Adolescent development

Advice in ABC of adolescence is potentially misleading

Editor—Christie and Viner say that delayed puberty in boys can be quite distressing but is almost always a normal variant. They say that boys aged 15 or over with a testicular volume of 4 ml or more can be reassured that puberty is beginning and, by inference, do not require referral to a specialist. This advice is potentially misleading.

For all that it is a variant of normality, constitutional delay in growth and puberty can have adverse psychosocial and skeletal consequences.¹ To deny an apubertal teenager the opportunity to choose low dose androgen treatment until he is into his 16th year would be unusual by present standards. Given the likely ensuing timescale, his doctor might well refer him straight to an endocrinologist instead of a paediatrician.

A testicular volume of 4 ml is well within the range found in boys with irreversible hypogonadotrophic hypogonadism and therefore by no means necessarily indicates that puberty is beginning. Many boys with hypogonadotrophic hypogonadism start puberty but fail to progress beyond the early stages.² Moreover, a history of cryptorchidism (especially if bilateral) or anosmia should prompt an even earlier referral.

Neither does a family history of pubertal delay necessarily support a diagnosis of constitutional delay in growth and puberty, given the high prevalence of constitutional delay in growth and puberty among first degree relatives of patients with hypogonadotrophic hypogonadism.

A recurring theme in the personal stories posted on the www.Kallmanns.org website by men with irreversible hypogonadotrophic hypogonadism is of just how difficult it was for them as teenagers to screw up the courage to go to see their family doctor about a lack of secondary sexual characteristics. On being told “not to worry, because it’s itself and consequently, the improvement is potential misleading, as 4 ml testes are within the range found in adult men with the uncommon condition of irreversible hypogonadotrophic hypogonadism.

Our advice is appropriate for boys who present to general practitioners. The absence of any signs of puberty, or lack of further progression through puberty over the next six months should, of course, be viewed with suspicion and merit referral to a paediatric endocrinologist service for full investigation, including consideration of conditions such as hypogonadotrophic hypogonadism.

Quinton thinks that even the normal variant of constitutional delay in growth and puberty can have adverse psychosocial consequences.³ However, newer larger studies have shown that boys who are small or whose adolescence is delayed are not psychologically disadvantaged.⁴ Rather than overtreating numbers of normal boys, we focus on improving the communication skills of general practitioners in discussing issues such as pubertal timing with young people.

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Cognitive behaviour therapy for adolescents with chronic fatigue syndrome

Data are insufficient and conclusion inappropriate

Editor—I have concerns about the design and interpretation of the study reported by Stulmeyer et al on cognitive behaviour therapy for adolescents with chronic fatigue syndrome.¹ The trial arms were not matched for the number of contacts with healthcare professionals. Experience from larger and more carefully controlled randomised interventional trials of patients with chronic fatigue syndrome has clearly shown that short term improvement in symptoms is related directly to the maintenance of regular contacts with healthcare professionals rather than the therapeutic effect of the intervention itself and consequently, the improvement is not sustained once the contact has ended.

The authors did not offer patients in their waiting list the opportunity to meet therapists regularly for five months but without having cognitive behaviour therapy. Few follow up data on patients in the intervention arm show that the specific treatment benefit was carried forward without regular contacts with the therapists. A cautious approach is essential in inferring direct benefit from cognitive behaviour therapy in the intervention arm (as opposed to short term benefit from close contact with therapists).

The level of activity in some of their participants whom the authors considered to be passive remained unclear.

In their summary points the authors claim that cognitive behaviour therapy was effective by challenging patients’ belief that activity aggravated symptoms. Epidemiological data, however, confirm that fatigue made worse by exercise is a characteristic feature of adolescents at risk of chronic fatigue syndrome.¹ Encouraging activity in disabled patients is entirely different from challenging an accepted feature of the disease. A rhetorical approach towards a physically and emotionally challenging condition does not help recovery and only encourages therapeutic failure.

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increasing in the United States and around the world. In the context of these increases, we are surprised that methods of care that might prevent caesarean delivery have not been pursued more aggressively.

Caesarean delivery is strongly correlated to the age of the mother, parity, and increasing gestational age within the term period of pregnancy.1 If caesarean delivery is an adverse outcome worthy of prevention, if risk factors for caesarean delivery can be identified, and if a latent period exists between the identification of risk and the development of situations requiring caesarean delivery then perhaps a preventive approach—encouraging patients with risk factors to enter labour before their risk can become disease—could lower caesarean delivery rates safely.

Our working group recently described the use of risk driven, progestin and assisted induction of labour, and this intervention was associated with a rate of caesarean delivery of only 4%.2 While Declercq et al think that research should be done to elucidate whether the risks of primary caesarean delivery in cases of no indicated risk will be offset by associated benefits, we hope that an equal amount of time and effort will be spent on developing and testing methods that might safely prevent, or lower, rates of caesarean delivery performed for this and the other more traditional indications.

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Early epidurals increase caesarean rate, meta-analysis shows

Editor—The study reported by Mayor in her news item uses the term “neuropsychal analgesia” and claims that early epidurals do not increase the rate of caesarean deliveries.3 This is confusing as the study was not of early epidural analgesia, and the oxytocin augmentation rate of 75% at first analgesia makes for lack of generalisability.

The claim that women need not worry to describe a new adverse drug reaction, this syndrome (any more than it can anti- convulsant hypersensitivity syndrome). The description is consistent with serotonin toxicity, a well described adverse reaction to serotoninergic antidepressants. In attempting to describe a new adverse drug reaction, Haddow et al have focused on non-specific clinical features that are present in many drug induced neuropsychiatric syndromes.4 Clomipramine, a potent serotonin reuptake inhibitor, has been associated with hyperthermia and was more correctly labelled a serotonin toxicity.5 Muscle rigidity and raised muscle enzyme activities also occur in severe serotonin toxicity.4

Neuroleptic malignant syndrome is an idiosyncratic reaction to therapeutic doses of neuroleptic agents. A pragmatic clinical
description of the syndrome includes four primary features: autonomic lability, hyperthermia (pyrexia) without other cause, extrapyramidal syndrome (cog-wheel or lead pipe rigidity), and enclephalopathy.\(^1\) Despite superficial clinical similarities between neuroleptic malignant syndrome and serotonin syndrome, they are usually easily differentiated on the basis of careful neurological examination. Neuroleptic malignant syndrome is associated with lead pipe rigidity, bradykinesia, and other extrapyramidal features.\(^1\) Conversely in serotonin syndrome there is hyperkinesia, hyperreflexia, and clonus.\(^1\)

Descriptions of adverse reactions to psychotropic drugs need detailed clinical descriptions of neuromuscular, central, and autonomic features. Using ambiguous or non-specific criteria to label adverse reactions as a particular syndrome while ignoring the pharmacology of the implicated drug may lead to false associations between particular drugs and clinical syndromes and to inappropriate treatment.

**Authors’ reply**

**Editor—Clomipramine is not a neuroleptic and—**

Clomipramine induced neuroleptic malignant syndrome and pyrexia of unknown origin. BMJ 2004;329:1333-5. (4 December.)

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**Risk of gene therapy should be weighed against lack of alternatives for many diseases**

**Levinson and Sternbach and referenced in our article.**

We described in this patient an earlier diagnosed episode of serotonin syndrome, and no clinical evidence of rigidity was found on that occasion.

In view of the action at dopamine sites of clomipramine, and the statement in the BNF from the BMA and the Royal Pharmaceutical Society of Great Britain, we would continue to support our diagnosis of neuroleptic malignant syndrome in this informative case.

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**Editors’ reply**

**—Devereaux et al discussed the need for expertise based randomised controlled trials for surgical procedures.**

Firstly, the use of expertise based designs does not necessarily enhance the validity of a surgical trial. Surgical outcome does not depend solely on the operation; other factors that influence the results of an operation are heterogeneous and immeasurable (postoperative management, the surgical team, equipment). A different bias is introduced by the expertise based design, the influence of the overall performance of the surgeon (A v B), and in this regard, expertise based design is not necessarily a more valid comparison of operation A v B. Secondly, the use of expertise based designs does not necessarily enhance the applicability of a surgical trial. The expertise based design assumes that an operation will only be performed by a select few. This action is recognised in the current edition of the BNF, which says that neuroleptic malignant syndrome may, very rarely, arise in the course of antidepressant treatment.

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both arms in proportions reflective of the population that will perform the operations. Academics can analyse the "expertise" subgroups while the rest of us can look at the overall results to determine how an operation will really perform.

**Letters**

**Target SHOs and registrars for communication skills training**

**Editor**—I suspect that seasoned doctors are better than their junior colleagues at some things. We should be the ones filmed on camera communicating, from pathologists to clinicians with experience, together with "communicators," and not only by people who actually made you a worse doctor is demotivating. Perhaps experienced doctors and patients would have a different set of criteria about what good care is?

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1 Spurgeon D Standard of care by doctors may drop with years spent in practice. BMJ 2005;330:374-5. (19 February)

**Efficiency is important**

**Editor**—In British general practice, where everyday demand exceeds capacity, the efficient general practitioner is king. That is one thing that experience should bring. If all general practitioners followed every guideline the system would collapse, and although a few patients would have exemplary care, many would have no care at all as they would just not get seen because they would find the wait intolerable. Perhaps this is what happens now in secondary care, where care delivered is often very good but access is less and less.

Every doctor needs to learn clinical and communication skills by Devereaux et al. is the norm in psychotherapy research when comparing two different psychotherapies. A similar debate on the interpretation of such trials occurred in the psychotherapy literature.1 Research in surgery and psychotherapy share other similarities beyond having to account for practitioner expertise. There is the issue of blindness—hard to achieve for both patient and doctor in these disciplines—as well as the "why test it, it's obvious it makes a difference" argument. Both disciplines could learn from each other about the design and analysis of clinical research.

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**Old docs and new tricks**

**Seasoned doctors may be better than young doctors at some things**

**Editor**—Spurgeon reports that the doctors' standards of care drop with years in practice.1 When I began clinical practice in the late 1980s I thought that one key to being a "good doc" was keeping up with the latest drugs and technologies. I was dismayed to see seasoned colleagues who were slow to change. I then saw many new drugs get pulled from the market (rofecoxib is not the first non-steroidal anti-inflammatory drug to be withdrawn) and various medical fads come and go. Evidence based medicine appropriately shed light on the poor evidence available to support most things that physicians do.

Armed with this keener analytical approach, I came to realise that most claims of benefit are greatly exaggerated compared with absolute incremental changes and that most patients are not like trial subjects. Although an intervention might benefit a population, it is much less certain that it will benefit the patient who sits before me.

I suspect that seasoned doctors are better than their junior colleagues at some things, and worse at others. Maybe the ability to see the big picture, diagnostically and therapeutically, is enhanced by experience. Meanwhile the emphasis on the newest treatment detail might wane. Perhaps that is a reason why I have conflicting opinions about whether doctors get better with time.

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1 Spurgeon D Standard of care by doctors may drop with years spent in practice. BMJ 2005;330:374-5. (19 February)