and 'call for help'</td>
</tr>
<tr>
<td></td>
<td>Thoracic &amp; cardiac pump models</td>
<td>Precordial thump</td>
</tr>
<tr>
<td></td>
<td>Survival factors and ethics</td>
<td>A-B-C &amp; C-A-B systems</td>
</tr>
<tr>
<td></td>
<td>Defibrillation</td>
<td>Ventilation pressure</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Trauma and CPR</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Defibrillation</td>
</tr>
<tr>
<td>Pediatric</td>
<td>chain of events</td>
<td>Defibrillation</td>
</tr>
<tr>
<td></td>
<td>BLS and ACLS technique</td>
<td>Airway management</td>
</tr>
<tr>
<td></td>
<td>Rhythm recognition</td>
<td>Medication dosages</td>
</tr>
<tr>
<td>Medication</td>
<td>Med. access routes</td>
<td>Diagnosis/recognition</td>
</tr>
<tr>
<td></td>
<td>Intoxication's and ACLS</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Drug classes</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Key items in the MC questions are listed to give the scope. Categories are somewhat arbitrary. ACLS questions have not been listed separately. Topics were used based on "useful to know" and "knowledge should improve resuscitation" criteria as judged by authors and an outside adviser.

4.2 Demographic and subjective information Questionnaire

Instructions: Check ☐ on right side of text as appropriate. Fill in ______, and place an "X" on scale from left (best) to right (10=worst) value in a fashion similar to a Visual analog scale (VAS).

Part 1: Demographics
1. Gender: Male ☐, Female ☐.
3. Experience since in-training in:
   ♦ The Op. theater(s): Gashuisbergen Yes ☐ No ☐, Pellenberg Yes ☐ No ☐, St. Pieter Yes ☐ No ☐.
   ♦ The Intensive Care unit(s): Yes ☐ No ☐.
   ♦ The Emergency Dept.: Yes ☐ No ☐.
   ♦ Pre-hospital EMS: Yes ☐ No ☐.

Part 2:
4. Did you receive BLS (Basic Life support) CPR instruction during Medical School? Yes ☐, No ☐.
   ♦ If yes: how long ago was the last course: (fill in) ______ years ago.
   ♦ How many hours did the course encompass: (fill in) ______ hours, or don't remember ☐.
5. When was the LAST BLS-CPR course you followed?
   ♦ In Medical School ☐, or (fill in) ______ years ago, or never followed a course ☐.
   ♦ Have you ever followed a CPR course organised for laypersons: Yes ☐, No ☐.
6. Have you followed an ACLS (Advanced Cardiac Life Support) course? Yes ☐, No ☐.
   ♦ If yes: how long ago was the last one: 0 years ☐, 1-2 years ☐, 3-4 years ☐, 5-6 years ☐, 6 years ago.
7. During the MOST recent CPR course you followed, which guidelines were used:
   ♦ Am. Heart Ass ☐, European Resuscitation Counc. ☐, unknown ☐.
8. Do you know what the Ulstein Methodology is: Yes ☐, No ☐.

Appendices 29
9. Do you instruct in CPR course(s): Yes-once ☐, Yes-regularly ☐, No ☐.
   ♦ If yes: how often (fill in) ______ per year
   ♦ If yes: for which organisations: (fill in)________________________, ________________________.

Part 3:
10. Have you, as part of your experience in anesthesiology, during the day, been involved in resuscitation incidents (defined as unintentional in/external cardiac massage, etc.): Yes ☐, No ☐.
11. Have you, as part of your experience in anesthesiology, during on-call periods, been involved in resuscitation incidents (defined as unintentional in/external cardiac massage, etc.): Yes ☐, No ☐.
   ♦ If yes: how often on average (fill in) ______ per month or ______ per year.
12. Have you, as part of your experience in anesthesiology, been involved in pre-hospital resuscitation incidents at any time: Yes ☐, No ☐.
   ♦ If yes: how often on average (fill in) ______ per month or ______ per year.

Part 4:
13. How well informed do you consider yourself in regard to (recent) developments in CPR research:
   ♦ (place tick on VAS) very up-to-date __________________________, not up-to-date at all.
14. How well informed do you consider yourself in regard to (recent) developments in CPR practice:
   ♦ (place tick on VAS) very up-to-date __________________________, not up-to-date at all.
15. How would you evaluate your BLS-CPR skills:
    ♦ (place tick on VAS) outstanding __________________________, very poor.
16. How would you evaluate your ACLS-CPR skills:
    ♦ (place tick on VAS) outstanding __________________________, very poor.
17. How would you evaluate your overall CPR related skills, knowledge and efficacy:
    ♦ (Place tick on VAS) outstanding __________________________, very poor.

4.3 Knowledge Questionnaire (translated from Dutch and validated).
   Layout compressed for efficiency.

Knowledge Inventory (Part 1)
1. Which of the following is incorrect, with regard to a CPR intervention in the pre-hospital situation?
   (a) If two rescuers are involved, one should examine the patient then "call for help" while the other starts CPR.
   (b) If one rescuer is available, he should resuscitate during one minute, then "call for help" after which he should continue CPR.
   (c) After the age of 12, children are treated as adults as far as CPR guidelines are concerned.
   (d) If a rescuer thinks that a corpus alienum is blocking the airway, airway maneuvers (i.e., Heimlich) should be performed before a "call for help".

2. Recent studies have shown that the use of "intermittent abdominal compressions" gives a consistent and significant improvement in survival when performed in the pre-hospital setting.
   (a) This is correct.
   (b) This is correct; however, a guarantied free airway (i.e., endotracheal intubation) must be established before this technique is used.
   (c) This is correct but only in conjunction with a two-rescuer technique.
   (d) This is incorrect. Evidence is currently equivocal.

3. The two most accepted (patho)physiological explanations for the generation of cardiac output during external cardiac massage have been shown to be the "thoracic pump" and "cardiac pump" models.
   (a) The "thoracic pump" model seems to be most relevant as a mechanism for flow in (very) high-frequency external cardiac massage rates. The (cardiac pump) model seems to be relevant only in the low-frequency external cardiac massage rates.
   (b) The "active compression-decompression" technique does not support either of these theories.
   (c) Both the "thoracic pump" and the "cardiac pump" models are applicable when performing external cardiac massage as described by current guidelines.
   (d) All the above (a,b,c) are incorrect.
4. The "generally accepted" definitive survival percentage following an acute myocardial infarction with circulatory collapse and CPR is:
   (a) Circa 15%.
   (b) Circa 45%.
   (c) Circa 20%.
   (d) Less than 8%.

5. Which of the listed items is most likely the most important factor with regard to change of survival after an acute circulatory collapse?
   (a) Performance of BLS-CPR sometime after the collapse.
   (b) The length of time between collapse and return of unassisted circulation.
   (c) The length of time between collapse and defibrillation.
   (d) The underlying reason for the circulatory collapse.

6. Cardiac massage, when performed well, gives …
   (a) 25-35% of cardiac output.
   (b) Relatively high diastolic pressures.
   (c) 40-55% of normal cardiac output.
   (d) Normal mean arterial pressures.

7. The "A-B-C" methodology for rapid evaluation of the patient has a European variation which is known as the "C-A-B-" methodology. The (patho)physiological basis for this methodology is …
   (a) The conclusion that respiratory insufficiency is the reason for most circulatory collapses.
   (b) That C-A-B is more efficient since most acute circulatory collapses are primarily pump-related (i.e., rhythm), and adequate \( P_{O_2} \) may be presupposed.
   (c) That during the first minutes of external cardiac massage sufficient ventilation is a by-product of the cardiac massage.
   (d) The return to use of the "precordial thump".

8. Which of the points listed below is not correct?
   (a) In order to minimize gastric insufflation, mouth-to-mouth ventilation should be performed slowly (1.5-2.0 sec).
   (b) The use of the "Sellick maneuver" during BLS is not a useful technique and may not be expected to protect against aspiration.
   (c) Mouth-to-nose artificial ventilation is a better technique to use with regard to maintenance of low insufflation pressures.
   (d) A good measure for ventilation volume is a visual check for expansion of the thorax during the insufflation.

9. What are the best positions and techniques to perform artificial (BLS) ventilation when confronted with a patient who may have a (posttraumatic) cervical vertebral column injury?
   (a) Avoid changing the patient's position at all costs until ACLS support is available and cervical stabilisation has been performed.
   (b) Use the "stable side" position (conform Rautek or NATO) and perform artificial ventilation with the patient in this position.
   (c) Move the patient to a (sufficiently) neutral position, and perform artificial ventilation using the "head tilt - chin lift" maneuver.
   (d) Move the patient to a (sufficiently) neutral position, and perform artificial ventilation using the "jaw thrust" maneuver.

10. Lidocaine is indicated for immediate use (primary medication) in pulseless VT or in VF. This is:
    (a) Correct. The dosage is 1.0 - 1.5 mg/kg IV push.
    (b) Correct, and it should be used immediately following the first defibrillation.
    (c) Incorrect as the use of lidocaine has a negative influence on the initial success of defibrillation.
    (d) Incorrect as the ratio therapeutic/toxic levels during circulatory collapse is too small, and the chance of concurrent renal insufficiency is too great.

11. Epinephrine has a central role in the pharmacological intervention in circulatory collapse and CPR. This is:
    (a) Correct. The dosage is increments of 1mg (10ml of a 1:10,000 solution) IV push.
12. Electric DC-defibrillation in apparent asystole (using the "quick look" or later lead II) is:
   (a) Incorrect. A needless and damaging act as the myocardium is depolarised already.
   (b) Incorrect. It has a negative influence on prognosis due to time lost for cardiac massage.
   (c) Correct, as it is seen on most popular medical soaps.
   (d) Correct, or at least an option, since an unrecognised but potentially treatable microvolt VF may otherwise remain untreated.

13. The currently accepted treatment for VF is electric DC-defibrillation with:
   (a) Immediate maximum energy (i.e., 360 J).
   (b) First 200 J, then 150 J due to decrease in impedance.
   (c) First 200 J, then 200 J and after that maximal energy (i.e., 360 J).
   (d) First 100 J, the increase in increments of 50 J with each defibrillation.

14. Of the factors listed below, the one with the most influence on the success of a defibrillation is:
   (a) The amperage of current passing through the heart (amp).
   (b) The total energy applied (joule).
   (c) The voltage applied (volt).
   (d) The thoracic impedance (ohm).

15. As soon as BLS-CPR has been started the survival percentage will remain the same.
   (a) This is correct, and the major motivation for BLS are layperson CPR courses.
   (b) This is correct, but the total BLS time is limited to circa 30 minutes, after which the prognosis decreases in an exponential curve.
   (c) This is incorrect as it is incomplete and does not consider one- versus two-rescuer CPR.
   (d) This is incorrect, the survival chance decreases with time in an approximately linear fashion.

16. When considering an "optimal" protocol for starting a resuscitation, this should be:
   (a) External cardiac massage/artificial ventilation; intubation; defibrillation; IV-access.
   (b) Intubation; defibrillation; external cardiac massage/artificial ventilation; IV-access.
   (c) Defibrillation; external cardiac massage/artificial ventilation; intubation; IV-access.
   (d) There is (patho) physiological basis for preference. Should be evaluated by case.

Knowledge Inventory (Part 2)

17. The "precordial thump" …
   (a) Is an unproven and potentially detrimental technique that is no longer advocated.
   (b) Should be advocated as an ACLS-oriented technique to be used in "witnessed arrest" situations (i.e., a form of rapid defibrillation).
   (c) Should be used at the beginning of every cycle of external cardiac massage following a "witnessed arrest".
   (d) Should only be used for cardioversion of VT (with or without output).

18. During a cardiac arrest, medication can be administered:
   (a) IV, and if a peripheral venous access has been used, this should be followed by a 10ml flush.
   (b) Endotracheal (depending on medication to be used), and in a two- or three-fold dosage regime.
   (c) Intra-osseus
   (d) All of the above options may be useful.

19. Which location is advocated as most suitable for quick venous access?
   (a) Peripheral on an extremity (i.e., Cephalic V.)
   (b) Peripheral but central (Ext. Jugular V.)
   (c) Central (Subclavian V.)
   (d) Central (Femoral V.)

20. If VF persists after defibrillation, epinephrine and lidocaine, the next preferred medication is …
   (a) Procainamide hydrochloride, in a dosage of 20mg/min to max of 17mg/kg.
   (b) Bretylium tosylate, in a dosage of 5mg/kg IV push followed by electric DC-defibrillation.
17. Knowledge Inventory (Part 2)

20. If VF persists after defibrillation, epinephrine and lidocaine, the next preferred medication is ...

Which location is advocated as most suitable for quick venous access?

When considering an "optimal" protocol for starting a resuscitation, this should be:

Of the factors listed below, the one with the most influence on the success of a defibrillation is:

The currently accepted treatment for VF is electric DC-defibrillation with:

As soon as BLS-CPR has been started the survival percentage will remain the same.

Should only be used for cardioversion of VT (with or without output).

Central (Femoral V.)

Peripheral but central (Ext. Jugular V.)

All of the above options may be useful.

Intra-osseus

IV, and if a peripheral venous access has been used, this should be followed by a 10ml flush.

Is an unproven and potentially detrimental technique that is no longer advocated.

There is (patho) physiological basis for preference. Should be evaluated by case.

Defibrillation; external cardiac massage/artificial ventilation; intubation; IV-access.

This is incorrect, the survival chance decreases with time in an approximately linear fashion.

This is correct, but the total BLS time is limited to circa 30 minutes, after which the prognosis decreases in an exponential curve.

This is correct, and the major motivation for BLS are layperson CPR courses.

The voltage applied (volt).

The total energy applied (joule).

The amperage of current passing through the heart (amp).

First 200 J, then 200 J and after that maximal energy (i.e., 360 J).

First 200 J, then 150 J due to decrease in impedance.

Correct, but only in cases of asystole.

Incorrect. It has a negative influence on prognosis due to time lost for cardiac massage.

Both a and b are correct.

Correct, as it is seen on most popular medical soaps.

Incorrect. It has a negative influence on prognosis due to time lost for cardiac massage.

Both a and b are correct.

Epinephrine in babys and children …

Which of the listed options is ...

Abdominal compressions can be performed in patient without regard for their position (standing, sitting, or lying down).

The Heimlich maneuver is indicated in all age groups.

Minimal intervention(s) including defibrillation until the patient's core temperature is at least 30o C.

A 5:1 compression-ventilation cycle is used in both 1 and 2 rescuers CPR.

Bretylium tosylate, in dosage of 5mg/kg IV push.

Magnesium Sulfate, in loading dosage of 1 to 2g, as IV-infusion over 5-60 minutes.

Procainamide, in dosage of 10mg/kg.

Adenosine, in dosage of 6mg IV push.

Both a and b are correct.

Should be given in dosages similar to those of adults.

Should be given in dosages of 0.01mg/kg for IV and 0.1mg/kg for endotracheal use.

21. During CPR sodium bicarbonate may be given via the endotracheal tube (i.e., no IV-access).

(a) Correct, however only in small volume, using 8.4% or better concentrations.

(b) Correct, but only when an isotonic solution is used (i.e., 1/6 M).

(c) Incorrect, since sodium bicarbonate activates epinephrine within the alveoli.

(d) Incorrect, since sodium bicarbonate is not lipid soluble.

22. For “Torsade de Pointes” the first choice medication is:

(a) Lidocaine, in dosage 0.5mg/kg IV push.

(b) Procainamide, in dosage of 10mg/kg.

(c) Magnesium Sulfate, in loading dosage of 1 to 2g, as IV-infusion over 5-60 minutes.

(d) Bretylium tosylate, in dosage of 5mg/kg IV push.

23. If there is no indication of a high serum potassium level, of hypocalcemia, or an intoxication with Ca++ entry blockers, the use of Ca++ during treatment of acute circulatory collapse is …

(a) A Class I action (always acceptable, useful and proven effective).

(b) A Class IIa action (acceptable, evidence shows tendency for benefit and effectivity).

(c) A Class IIb action (acceptable, no evidence for effectivity, may be useful, most likely not damaging).

(d) A Class III action (use of this medication in this situation is incorrect and could be detrimental).

24. If considering ending an ACLS resuscitation attempt, in which no recuperation has been seen, after maximal therapy, the attempt should be continued if:

(a) The patient is hypothermic.

(b) There may be an intoxication with tricyclic antidepressants.

(c) The patient is hyperthermic.

(d) Both a and b are correct.

25. The pre-hospital treatment of severe hypothermia entails …

(a) Rapid intubation and (IV-)pharmacological support.

(b) Rapid, high energy, defibrillation.

(c) Minimal intervention(s) including defibrillation until the patient's core temperature is at least 30° C.

(d) Both a and b are correct.

26. One rescuer (BLS) CPR for a child (< 1year old) is performed by starting with two ventilations followed by a compression-ventilation cycle of 5:1, at a frequency of 100 compressions per minute and a depth of approximately 0.5 to 1 cm depth.

(a) This is correct. Ventilation volume should be visually controlled by observing thoracic rise.

(b) The compression frequency should be 80 compressions per minute.

(c) The compression depth should be 1.5 to 2.5 cm.

(d) The compression cycle should be 15:2.

27. Which of the listed options is incorrect when treating an airway obstruction?

(a) The Heimlich maneuver is indicated in all age groups.

(b) Abdominal compressions can be performed in patient without regard for their position (standing, sitting, or lying down).

(c) The so called "blind finger sweep" designed to find and remove foreign matter from the mouth after the Heimlich maneuver is no longer a recommended technique.

(d) If, following five abdominal compressions, artificial ventilation is not possible, the five compressions should be repeated immediately.

28. Which of the statements is incorrect, with regard to CPR in children 1-8 years old?

(a) A 5:1 compression-ventilation cycle is used in both 1 and 2 rescuers CPR.

(b) The patency of the circulation is monitored preferentially using the carotid artery.

(c) The compression frequency is 100 per minute, in order to achieve an effective rate of 80.

(d) The sternum should be compressed 1.25 to 2.5 cm.

29. Epinephrine in babys and children …

(a) Should be given in dosages similar to those of adults.

(b) Should be given in dosages of 0.01mg/kg for IV and 0.1mg/kg for endotracheal use.
(c) Should preferentially be given via the endotracheal tube.
(d) Should not be used (Class III action).

30. Arrhythmias seen most often in combination with pediatric circulatory collapse are …
   (a) Tachyarrhythmias, leading to collapse.
   (b) Bradyarrhythmias, leading to collapse.
   (c) Immediate ventricular fibrillation.
   (d) Immediate asystole.

31. Medication used to compensate acidosis during CPR, (i.e., sodium bicarbonate) …
   (a) Has limited use in children since most circulatory collapses have a respiratory origin.
   (b) May be used if the resuscitation progresses longer than 10 minutes, in the patient has been intubated and can
      be adequately ventilated, in a dosage of 1mEq/kg.
   (c) Is a Class II B action.
   (d) All of the items listed above are correct.

32. Bradycardia in a child younger than 5 years old, is defined as:
   (a) Frequency < 100 beats per minute.
   (b) Frequency < 80 beats per minute.
   (c) Frequency < 60 beats per minute.
   (d) Frequency < 50 beats per minute.

33. Defibrillation for a child …
   (a) Should be performed using adult paddles if the weight is > 25kg.
   (b) Is performed using 2J/kg (first defibrillation) and thereafter with 4J/kg.
   (c) Should be done transthoracic by placing the paddles in the axillair line level with the heart.
   (d) Should always be done in synchronised mode.

34. In babies with a supraventricular tachycardia (SVT) …
   (a) Synchronised defibrillation should be avoided.
   (b) Adenosine IV is not recommended.
   (c) Verapamil IV should be avoided.
   (d) The use of antiarrhythmic medication via a central venous catheter should be avoided.

35. Synchronous defibrillation is indicated in children with …
   (a)VF.
   (b) SVT in the presence of hypotension.
   (c) Pulseless VT.
   (d) Asystole.

Answers (see also JAMA 1992; 268(16): 2171 - 2298):

<p>| | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>b</td>
<td>a</td>
<td>a</td>
<td>c</td>
<td>d</td>
</tr>
<tr>
<td>2</td>
<td>d</td>
<td>a</td>
<td>c</td>
<td>a</td>
<td>c</td>
</tr>
<tr>
<td>3</td>
<td>c</td>
<td>b</td>
<td>a</td>
<td>d</td>
<td>b</td>
</tr>
<tr>
<td>4</td>
<td>a</td>
<td>d</td>
<td>a</td>
<td>d</td>
<td>a</td>
</tr>
<tr>
<td>5</td>
<td>c</td>
<td>c</td>
<td>d</td>
<td>b</td>
<td>d</td>
</tr>
</tbody>
</table>

Appendices
5 Questionnaires used in Chapter 12

5.1 The pre-assessment questionnaire.

<table>
<thead>
<tr>
<th>Question</th>
<th>VAS (place an “X” at your choice on the scale)</th>
</tr>
</thead>
<tbody>
<tr>
<td>With regard to your knowledge: How well do you feel that you were</td>
<td>0 .................................................. 10</td>
</tr>
<tr>
<td>equipped to perform BLS-CPR at the end of your last training course?</td>
<td>(not at all) (perfectly)</td>
</tr>
<tr>
<td>With regard to your practical skills: How well do you feel that you were</td>
<td>0 .................................................. 10</td>
</tr>
<tr>
<td>equipped to perform BLS-CPR at the end of your last training course?</td>
<td>(not at all) (perfectly)</td>
</tr>
<tr>
<td>With regard to your knowledge: How well do you feel that you are</td>
<td>0 .................................................. 10</td>
</tr>
<tr>
<td>equipped to perform BLS-CPR this minute?</td>
<td>(not at all) (perfectly)</td>
</tr>
<tr>
<td>With regard to your practical skills: How well do you feel that you were</td>
<td>0 .................................................. 10</td>
</tr>
<tr>
<td>equipped to perform BLS-CPR this minute?</td>
<td>(not at all) (perfectly)</td>
</tr>
</tbody>
</table>

This was filled in by the candidates before they had been briefed on the explicit purpose of the assessment. The visual analogue scale (VAS) was measured to be exactly 10 cm. The candidates placed an ‘x’ at a position of their self-evaluation. Time to last course was validated using the hospital’s training database. BLS-CPR = basic life support cardiopulmonary resuscitation.

5.2 The post assessment questionnaire.

<table>
<thead>
<tr>
<th>Question</th>
<th>VAS (place an “X” on the 10 cm scale)</th>
</tr>
</thead>
<tbody>
<tr>
<td>How well do you feel that you just did with regard to compression depth?</td>
<td>0 .......................................... 10</td>
</tr>
<tr>
<td>(very poorly) (perfectly)</td>
<td></td>
</tr>
<tr>
<td>Do you feel your compressions were generally too deep or too shallow?</td>
<td>0 .......................................... 10</td>
</tr>
<tr>
<td>(too deep) (too shallow)</td>
<td></td>
</tr>
<tr>
<td>What percentage (%) of compressions do you think were in the correct range?</td>
<td>0 ...................................... 100</td>
</tr>
<tr>
<td>(none) (all)</td>
<td></td>
</tr>
<tr>
<td>How tiring did you find performing the chest compressions to be?</td>
<td>0 .......................................... 10</td>
</tr>
<tr>
<td>(extremely) (not at all)</td>
<td></td>
</tr>
<tr>
<td>How well do you feel that you just did with regard to compression frequency?</td>
<td>0 ..................................... 10</td>
</tr>
<tr>
<td>(very poorly) (perfectly)</td>
<td></td>
</tr>
<tr>
<td>What is the correct compression frequency, according to the guidelines?</td>
<td>........................ compression min⁻¹ (adults)</td>
</tr>
<tr>
<td>What is the correct compression depth</td>
<td>...... to ...... cm deep (adults)</td>
</tr>
</tbody>
</table>
This was filled in immediately after completion of the practical session. No feedback was given, or opportunity offered to see the written print-outs.

5.3 The effects of combining springs with different properties as applies to CPR (appendix to the manuscript).

During CPR courses caregivers are taught to compress the chest and to strive for 4 to 5 cm compression depth. Little is said about the role of force needed for this. Applying sufficient force to compress the chest deep enough can be a challenge in and of itself.

The CPR-Ezy technology suggests that a force of 50-54kg on the CPR-Ezy may be adequate for compressions in an adult. Most adults can produce such a force for limited periods of time and we suggest that this feedback may help in controlling and maintaining this effort. But does the use of the feedback system also influence the work the caregiver must produce?

This appendix approaches that question from a simplified, mathematical, point of view.

In Figure App-5-3a, an object such as a manikin or the chest wall of a human being, represented as a weak spring is compressed while lying on a firm surface. The stiffness of the chest wall, $d$, and the spring compression $x_1$ are related to the loading force $F_1$ via the equation

\[ F_1 = dx_1 \]  
\[ \text{(app 5-eq. 1)} \]

In Figure App-5-3b a spring-loaded device (e.g. the CPR-Ezy) is put on a firm surface. The stiffness of the spring $D$ and the compression of the spring $x_2$ are related to a loading force $F_2$ via the equation

\[ F_2 = Dx_2 \]  
\[ \text{(app 5-eq. 2)} \]

If the above two springs are placed one on top of the other (Figure App-5b-c), i.e. in series, such as the CPR-Ezy on the sternum of the chest, with the patient lying on a firm surface, continuity of force requires that

\[ F_1 = F_2 \]  
\[ \text{(app 5-eq. 3)} \]

Hence

\[ x_1 = \left(\frac{d}{D}\right) x_2 \]  
\[ \text{(app 5-eq. 4)} \]

The work done on the spring in the first example (Figure App-5-3a) equals

\[ W_1 = \frac{Fx_1}{2} \]  
\[ \text{(app 5-eq. 5)} \]

The work done on the combined springs (Figure App-5-3c) is equal to

\[ W_2 = \frac{Fx_2}{2} + \frac{Fx_1}{2} = \left(\frac{F}{2}\right) (x_1 + x_2) = \left(\frac{F}{2}\right) \left(1 + \left(\frac{d}{D}\right)\right) \]  
\[ \text{(app 5-eq. 6)} \]

Hence, for the same compression of the lighter spring (i.e. the patient or the manikin, $x_1$) the ratio of the amounts of work equals

\[ \frac{W_2}{W_1} = \left(1 + \left(\frac{d}{D}\right)\right) \]  
\[ \text{(app 5-eq. 7)} \]

For the particular combination of the CPR-Ezy with our manikin, measurements show that $d/D = 1.07$. Using eq. (1) compressing the combination of springs will require close to twice as much work as compressing the CPR-Ezy or, in this example the manikin, alone. The results of the study demonstrate that this should not be a clinical concern.

---

5.3 The effects of combining springs with different properties as applies to CPR (appendix to the manuscript).

During CPR courses caregivers are taught to compress the chest and to strive for 4 to 5 cm compression depth. Little is said about the role of force needed for this. Applying sufficient force to compress the chest deep enough can be a challenge in and of itself.

The CPREzy technology suggests that a force of 50-54kg on the CPREzy may be adequate for compressions in an adult. Most adults can produce such a force for limited periods of time and we suggest that this feedback may help in controlling and maintaining this effort. But does the use of the feedback system also influence the work the caregiver must produce?

This appendix approaches that question from a simplified, mathematical, point of view.

In Figure App-5-3a, an object such as a manikin or the chest wall of a human being, represented as a weak spring is compressed while lying on a firm surface.* The stiffness of the chest wall, $d$, and the spring compression $x_1$ are related to the loading force $F_1$ via the equation

$$F_1 = dx_1 \quad \text{(app 5-eq. 1)}$$

In Figure App-5-3b a spring-loaded device (e.g. the CPREzy) is put on a firm surface. The stiffness of the spring $D$ and the compression of the spring $x_2$ are related to a loading force $F_2$ via the equation

$$F_2 = Dx_2 \quad \text{(app 5-eq. 2)}$$

If the above two springs are placed one on top of the other (Figure App-5-3c), i.e. in series, as the CPREzy on the sternum of the chest, with the patient lying on a firm surface, continuity of force requires

$$F_1 = F_2 \quad \text{(app 5-eq. 3)}$$

Hence

$$x_1 = \frac{d}{D} x_2 \quad \text{(app 5-eq. 4)}$$

The work done on the spring in the first example (Figure App-5-3a) equals

$$W_1 = \frac{Fx_1}{2} \quad \text{(app 5-eq. 5)}$$

The work done on the combined springs (Figure App-5-3c) is equal to

$$W_2 = \frac{Fx_2}{2} + \frac{Fx_1}{2} = \frac{F}{2}(x_1 + x_2) = \frac{Fx_1}{2}(1 + \frac{d}{D}) \quad \text{(app 5-eq. 6)}$$

Hence, for the same compression of the lighter spring (i.e. the patient or the manikin, $x_1$) the ratio of the amounts of work equals

$$\frac{W_2}{W_1} = 1 + \frac{d}{D} \quad \text{(app 5-eq. 7)}$$

For the particular combination of the CPREzy with our manikin, measurements show that $d/D = 1.07$. Using eq. (1) compressing the combination of springs will require close to twice as much work as compressing the CPREzy or, in this example the manikin, alone. The results of the study demonstrate that this should not be a clinical concern.


Conclusions:

In these models, force indicators are not significantly disturbed by multiple layers, when their masses can be ignored. However, levels of compression may be entirely different, depending on the stiffnesses of the springs. The physical work performed by the caregiver also depends on spring stiffnesses and is increased in our example.

Figure App-5-3a: See text Appendix 5.3 for explanation of the symbols. Schematic of a weak spring (e.g. the human chest) on a firm surface.

Figure App-5-3b: See text Appendix 5.3 for explanation of the symbols. Schematic of a stiff spring on a firm surface.

Figure App-5-3c: See text Appendix 5.3 for an explanation of symbols. Schematic of the CPREzy on the chest of a manikin lying on a firm surface demonstrating the accumulation of work.
6 Appendix VI to chapter 14 Modelling the characteristics for any local situation.

When taught chest compressions, candidates aim for a 40-50 mm downward movement of the sternum. This is valid if the chest compressions are performed on a firm surface, as is generally the case in out-of-hospital arrests. In most courses students will be instructed to move the patient to the floor if he/she is on a compressible surface (e.g., a bed at home).

Relevance of non-firm surfaces, such as mattresses, emergency room stretchers and other structures in common use can be analyzed so that appropriate decisions can be made as to the need for a backboard or other corrective measures. The importance of avoiding shallow compressions has been repeatedly and sufficiently demonstrated.

System:
The components in an idealized system involved in chest compressions on a hospital bed are shown in Figure A1. In order to compute the effects of each of these components, five distances as well as two β’s (viscous properties of the chest and the mattress) must be determined.

When the mattress compression is large, the idealized system needs further elaboration: the first order differential equations convert to second order differential equations and the effective mass of the patient and of the weighted backboard (see methods section) must be included. Time constants also enter the equations, as demonstrated in Figure 6.1. The coefficients $D_4$ and $β_2$ may become time-variant for the ‘slow’ foam mattress (B in our experiments).

If, however, the mattress compression is sufficiently small, the system can be simplified to that supplied in figure A2, providing that (a) the bed frame is accepted to be rigid, b) the backboard is accepted to be far more rigid ($> 100x$) than the mattress and operates only to increase the effective surface area on the mattress and c) any feedback device is non-compressible. The simplified model consists of two components.

The indented area of the mattress is defined by the body size of the patient. However, in foam, as well as other “pressure-distributing” mattresses, the properties of the mattress negate the need to incorporate the complete mass or surface area. For chest compressions, the area of interest is denoted by the trunk, denoted $A_T$. The backboard enlarges this area to $A_B$. The ratio between the two is $a = A_B/A_T$, with $a > 1$ if a backboard is present and $a = 1$ if it is absent.

In the simplified model (Figure A2):  
\[ F_2 = D_2 (y_1 - y_2) + β_1 \frac{d}{dt} (y_1 - y_2) \]  
(app 6-eq. 1)

And the description of the rest of the system to:  
\[ F_2 = D_4 (y_2 - y_3) + β_2 \frac{d}{dt} (y_2 - y_3) \]  
(app 6-eq. 2)

Note that $β$ is zero for a non viscoelastic, or purely elastic, mattress such as a pure foam mattress might be. In addition, the two equations (appendices 1 and -2) should be solved for the variables $(y_1 - y_2)$ and $(y_2 - y_3)$ instead of the individual y’s which are not relevant here.

Example:

For non mathematical testing in the local situation, a practical, bedside, approach is:
- Mark the side of a manikin by placing a ruler against the side of the chest wall, such that it can be read easily.
- Place the manikin on the local bed/mattress combination.
- Place a bedside table with a over-the-bed surface just above the mattress and against the side of the manikin. Make sure that the ruler can be read.
- Perform chest compressions from the other side of the manikin, allowing a co-worker to read the over-the-bed surface in relation to the ruler.
- Using the manikin SkillReporter perform chest compressions at different depths and record the different effects on the total hand travel. This is the sum of the sternum-to-spine and the compression depth read off the ruler.
Appendices 14: Modelling the characteristics for any local situation.

When taught chest compressions, candidates aim for a 40-50 mm downward movement of the sternum. This is valid if the chest compressions are performed on a firm surface, as is generally the case in out-of-hospital arrests. In most courses students will be instructed to move the patient to the floor if he/she is on a compressible surface (e.g., a bed at home).

Relevance of non-firm surfaces, such as mattresses, emergency room stretchers and other structures in common use can be analyzed so that appropriate decisions can be made as to the need for a backboard or other corrective measures. The importance of avoiding shallow compressions has been repeatedly and sufficiently demonstrated.

System:
The components in an idealized system involved in chest compressions on a hospital bed are shown in Figure A1. In order to compute the effects of each of these components, five distances as well as two \( \xi \)'s (viscous properties of the chest and the mattress) must be determined.

When the mattress compression is large, the idealized system needs further elaboration: the first order differential equations convert to second order differential equations and the effective mass of the patient and of the weighted backboard (see methods section) must be included. Time constants also enter the equations, as demonstrated in Figure 6.1. The coefficients \( D_4 \) and \( \beta_2 \) may become time-variant for the 'slow' foam mattress (B in our experiments).

If, however, the mattress compression is sufficiently small, the system can be simplified to that supplied in figure A2, providing that (a) the bed frame is accepted to be rigid, b) the backboard is accepted to be far more rigid (\( \geq 100x \)) than the mattress and operates only to increase the effective surface area on the mattress and c) any feedback device is non-compressible. The simplified model consists of two components.

The indented area of the mattress is defined by the body size of the patient. However, in foam, as well as other "pressure-distributing" mattresses, the properties of the mattress negate the need to incorporate the complete mass or surface area. For chest compressions, the area of interest is denoted by the trunk, denoted \( AT \). The backboard enlarges this area to \( A_B \). The ratio between the two is \( a = \frac{A_B}{AT} \), with \( a > 1 \) if a backboard is present and \( a = 1 \) if it is absent.

In the simplified model (Figure A2):

\[
F_2 = D_2 \left( y_1 - y_2 \right) + \xi_1 \frac{d}{dt} \left( y_1 - y_2 \right)
\]

And the description of the rest of the system to:

\[
F_2 = D_4 \left( y_2 - y_3 \right) + \xi_2 \frac{d}{dt} \left( y_2 - y_3 \right)
\]

Note that \( \xi \) is zero for a non viscoelastic, or purely elastic, mattress such as a pure foam mattress might be. In addition, the two equations (appendices 1 and -2) should be solved for the variables \( y_1 - y_2 \) and \( y_2 - y_3 \) instead of the individual \( y \)’s which are not relevant here.

Example:
For non mathematical testing in the local situation, a practical, bedside, approach is:

- Mark the side of a manikin by placing a ruler against the side of the chest wall, such that it can be read easily.
- Place the manikin on the local bed/mattress combination.
- Place a bedside table with a over-the-bed surface just above the mattress and against the side of the manikin. Make sure that the ruler can be read.
- Perform chest compressions from the other side of the manikin, allowing a co-worker to read the over-the-bed surface in relation to the ruler.
- Using the manikin SkillReporter perform chest compressions at different depths and record the different effects on the total hand travel. This is the sum of the sternum-to-spine and the compression depth read off the ruler.

Note that if specifications are available, a mattress with a stiffness of 750 N/cm or more should be suitable for adequate chest compressions with a backboard, and offer limited (< 10 mm) spine-to-bed impression.

6.1 Captions for the figures.

Caption Figure Appendix VI-1: General model for components for in-hospital resuscitation. Representation of the idealized system: \( F_1(t) \) is the force being applied with \( x \) representing the displacement of individual components and \( D \) the stiffness of that component. \( \beta \) represents the viscous characteristics of the chest and of the mattress.

Figure Appendix 6-1

| \( F_1 \) (t) | \( x_1 \) feedback device | \( D_1 \) | \( F_1 = D_1 \left( x_1 - x_2 \right) \) |
| \( x_2 \) chest (sternum-to-spine) | \( D_2 + \beta_1 \) | \( F_1 = D_2 \left( x_2 - x_3 \right) + \beta_1 \frac{d}{dt} \left( x_3 - x_2 \right) \) |
| \( x_3 \) backboard | \( D_3 + \beta_2 \) | \( F_1 = D_3 \left( x_3 - x_4 \right) \) |
| \( x_4 \) mattress | \( D_4 + \beta_2 \) | \( F_1 = D_4 \left( x_4 - x_5 \right) + \beta_2 \frac{d}{dt} \left( x_5 - x_4 \right) \) |
| \( x_5 \) bed frame | \( D_5 \) | \( F_1 = D_5 \left( x_5 - x_6 \right) \) |
| \( x_6 \) rigid floor | | |

Figure Appendix 6-2

| \( F_2 \) (t) | \( y_1 \) feedback device | \( D_1 \) | \( F_2 = D_1 \left( y_1 - y_2 \right) \) |
| \( y_2 \) chest (sternum-to-spine) | \( D_2 + \beta_1 \) | \( F_2 = D_2 \left( y_2 - y_3 \right) + \beta_1 \frac{d}{dt} \left( y_3 - y_2 \right) \) |
| \( y_3 \) chest | \( D_3 + \beta_2 \) | \( F_2 = D_3 \left( y_3 - y_4 \right) + \beta_2 \frac{d}{dt} \left( y_4 - y_3 \right) \) |
| \( y_4 \) mattress | \( D_4 + \beta_2 \) | \( F_2 = D_4 \left( y_4 - y_5 \right) + \beta_2 \frac{d}{dt} \left( y_5 - y_4 \right) \) |
| \( y_5 \) rigid floor | | |
7. Appendix VII (Chapter 15): **Mathematics involved in feedback with accelerometers in chest compressions**\(^\dagger,\)\(^\ddagger\)

The force (F) for the moving element of an accelerometer consists of three parts shown on the right in equation app VII eq.-1, where x is displacement in line with gravity, a term containing the effect of inertia of the mass of cantilever, a frictional term with β as its coefficient of friction and a restoring term with D as the stiffness of the coupling with the attachment of the cantilever to its container.

\[
F = mx'' + \beta x' + Dx \tag{app 7-eq. 1}
\]

Or, after division by m, the measured acceleration signal, a (m/s\(^2\))

\[
a = x'' + \beta/m x' + D/m x \tag{app 7-eq. 2}
\]

Single integration with respect to time yields velocity (v, m/sec)

\[
v = x' + \beta/m x \cdot x + \frac{D/m}{x} dt \tag{app 7-eq. 3}
\]

Accepting that the system is linear and movement is periodic and starts and stops at the same place, the integration in app VII eq. 3 vanishes. Repeated single integration leads to displacement “D” in which the integral is rendered equal to zero by the same conditions.

\[
D = x + \frac{\beta/m}{x} dt \tag{app 7-eq. 4}
\]

Hence, under these ideal conditions,

\[
d = x \tag{app 7-eq. 5}
\]

If the conditions are less than ideal, resort may be taken to data processing techniques.
