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Multiple sclerosis, depression, and suicide

Clinicians should pay more attention to psychopathology

Mental illness leaves patients at risk for harming or killing themselves, none more so than major depression, with which a 15% lifetime prevalence of suicide has been consistently noted.[1] Less clear is how these figures translate when applied to patients with neurological disease, particularly those conditions known to be associated with a high risk of comorbid depression.

An example is multiple sclerosis, the leading neurological cause of disability in young and middle aged adults. Depressive symptoms of sufficient severity and duration to warrant a diagnosis of major depression affect up to half of patients during the course of their illness.[2] This is three times the prevalence reported for major depression and psychiatric comorbidity in community based samples, and it also exceeds that for other neurological disorders.[3] Depression in multiple sclerosis is not linked to a family history of affective disorder,[2] nor is it more likely to occur before the start of neurological symptoms.[4] While detection of brain lesions by magnetic resonance imaging has been shown to correlate with cognitive dysfunction in multiple sclerosis, a correlation with depression has proved more elusive, with evidence for and against being noted. Evidence that increased social stressors and inadequate family and community support are important suggests that depression in multiple sclerosis has a complex, multifactorial pathogenesis.[5]

While depression undoubtedly adds to morbidity, some data suggest it may also lead to suicide. Not all studies agree on this, although those that failed to find an association have been the most methodologically flawed, containing small sample sizes and failing to standardise results for age and sex with control populations. One study that overcame these limitations investigated cause of death in a Danish cohort of 5525 patients with onset of multiple sclerosis between 1953 and 1985. Twice the expected number of suicides was found, with men and those with symptoms starting before the age of 30 years being most at risk.[6] While of concern, these results should be interpreted with caution as the actual number of patients who killed themselves was small (53), and case registries may not always record the cause of death accurately.

Nevertheless, these findings are supported by two other sources: a Medline search of all published data, which shows that patients with multiple sclerosis were generally more likely to attempt or commit suicide than patients with other common neurological disorders,[7] and a Canadian study of 3126 patients with multiple sclerosis who were followed longitudinally at two clinics between 1972 and 1988.[8] Comprehensive databases kept track of virtually all patients within the respective catchment areas, each of whom received at least a yearly follow up examination. Suicide accounted for 15% of all ascertained deaths during this 16 year period, proportionately 7.5 times that for the general population matched for age but not sex. While this figure exceeds that of the Danish epidemiological investigation,[6] failure to control for sex in a disorder with a known female preponderance should again prompt caution when interpreting the findings. The overall picture to emerge is therefore one of a slightly increased risk of suicide probably related to the high prevalence of depression, although none of the studies cited specifically addressed aetiology.

With these statistics, it is surprising that the treatment of depression in multiple sclerosis has received scant attention. There is only one report of a double blind, placebo controlled drug trial showing that desipramine, although modestly effective, produced troubling anticholinergic side effects that limited the dose.[9] Anecdotal reports of fluoxetine being effective and better tolerated suggest that newer antidepressant compounds are probably the treatment of choice. Psychotherapy is a useful adjunct and should not be overlooked. For the acutely suicidal, depressed patient, electroconvulsive therapy remains an option. It may, however, trigger an exacerbation of multiple sclerosis, the presence of contrast-enhancing brain lesions on a pretreatment magnetic resonance scan being a warning sign.[10]

While the past decade has undoubtedly brought a greater awareness of the neurobehavioural sequelae of multiple sclerosis, the risk remains that clinicians may yet miss a treatable cause of morbidity and mortality. The measure by which disability is assessed remains the expanded disability status scale,[11] which affords little weight to psychopathology. Attention remains largely focused on more easily discernable and quantifiable evidence of disease, such as how far patients can walk unaided, the degree of cerebellar disturbance, or measurements of visual acuity. The paucity of studies of treating depression related to multiple sclerosis attests to this. The problem is compounded by new treatment modalities such as interferon beta-1b, in which physical improvement may be offset by a potentially deleterious effect on mood.[12]

While it is premature to conclude that depressed mood represents a core symptom of multiple sclerosis, it has taken psychiatrists and neurologists almost a century to realise that Charcot's astute observation of altered affect in the disorder he helped define demands prompt and careful management.

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Growth hormone: panacea or punishment for short stature?

Learning to live with being short is more important for short normal children

See p 708

After the nutrition dependent phase of fetal and infant growth has ended (towards the latter part of the first year of life) growth hormone secretion becomes the predominant controller of the rate of human growth.[1] Almost any child given growth hormone in sufficient doses will grow more quickly, and dose-response curves for human growth hormone treatment have been with us for some years.[2]

Although much has been written about the predictors of response to treatment, only final height really matters. The French study reported on page 708 by Coste et al is important because it is concerned solely with that end point.[3] As the authors make clear, their study has all the merits (large numbers) and demerits (different data collection staff, reporting errors, different laboratories) of a register based study, but it shows clearly that those patients treated earliest do best—and this is the most important message. Not for a long time has it been believed that growth hormone treatment would restore a normal height to patients unless it were started at a time when there was no growth deficit. The paper is also important because it points out that the treatment of short normal patients in the mistaken ("compassionate") belief that treatment could improve final height is a cruel illusion and an expensive mistake.

So who does need growth hormone? The short answer is those who are deficient of it, but the argument about how to define cut off values for a continuously distributed variable like growth hormone secretion has occupied thousands of journal pages. Such values are as illusory as those for obesity or hypertension. When a child is deficient in growth hormone his or her growth rate falls as the infancy curve of growth comes to its end,[4] and, without growth hormone, a low growth rate leads to extremely short final stature. The earlier growth hormone is introduced, the better will be the result because treatment only enables the height prediction prevailing at the start of treatment to be realised.[5] Any difference between that value and the genetically determined target is not redeemable. Thus, for an affected child to attain a height normal for the family, treatment has to start early and continue throughout childhood. This is why children who acquire growth hormone deficiency later in childhood, when they have already achieved some normal growth, have a better final height than those with growth hormone deficiency recognised by short stature at 3 years of age.

When pituitary growth hormone was withdrawn in 1985 and replaced with the synthetic preparation, clinicians had an opportunity to test a product whose supply was limited only by cost on many other patients with short stature. The results of treatment in patients with these so called wider indications are still debated, but the size and the length of the French study indicate how long we may have to wait for definitive conclusions. Growth hormone clearly has a place in managing short stature in Turner syndrome, renal failure, dysmorphic syndromes, and some skeletal dysplasias, but, because final heights are what matter, trials of treatment in these conditions should still be restricted to centres where long term results can be collated and monitored. The system for central reporting available in France is not universal.

Although many doctors and parents attest to the unhappiness, loss of quality of life, and educational disadvantage of short children, these are not generally identifiable in the community; nor is the functioning of adults who were short as children disadvantaged.[6] Even in a clinic population, we could not identify sufficiently hard psychological end points to test the hypothesis that the increase in growth rate which growth hormone treatment will achieve in short children would be beneficial.[7] In other words, it is much more important for a short child to acquire coping skills than to buy inches through pharmacological means. The many reports quoted by Coste et al attest to a socially unimportant, even if statistically significant, increase in

final height in short children treated with growth hormone. Doctors pressurised into prescribing growth hormone may care to contemplate the aggregation of risk factors for coronary heart disease and the problems of stigmatising otherwise normal children. Growth hormone is contraindicated for normal short children because it is expensive and does little to increase adult stature.

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Occipital plagiocephaly: an epidemic of craniosynostosis?

Craniosynostosis needs to be distinguished from more common postural asymmetry

"A bizarre epidemic ... 400% increase since 1992." "Unkind cut: some physicians do unnecessary surgery on heads of infants."[1] These terms were used last year by the *Wall Street Journal*, in company with the British media, to report that the incidence of posterior skull asymmetry, or occipital plagiocephaly, and its surgical management, had increased to epidemic proportions. Such headlines have resulted in anxiety among parents, general practitioners, and paediatricians, and it is therefore important to be clear about the causes of the asymmetry.

One cause of plagiocephaly is craniosynostosis, which is premature fusion of the cranial sutures—the adaptive fibrous joints between the bones of the skull. The resulting abnormal skull shape is usually an isolated anomaly but it may be associated with a craniofacial syndrome such as that of Crouzon or Apert. The skull shape is predictable from the suture or sutures involved. Premature fusion of the lambdoid sutures, which separate the occipital from the parietal bones bilaterally, may cause occipital plagiocephaly.

Premature lambdoidal fusion is nevertheless rare.[2 3] The apparently increasing incidence of babies presenting with occipital flattening, either unilaterally or bilaterally, has coincided with current advice to nurse infants in a supine position to combat cot death.[4] This phenomenon represents postural, or deformational, plagiocephaly, resulting from unrelieved pressure on the occiput. Asymmetrical influences on the skull base caused by torticollis or cervical spine anomaly may have a similar effect.

In reviewing 75 children presenting consecutively with occipital plagiocephaly to the supraregional craniofacial centre at Great Ormond Street Hospital for Children we have identified some characteristic features of the course of this "epidemic." Two thirds of the infants and children were boys; two thirds presented with a right sided unilateral flattening; and in two thirds this asymmetry had started some time after birth, progressed until around the age of 6 months, and remained stable thereafter. Half the group showed asymmetry of head turning. The delayed presentation and consistent history of asymmetrical head turning is not characteristic of lambdoid synostosis, in which the head shape is abnormal from birth. Postural plagiocephaly, related to the deformational asymmetry of head turning, is a far more likely cause.

Postural plagiocephaly and true lambdoidal synostosis can be distinguished by clinical examination and confirmed by routine radiological investigation.[2] Lambdoid synostosis produces a unilateral occipitoparietal flattening when viewed from behind. The territory of the lambdoid suture presents as a palpable bony ridge, with a bony prominence at the skull base behind the ear on that side, which is displaced inferiorly and posteriorly. The skull base thus seems to tilt to the affected side, giving the head a parallogram shape when viewed from behind. Although the postural group also displays unilateral occipitoparietal flattening, there is no palpable bony ridge of sutural fusion and no bony prominence at the skull base behind the ears, which are level. The ear on the flat side is displaced anteriorly rather than posteriorly and often folded forward. The skull base has no tilt, and therefore the head does not appear as a parallelogram from behind. True lambdoid synostosis is readily distinguishable radiologically, appearing sclerotic on plain radiography and obliterated by bone on computed tomography. The suture remains radiologically open in the postural group.

Why is it important to distinguish the synostotic from the deformational? Clearly the course of the two conditions is different, and this directly affects clinical management. True synostosis is likely to have been present from birth, is progressive, and is unlikely to improve spontaneously. It confers the risk that intracranial pressure may be raised, although this occurs in less than 10% of cases when only a single suture is implicated.[5] Postural plagiocephaly, however, confers no risk of intracranial pressure disturbance, and in over 70% of our series the skull shape improved spontaneously. As the child matures and head control improves, the plasticity of the skull base initially allows the deformity to stabilise and then gradually improve. As scalp hair grows the cosmetic deformity becomes less noticeable. The spontaneous improvement may be encouraged by regular changes in sleeping position,

perhaps helped by orthotics.[6] In severe cases surgical correction may be proposed, but parents should be aware that this would be a cosmetic procedure and not without risk in view of the proximity of the posterior dural venous sinuses.

Thus in most children presenting with an occipital plagiocephaly there will be no craniosynostosis, and even when there is, functional impairment is unlikely. Distinguishing between craniosynostotic and positional plagiocephaly is, however, very important, as surgical intervention for cosmesis is only rarely indicated.

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Child health promotion and its challenge to medical education

Doctors need practical preventive skills they can use in clinical settings

The recently published recommendations for a national programme of child health promotion[1] provide a structured framework for addressing the primary prevention of many of the major causes of illness and disability in preschool children. This is the latest in a series of important statements from the British Paediatric Association (now the Royal College of Paediatrics and Child Health) working party on child health surveillance.[2,3] It repeats earlier calls for a targeted programme of secondary prevention measures, selected on the basis of evidence of efficacy, together with a greater emphasis on health promotion. Taken together, these reports represent a major change in the role of community child health services away from mechanistic attempts at early detection of developmental and other problems towards a more holistic approach to child health. They also have important implications for the way that clinicians are trained.

Perhaps the most radical departure is the suggestion that the term child health promotion is a more appropriate title for the programme than child health surveillance, which would be that part of the programme concerned with secondary prevention by early detection.[1] The report emphasises the need for more resources for primary prevention and the opportunities for health education which could be given by doctors at various stages of the life cycle, beginning in pregnancy (and preferably before conception) and continuing throughout childhood. Examples include giving advice about immunisation, reducing the risk of cot death, encouraging breast feeding, dental prophylaxis, avoiding passive smoking, avoiding behaviour problems, and accident prevention. If accepted by the relevant professionals the proposed child health promotion programme will require those working in community child health services to develop skills in individually oriented health promotion techniques, notably health education, along with the more familiar forms of clinical prevention such as immunisation and screening. Inevitably this has implications for the content of undergraduate and postgraduate medical education.

In most medical schools health promotion is covered, to a greater or lesser extent, in the public health course. In Britain and many other countries this teaching tends to be self contained, with few points of contact with other disciplines. Indeed, public health has become so separated from clinical instruction that its relevance in the medical course has been questioned.[4] As a result most medical students are unlikely to acquire the knowledge and skills necessary to apply epidemiological and preventive principles to clinical settings such as child health promotion. As postgraduates too, doctors have few opportunities to undertake training which includes a strong element of clinical prevention. To date the input of public health disciplines to the training programmes of community based clinical specialties (including community child health and general practice) has been minimal.

The General Medical Council (GMC) appears to have recognised the need to reinforce the role of public health in the training of tomorrow's doctors.[5] The main challenge facing medical educationalists is one which the GMC may have underestimated: to provide students with practical preventive and health promotional skills that they can use in clinical settings, rather than merely theoretical knowledge of epidemiology and related fields.[6] This is a difficult task that calls for an injection of ingenuity and innovation into the teaching of both public health and clinical practice to emphasise their interdependence as well as their differing perspectives. Role models are few, but the integration of epidemiological and public health teaching with clinical instruction has been achieved successfully at schools in Canada,[7] the United States,[8] Israel,[9] and elsewhere.

With few exceptions, British medical schools have been reluctant to accord public health a central presence in the clinical curriculum. Part of the blame

may lie with public health teachers themselves, many of whom regard their subject as exclusively population based and therefore outwith the clinical domain. These attitudes must change urgently if all British children being born in the 1990s are to benefit from the range of preventive, educational, and clinical skills that will be required of doctors to meet the objectives of child health promotion and other preventive programmes. The first step is for those responsible for developing undergraduate and postgraduate teaching in paediatrics, general practice, and public health to recognise the necessity to work together to help students acquire these new clinical skills. The GMC's initiative presents a rare opportunity for curriculum planners to respond positively and imaginatively to the challenge.

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The future of vascular services: the need for a strategy

Which could also be a model for other specialist services

How big should an acute general surgical hospital be and what population should it serve? Patients want a local service, but technological advances, increasing expectations, and escalating costs make this difficult. Vascular surgery provides an important benchmark against which to judge the optimum size of acute surgical units.

Since the 1960s vascular surgery has been one of the most rapidly expanding specialties. Indeed in 1994-5, 25% of general surgical appointments requesting a special interest were for a vascular surgeon (the next most popular request was colorectal surgery, at 10%). Patient morbidity and mortality have been dramatically reduced by (expensive) improvements in anaesthetic and perioperative care. A better understanding of the widespread nature of the disease has led to close cooperation between surgeon, cardiologist, renal physician, and neurologist. Furthermore, interventional radiologists have made an enormous impact over the past 20 years with balloon dilatation, thrombolysis, and more recently, the placing of covered stents. So why, chief executives may be asking themselves, are they now being asked not to reappoint singlehanded vascular surgeons? The answer, briefly, is that there has been a lack of strategic planning which we must now address.

Vascular surgeons have three main goals: to prevent amputation (an aggressive approach to distal reconstruction is effective in terms of both quality of life and cost[1]); to prevent stroke in patients with carotid disease[2]; and to prevent ruptured aneurysms.[3] To do this we need all the facilities of a full vascular service. At the moment a district of 250 000 people is lucky to have two vascular surgeons and a vascular radiologist. It is unreasonable to expect these three individuals to provide the cover all the time, particularly since 42% of vascular interventions take place out of hours.[4] For four months of the year such a hospital would be covered by a singlehanded vascular surgeon.

A pragmatic immediate solution may be a "hub and spoke" arrangement so that patients are transferred to a more central unit when no local surgeon or radiologist is available. In more sparsely populated areas, such as the Highlands, Northumberland, Cumbria, and East Anglia, transfer over long distances has logistic problems and this type of cooperative service may be the long term solution. Most patients in Britain, however, are in urban areas and we should coalesce units so that a critical mass of expertise is provided.

In 1990 only four units in Great Britain had three vascular surgeons; in 1995 there were 24. We are clearly moving in the right direction, and, with the consultant:trainee ratio continuing to fall, a 1 in 4 emergency on call rota for consultants seems reasonable. These four surgeons need an appropriate workload, and three local audits in Great Britain produced a figure of 90 operations per 100 000 population per year (Belfast, Bournemouth, Sheffield; personal communications), which is identical to the figure in the Swedvasc registry in Sweden.[5] On this estimate the four vascular surgeons require a population of about 600 000 to provide an efficient service. If the unit is smaller the cost per case increases and the facilities are underused. Furthermore, there will be times during the year when a true vascular service is not provided.

Obviously the development of larger units requires considerable strategic planning, which is difficult within the current system,[6] where hospital trusts tend to compete rather than cooperate. Many hospitals will not wish to lose their vascular service; others will not wish to be swamped by a major unit.

There has been a plethora of advertisements for vascular surgeons for relatively small units, and these young surgeons will take up their task with enthusiasm. When

they realise the chief executive's inability to provide the necessary facilities they may become dispirited. Vascular surgery is a young, dynamic, and rapidly expanding specialty that requires careful strategic consideration if we are to provide the high standard of care that patients with this endemic disease should expect.

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Authorship is dying: long live contributorship

The BMJ will publish lists of contributors and guarantors to original articles

In April this year we suggested that the concept of authorship in science was so broken that it should be scrapped and replaced by something different.[1] Instead of authors there should be contributors and guarantors. Since then the debate has begun to motor. The originator of the idea of contributors—Drummond Rennie, deputy editor (west) of *JAMA*—has spelt out the case for change in detail.[2] The *Lancet* has adopted the system.[3] Many people have written to us about the subject, and most favour change: today we publish 16 of their letters (p 744).[4] We propose that, where the authors want it, we will publish lists of contributors and guarantors with papers describing original research. When authors choose not to do this, however, we will continue to publish lists of authors in the traditional way.

We will need to experiment with exactly how the new system will work. Rennie et al suggest that all contributors should meet and agree a description of who did what.[2] This might be detailed—as in the paper by Rennie and others—or brief, as has been the case with most *Lancet* papers. The optimum probably lies between the two. One principle is that somebody should accept credit and accountability for every part of the process, including having the idea, undertaking a literature search, design, collecting and analysing the data, interpreting the results, and writing the paper.

Contributors will also have to decide where to draw the line between contributors and those who will be acknowledged, but we join Rennie et al in suggesting that contributors should include all those who "have added usefully to the work."[2] They might include somebody who suggested the idea and design for the study but did nothing further, or somebody who collected many of the data but was not concerned with design or analysis. Rennie et al suggest that contributors should agree on the relative size of their contributions to decide on the order in which they will be listed. Those who have contributed most will come first. We are not convinced that this is necessary but will be happy to be guided by the experiences of contributors. If researchers want to be listed in order of size of contribution we will do so, but we won't insist on it. The current Vancouver guidelines point out that readers should infer nothing from the order of authors since conventions differ.[5] This warning should probably still apply to lists of contributors, at least for now.

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Contributors will be fully responsible for their contribution, but at least one person—the guarantor—needs to accept accountability for the whole work. "Guarantors," say Rennie et al, "are those people who have contributed substantially, but who have also made added efforts to ensure the integrity of the entire project. They organise, oversee, and double check and must be prepared to be accountable for all parts of the completed manuscript, before and after publication."[2] The *Lancet* has not adopted a system of guarantors, but we agree with Rennie et al that at least one person must take overall responsibility. There have been too many cases of fraudulent research where nobody accepts responsibility. But we are not convinced that guarantors have to have double checked every aspect of the research. Will this be possible if the study includes molecular biology, statistics, and economics? More controversially, does the guarantor necessarily have to have contributed substantially? Mightn't the guarantor sometimes be the person who hired all the researchers even if he or she hasn't contributed much to the particular study? In other words, guarantorship might be akin to ministerial responsibility. The role of the guarantor will become most important when there are serious questions about the integrity of the research. One thing a guarantor might therefore want to do, and which in turn will lend strength to the role, is to satisfy himself or herself that standards of research ethics and supervision are high in the departments where the contributors come from.

In moving from authors to contributors and guarantors we are entering a new era, and it seems wise not to be too prescriptive. We need to learn from experience and adapt the new system. We look forward to working with contributors to improve both the credit and the accountability of published research.

Richard Smith *Editor, BMJ*

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