rates increasing only slightly over the same 30-beat period. These diabetics had other evidence of vagal damage, with abnormal responses to the Valsalva manoeuvre and reduced R-R interval variation. The results of the drug studies on the three controls confirm that the reflex is mediated through the vagus.

In 1895 Hill suggested that changes in posture might provide a “most delicate test of the condition of the vasomotor mechanism,” yet surprisingly little attention has been paid to the normal heart-rate response to standing. It is well recognised that there is a transient fall in blood pressure on standing, with stimulation of the carotid baroreceptors and consequent reflex tachycardia and peripheral vasoconstriction. Although it has long been known that in normal people the heart rate increases on standing, the immediate heart-rate response has only recently been briefly documented. So far as we are aware, the characteristic pattern that we describe has not previously been analysed in detail.

Although our results were first obtained from an accurate R-R interval analysis by computer, this study shows that heart-rate changes may also be detected with routine electrocardiography. As loss of a normal response is due to vagal damage, this provides the basis for a simple test of autonomic function that has considerable advantages over those now in use. Measurement of the 30:15 ratio gives a simple numerical value that reflects the presence or absence of the relative bradycardia. When the ratio is 1:00 or less vagal damage is probably present, although a value of less than 1:00 does not necessarily indicate more severe damage, as it will occur when there is a slight increase in heart rate over the 30-beat period. In the most severe cases, in which there is no change in heart rate on standing, the value will be exactly 1:00.

This test is simple to use and requires only a standard electrocardiograph and the ability of the patient to stand up. It is not effort-dependent and, so far as we know, cannot readily be “cheated.” It correlates well with other recognised tests of cardiovascular reflex function in diabetes, is objective, requires no special patient co-operation, and is readily applicable as an outpatient procedure.

We thank Dr W G Macfie for allowing us to study subjects from a primary prevention trial of ischaemic heart disease, and Dr P J Watkins for his initial suggestion that we should look at this reflex response.

References
3 Ewing, D J, et al, Clinical Science and Molecular Medicine, 1974, 46, 295.
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SHORT REPORTS

Functions of phagocytic cells in chronic mucocutaneous candidiasis

Chronic mucocutaneous candidiasis (CMC) is a group of uncommon immunodeficiency disorders characterised by chronic infection of the skin, nails, and mucosal surfaces caused by Candida albicans. There are few reports about the functions of phagocytic cells in CMC. This led us to investigate phagocytosis and intracellular killing of candida by granulocytes and monocytes in CMC with a recently developed technique.

Patients, methods, and results

Leucocyte functions were tested in five patients (two men (cases 2 and 5) and three women (1, 3, and 4)) with CMC. In four of these patients the candida infection was limited to the skin, nails, and mucosal surfaces caused by Candida albicans. There are few reports about the functions of phagocytic cells in CMC. This led us to investigate phagocytosis and intracellular killing of candida by granulocytes and monocytes in CMC with a recently developed technique.

Phagocytosis was assessed from the decrease in the number of viable yeast cells caused by clumping of the yeast during the test period. In the most severe cases, in which there is no change in heart rate on standing, the value will be exactly 1:00.

This test is simple to use and requires only a standard electrocardiograph and the ability of the patient to stand up. It is not effort-dependent and, so far as we know, cannot readily be “cheated.” It correlates well with other recognised tests of cardiovascular reflex function in diabetes, is objective, requires no special patient co-operation, and is readily applicable as an outpatient procedure.

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was slightly diminished in one (case 3). Intracellular killing by mono­
cytes showed a heterogeneous picture: either the monocytes were
hyperactive or defective in this respect. The hyperactive killing might
be explained as a compensatory mechanism in chronic candida infec­
tion. The slightly decreased killing by monocytes in case 3 was prob­
ably also due to diminished phagocytosis as our killing assay measures
the overall result of phagocytosis and intracellular killing.

In cases 1 and 2 the killing defect was remarkable because this
defect is serum-dependent. The conclusion that only the disturbance of
intracellular killing is serum-dependent is justified, because the
phagocytosis assay showed no difference between normal and patient’s
serum. The question of whether the serum of these two patients con­
tains an inhibitor or lacks a stimulant is under investigation. In two
patients with CMC an inhibitor of intracellular killing of candida by
granulocytes has been reported.1

We thank Dr C M R Weemaes (Nijmegen), Dr G Hendricks and Dr D B
de Geer (Utrecht), and Dr R Schuurman (Rotterdam) for referring their
patients. This study was supported by the J A Cohen Institute of Radio­
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4 Leijh, P C J, van den Barselaar, M Th, and van Furrh, R, Infection and
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Prophylaxis with deglycyrrhizinised
liquorice in patients with healed
gastric ulcer

Recurrence of gastric ulceration after medical treatment is common,
with recorded rates of 40%,1 and 60%2 in the first two years. The use of
healing drugs to prevent relapse has received relatively little
attention, though carbinoxalone has been used, but with little
success.3 An acceptable prophylactic drug would be of considerable
value in the management of gastric ulceration, and we undertook a
trial of deglycyrrhizinised liquorice (DGL) to assess its merit in
this respect.

Patients, methods, and results

Forty-one patients (23 men, 18 women) with benign chronic gastric
ulceration of healed ulcerous ulcers had been shown both radio­
logically and endoscopically within the previous four weeks, were selected for
this study. All were aged under 75, and women of child-bearing age were
excluded.

The trial was a double-blind controlled study, in which participants
received five capsules a day, each containing either 450 mg of DGL (Ulccdal)
or an identical placebo. A normal diet was allowed and alcohol and tobacco
were permitted in moderation. Antacids were taken as required. We reviewed
the patients monthly for recurrence of symptoms, and a full haematological
and biochemical profile was taken at each visit. Gastroscopy and barium-meal
examinations were performed at six-monthly intervals, or earlier if dyspeptic
symptoms arose in the meantime. Patients were followed up for at least
two years or until the ulcer recurred.

On completion of the study eight patients had withdrawn, leaving 33
patients for analysis, of whom 22 had received placebo and 11 DGL. The
composition of these groups and the ulcer recurrence rate on DGL and
placebo is shown in the table. Eighteen patients developed a further gastric
ulcer: five were receiving DGL and 13 placebo. This represents a relapse
rate during follow-up of 45% for DGL and 59% for placebo. This difference

1 Veterans Administration Co-operative Study on Gastric Ulcer, Gastro­
enterology, 1971, 61, No 4, Part 2, 567.
3 Montgomery, R D, Mehta, S C, and Lawrence, I H, Practitioner, 1969,
202, 398.

(Accepted 29 November 1977)

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Comparative clinical data (ranges given in parentheses) and ulcer recurrence
rates in 33 patients with a healed chronic gastric ulcer treated prophylactically
with either deglycyrrhizinised liquorice (DGL) or placebo

<table>
<thead>
<tr>
<th></th>
<th>DGL</th>
<th>Placebo</th>
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<tbody>
<tr>
<td>No of patients</td>
<td>11</td>
<td>22</td>
</tr>
<tr>
<td>Mean age (years)</td>
<td>61 (44-74)</td>
<td>54 (39-74)</td>
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<tr>
<td>Male</td>
<td>5</td>
<td>16</td>
</tr>
<tr>
<td>Female</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>Mean duration of symptoms (months)</td>
<td>7 (1-15)</td>
<td>7 (1-20)</td>
</tr>
<tr>
<td>Mean duration of treatment (months)</td>
<td>16 (4-29)</td>
<td>12 (5-24)</td>
</tr>
<tr>
<td>Recurrent ulcer (%)</td>
<td>5*</td>
<td>13*</td>
</tr>
<tr>
<td>Recurrence (%)</td>
<td>45*</td>
<td>59*</td>
</tr>
</tbody>
</table>

*Difference not significant.

We thank Mrs E M McCrery and Miss T M Hughes for typing the
manuscript and Boehringer Ingleheim Ltd for supplying active drug and
placebo.

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