Outbreak of legionnaires' disease in the United Kingdom. Vigilance must be eternal but balanced

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Local funding would reduce waiting lists for cataracts

Editor—In her editorial on recruiting overseas doctors Rosen makes several important points.1 We have been informed by the strategic health authority for Avon, Gloucestershire, and Wiltshire Strategic Health Authority that many patients with cataracts from Bristol Eye Hospital will have surgery carried out at a local district general hospital by a European team. Our nursing staff were asked to provide information about the number of “straightforward” cataract cases on our waiting list. We expressed a willingness to carry out this work ourselves and were told by the Department of Health that bids to carry out surgery to reduce numbers on the waiting list would be favourably received. Our highly competitive bid was, however, turned down, without having ever been looked at, despite having the obvious advantages of audit, appraisal, and continuity of care.

Bristol Eye Hospital has consistently been at the forefront of innovation in ophthalmology and cataract surgery in particular.1,2 We have met all our “Action on Cataract” targets and increased our annual cataract throughput by 60% in the past 18 months. We have repeatedly applied to do more cataract surgery but have been unable to do so because funding has not been available.

It is difficult to maintain staff morale and motivation when our local surgical teams see funds that we have repeatedly requested for delivering this work.

Richard Harrad clinical director Bristol Eye Hospital, Bristol BS1 2LX r.a.harrad@bristol.ac.uk

On behalf of the 14 consultant ophthalmologists at Bristol Eye Hospital.

Competing interests: The Bristol Eye Hospital wishes to be considered in open competition for delivering this work.

1 Rosen R. Recruiting overseas doctors. BMJ 2002;325:290-1. (10 August.)

Outbreak of legionnaires’ disease in the United Kingdom

Vigilance must be eternal but balanced

Editor—Joseph underlines the paradox of larger outbreaks of legionnaires’ disease when understanding of causality is greater than ever. She gives four explanations—loss of vigilance in maintenance of water systems, greater clinical awareness, better surveillance, and easier diagnosis. She calls for enhanced surveillance of both sporadic disease and outbreaks and for greater vigilance in control. Some lessons from studies of legionnaires’ disease in Scotland are pertinent to concerns fuelled by outbreaks in England this summer.

In Glasgow a survey conducted after two outbreaks, including the largest in the United Kingdom up to 1984, showed up difficulties in maintaining an accurate register of cooling towers, poor understanding among some managers of premises about the nature and location of cooling towers and evaporative condensers, and breaches of guidelines, usually on structural issues—for example, control of the drift of cooling towers rather than non-use of chemicals. The problems would have been even greater without the publicity of the preceding outbreaks. Breaches of guidelines on the maintenance of hot water systems were also of concern and constituted a hazard for legionnaires’ disease.

Apparely sporadic cases were often part of mini-clusters.1 The conclusion, anticipating that of Joseph, was that surveillance needed strengthening and that solitary cases needed investigation promptly for potential early warning of an outbreak. Information crucial to surveillance—address, postcode, and date of onset—was often missing from laboratory request forms, which contribute to surveillance. Clinicians must understand why such information is needed so they are motivated to provide it.

Studies of sporadic disease suggested the sources of infection were similar to those for outbreaks, for the epidemiological patterns were similar, with proximity of the home to a cooling tower being a risk factor.2 The costs of maintaining water systems—both financially and in terms of environmental contamination—are high, so choices need to be made. Preliminary economic analysis showed the emphasis needs to be placed on maintenance of cooling towers rather than domestic water systems, but more work is needed on this.

Elimination of legionnaires’ disease is not achievable, so vigilance combined with a balanced response based on an understanding of costs and benefits is required—neither panic nor media pressure should drive priorities. These lessons based on studies in the 1980s remain relevant to understanding and controlling the outbreaks that have gripped the nation.

A longer version of this letter with a complete list of references is available at bmj.com/cgi/letters/325/7360/347#25051.

Raj S Bhopal professor of public health
Public Health Sciences Section, Department of Community Health Sciences, University of Edinburgh, Edinburgh EH8 9AG
rajbopal@es.ac.uk

We have used extracorporeal membrane oxygenation in 16 adult patients with the most severe form of legionella infection between 1989 and 2001. Their modal ratio of pulmonary artery oxygen content to fractionized inspired oxygen ratio before oxygenation was 8.7 kPa (range 4.1-27.1 kPa), 13 were male, and their mean age was 43 years (SD 10.6). They all received venovenous extracorporeal membrane oxygenation for a mean time of 258 hours (SD 235 hours).

Survival to hospital discharge was 69%, with 11 of the 16 patients surviving at six months. This is similar to the 66% survival that we have reported for adult patients with a variety of respiratory diagnoses.3

All extracorporeal membrane oxygenation of adults in the United Kingdom now falls within the remit of the CESAR trial (www.cesar-trial.org). To be eligible for the trial, patients must be aged between 18 and 65, have a Murray lung injury score of >3.0 and a duration of high pressure or high oxygen ventilation of <7 days. We recommend that any patients with severe legionella infection who are deteriorating optimally after conventional intensive care should be considered for the CESAR trial.

Nikki C Jones
CESAR trial research fellow
Glenfield Hospital, Leicester LE3 9QP
nikki.jones@ulg-tr.nhs.uk

On behalf of C J Peck, N Roberts, C Harvey, A W Somosowski, H M Killer, R K Firmin (Glenfield Hospital); D Jenkins (Leicester General Hospital); and A Truesdale, D Elbourne (London School of Hygiene and Tropical Medicine).


Antenatal screening policies for Down’s syndrome

Audit of Down’s syndrome screening is not valid

Entorr—Wellesley et al are unjustified in their view that serum screening for Down’s syndrome is not worth while.1 They ignore evidence from previous studies that shows the substantial advantage of serum screening over screening based on maternal age alone.2-4 Their view is based on the results from two districts, the only two out of the eight where serum screening was routinely offered (using the Double test).

In these two districts only 24% of affected pregnancies (22 of the 91) were detected antenatally by using serum screening, though the Double test has a detection rate of 58% for a false positive rate of 5%.1 This could, at least in part, be due to a low screening uptake (in which case the focus should be on why this was so), but the uptake of screening cannot be determined from the paper. It could also be due to affected pregnancies that were positive on the serum test and positive on other tests being classified as positive under the other tests.

This problem arises because the detection rate used in this paper includes women who declined screening (the performance of screening tests, the uptake of screening, and the uptake of amniocentesis should all be reported separately). Methods of detection (screening, scan, maternal age, etc) were also tabulated in mutually exclusive categories, which cannot be correct because there will be pregnancies positive for Down’s in two or more of these categories.

Wellesley et al compare screening results across different districts. In doing so they cannot (through lack of data) consider the substantial variations between districts in important factors that influence screening such as maternal age, uptake of screening, and use of ultrasonography. This problem is solved by comparing different screening methods in the same women—each woman acting as her own control. There is no better controlled study. Had they performed such an analysis, as we suggested in response to their earlier paper,3 they would have confirmed the substantial advantage of serum screening over age screening. Their call for controlled trials is unjustified.

Wellesley et al are incorrect in saying that the mathematical modelling carried out by others to estimate the effect of serum screening assumes that only 5% of pregnant women were aged over 35. Such modelling takes account of the maternal age distribution of the relevant population. The paper does not specify the performance of individual screening methods and does not assess the benefits of one over another. It is impossible to draw any valid conclusions about the different methods of screening for Down’s syndrome from it.

Nicholas J Wald
professor
mjwald@gmul.ac.uk

Wayne Huntly
screening manager
Allan K Hackshaw
statistician
Wolfson Institute, Barts and the Royal London School of Medicine and Dentistry, London EC1M 6BQ

1 Wellesley D, Boyle T, Barber J, Howe DT. Retrospective audit of different antenatal screening policies for Down’s syndrome in eight district general hospitals in one health region. BMJ 2002;325:15. (8 July.)


Serum screening for Down’s syndrome is better than age screening

Entorr—Wellesley et al performed a retrospective audit of different policies of antenatal Down’s syndrome screening in eight hospital districts and concluded that serum screening is not significantly better...
than age screening." This conclusion contradicts the international experience, and cannot be made from the reported data.

The activities in the different districts do not reflect the policies stated—for example, a policy, as in district A(1), in which serum screening is offered without a routine anomaly scan resulted in 11 out of 33 detected fetuses with Down's syndrome being found through an anomaly scan. Was serum screening performed in these patients? For each test we lack information on the number of women tested and the detection and screen positive rates for each test. Without this, a comparison between different policies is impossible.

We believe that a mistake by not correcting for the substantial death rate in utero when calculating the total prevalence. This is important when cases of Down's syndrome may be detected as early as week 12 by chorionic villus sampling and as late as week 24 from an anomaly scan.

Screening performance obviously depends on the age distribution of the examined population. The performance of age screening can be calculated from the age distributions of pregnant women and is certainly inferior to the performance of second trimester serum screening, well documented in prospective settings and in audit reports from the United Kingdom. Modelling studies have not presumed that 5% of pregnant women were older than 55 years, the proportion, unsurprisingly, being 15%.

Screening also reduces the rate of invasive procedures in older women.

Detection rates are partly based on cases of Down's syndrome in which mothers refused prenatal testing. In district A.A among women older than 55 years, nine babies with Down's syndrome out of 31 registered were not detected antenatally because their mothers refused the test and two because serum screening failed. The detection rate is therefore 20 detected cases from 22 total cases=91% in the screened population and not 65%. One dare not think of the poor performance of the tests if a greater part of the population had refused to participate in the screening.

We hope that Wellesley et al will not confuse the issue and delay the abandonment of the obsolete age screening that is causing an unacceptable number of unnecessary procedure related abortions of healthy fetuses.

Michael Christiansen consultant
nic@sst.dk
Sverre Olesen Larsen biostatistician
Bent Nærgaard-Pedersen professor
Department of Clinical Biochemistry, Statens Serum Institut, 5 Artillerivej, DK-2300 Copenhagen S, Denmark

Authors' reply

Entror—Studies of screening policies for Down's syndrome provide answers for two constituencies. The first is the individual woman, who wishes to know if she accepts it, how likely the test is to detect an affected fetus and how likely she is to be offered an invasive test. The second is public health policy makers, who must decide whether introducing screening on a population wide basis will achieve its goals of increasing the detection rate of Down's syndrome or reducing the invasive procedure rate.

The studies to which Wald et al refer, and their suggestion that women should act as their own controls, answer the first question, but not the second.

Christiansen et al correctly point out the disparities between policies and practice. The purpose of our study was to audit screening in day to day practice, where theory is disrupted by reality. A reality that includes routine anomaly scanning, women declining serum screening, older women opting for the certainty of an invasive test rather than screening, women declining amniocentesis offered on the basis of their age or serum screen result, etc. In this reality women choose not to behave as expected, hence screening programmes do not perform as predicted by theoretical models.

Wald et al correctly say that higher uptake of screening would improve the detection rate, but we should guard against coercing women into having the test. Good counselling allows women to make a fully informed choice, and this may lower uptake. Not every mother agrees that it is desirable to identify and terminate a fetus with Down's syndrome. Women offered prenatal screening found it difficult to decline because they felt there was an expectation they should participate.

Another study of serum screening found that only 38% of women having the blood test understood they were being screened for Down's syndrome. In an ethnically diverse population in the West Midlands only 35% of Asian women with a high risk result opted for amniocentesis. This implies that these women did not understand the full implications of the screening test when the blood sample was taken and that better counselling would have lowered the uptake.

Our study shows that many factors extraneous to the screening test itself modify the performance of a population programme, with much greater effects than the small differences achieved by different combinations of serum analytes. We stand by our recommendation that screening programmes should be tested in controlled trials.

David Howe consultant in foetal medicine
Diana Waddington associate specialist in clinical genetics
Princess Anne Hospital, Southampton SO16 5YA

Confusing myopia with hypermetropia is dangerous

Entror—The front cover of the BMJ on 18 May 2002 included an important error. Above the headline, “Myopia: does reading damage your eyes?” was a photograph of a man and boy with hypermetropia, the opposite condition. Hypermetropia, or long sightedness, is corrected by spectacles with convex (magnifying) lenses that make the eyes appear larger and as shown in the photograph. By contrast, myopia (near sightedness) is corrected by concave lenses, which make the eyes appear smaller.

Figure 1 of the article itself showed a girl wearing myopic spectacles, though the degree of myopia was only modest, about —2 D and certainly not the high (pathological) myopia referred to in the legend. Indeed, the legends for figures 1 and 2 seem to have been transposed.

Doctors need to be able to distinguish hypermetropia from myopia. People with hypermetropia are at increased risk of developing acute angle closure glaucoma, an unpleasant and sight threatening condition. In predisposed eyes acute angle closure glaucoma may be induced by eye drops that dilate the pupils or by drugs with pupil dilating side effects such as many antihypertensives. By contrast, iatrogenic angle closure is almost unheard of in people with myopia. We teach our students to look at a patient’s spectacles as the first step in assessing the risk of iatrogenic acute angle closure glaucoma. For further practical help in assessing this risk, non-ophthalmologists should consult a general textbook.

Martin Barnes Senior house officer in ophthalmology
Leicester Royal Infirmary, Leicester LE1 5WW
martin.barnes@doctors.org.uk

Tom Eke consultant ophthalmologist
Norfolk and Norwich University Hospital, Norwich NR4 7UZ

Myopia

1 Wellesley D, Boyle T, Barber J, Howe DT. Retrospective audit of different antenatal screening policies for Down’s syndrome in eight district general hospitals in one health region. BMJ 1997;325:15 (6 July).

References

1 Sjogren B, Uddenberg N. Decision making during the prenatal diagnostic procedure. A questionnaire and interview study of 211 women participating in prenatal diagnosis. Prenatal Diagnosis 1988;8:265-73.

Myopia

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Martin Barnes Senior house officer in ophthalmology
Leicester Royal Infirmary, Leicester LE1 5WW
martin_barnes@doctors.org.uk

Tom Eke consultant ophthalmologist
Norfolk and Norwich University Hospital, Norwich NR4 7UZ

References

1 Sjogren B, Uddenberg N. Decision making during the prenatal diagnostic procedure. A questionnaire and interview study of 211 women participating in prenatal diagnosis. Prenatal Diagnosis 1988;8:265-73.
Convergence might cause myopia

EDITOR—In his article on myopia Fredrick said that most research into myopia has been limited by its retrospective nature and lack of control group and follow up.1 We conducted a prospective, controlled, three year follow up study of myopia and showed direct evidence of myopic shift in students reading and doing intensive near work compared with children who were not attending school, schoolchildren who did not read much, and skilled manual labourers.2

Fredrick mentioned three possible causes for the development of myopia: retinal blur, accommodation, and familial factors. A fourth possibility exists: convergence. Convergence, rather than accommodation, could be an important factor in myopic progression.3,4

In their three year follow up study Perssiner et al showed that neither the use of bifocals nor avoiding the use of myopic spectacles in reading slowed down myopic progression.3 Perssiner and Lyrya found more myopic shift in subjects needing less accommodation than in those needing more.7 They concluded that if accommodation played a significant part in myopic progression, reading with undercorrected glasses or without glasses would probably halt the process through a feedback mechanism. In our study we observed a significant axial length elongation during near fixation both with and without cycloplegia (with and without accommodation).3 These results do not support the hypothesis of accommodation as a significant cause of myopia. Rather, axial elongation during near focusing suggests that convergence may be one factor inducing myopia. Perssiner et al and Perssiner and Lyrya thought that constant saccadic eye movements during reading could cause repeated pressure and stretch pulses on the eye.

I think therefore that axial elongation, which is a main cause of myopic progression, seems to result from the effect of accommodative convergence rather than accommodation itself. Much use of convergence may be one of the contributing factors in adult onset and adult progression of myopia.

Huseyin Bayramlar
associate professor in ophthalmology
Inonu University, Turgut Oral Medical Centre, 44350, Malatya, Turkey
hbayramlar@yahoo.com

Message about hormone replacement therapy is unclear

EDITOR—Two contrasting leading articles followed publication of the randomised trial of the women’s health initiative study of hormone replacement therapy.1,2

Stevenson and Whitehead in the BMJ said that the increased risk of breast cancer in the study was small, but they did not mention that during the study 42% of women taking active drug and 38% receiving placebo stopped the assigned treatment.3

In contrast, Fletcher and Colditz reported in JAMA that the intention to treat analysis may have underestimated the true effects.4

We conclude that Fletcher and Colditz’ results present a picture of our case is most likely an idiosyncratic drug reaction, and yet they describe our report as “careless talk” and “careless talk that may cause harm.” We refute these reports.

Firstly, Green et al agree that the clinical picture of our case is most likely an idiopathic drug reaction, and yet they describe our report as “careless talk,” which is rather contradictory. They indicate that to confirm this association the patient should be rechallenged with the drug and the reaction documented. This is certainly true but not practical in clinical practice. Many physicians would consider it unethical to rechallenge a patient with a drug that they consider to be harmful. They do not believe that patients would consent to such an experiment.

We were very careful in our concluding sentence in the report to suggest a possible association between ticlopidine and acute arthritis, the consequences may include not providing optimal treatment. Our job as a scientific medical community is not to develop a good or an erroneous reputation for a given drug but to report facts and try to interpret them.

We were very careful in our concluding sentence in the report to suggest a possible association between ticlopidine and acute arthritis rather than to confirm this association. Educated physicians do not build the clinical practice on one or two case reports. The fact that three patients developed acute arthritis out of hundreds of thousands of patients taking an ADP receptor antagonist should not prevent doctors from prescribing these extremely beneficial drugs in cardiovascular disease. Similarly, the 0.5–1.0% risk of intracranial bleeding associated with thrombolysis did not prevent doctors using it for acute myocardial infarction.

Clinical practice is based on an assessment of both the benefits and risks of drugs.

1 Stevenson JC, Whitehead MI. Hormone replacement therapy. BMJ 2002;325:113-4. (20 July)
Prophylaxis for early onset group B streptococcal sepsis is not so effective in practice

Editor—Oddie and Embleton highlight the dangers of and risk factors for early onset group B streptococcal sepsis.1 They analysed their data retrospectively and estimate that most cases (78%) could have received effective antenatal or intrapartum prophylaxis but accept that this interpretation might be an overestimate.

This maternity unit recently introduced a protocol for selective intrapartum prophylaxis against group B streptococcal infection on the basis of recognised clinical risk factors and subsequently audited our practice with respect to whether eligible women received adequate prophylaxis or not. Over an initial four week period 57 (10.3%) of 550 women were eligible for antibiotic prophylaxis, but only 12 (22%) of those eligible received adequate prophylaxis. After minor changes to the protocol and staff education, a re-audit over a subsequent four week period identified 49 (13%) of 378 women eligible with 20 (42%) of those eligible receiving adequate prophylaxis. This represents a modest improvement.

This audit represents clinical practice in a teaching hospital delivery suite. When guidelines for prophylaxis against group B streptococci are being designed, the difference between that which might be achieved (78%) and that which is achieved (32% to 42%) needs to be recognised.

Philip Owen consultant obstetrician Princess Royal Maternity Unit, Glasgow G31 2ER philipowen1@hotmail.com


Antibiotic prophylaxis after CSF leaks lacks evidence base

Editor—Sanarturs et al in their Minerva article perpetuate the unconfirmed and potentially harmful myth that leaks of cerebrospinal fluid should be treated with antibiotic prophylaxis.2 This lacks an evidence base, and there are sound practical reasons why this practice will not be effective.

This issue was addressed by a working group of the British Society for Antimicrobial Chemotherapy.3 The reasons put forward were, firstly, that commonly used antibiotics such as cephalosporins penetrate the non-inflamed meninges poorly, and secondly, that antibiotics are unlikely to eradicate potential pathogens such as the pneumococci from the upper respiratory tract. Conversely, treatment may lead to colonisation with strains that are resistant to antibiotics—for example, to penicillin.4 These may then more easily treatable sensitive strains in any future episodes of meningitis. Published reviews have not shown that prophylactic treatment is effective, and as such it should be discouraged.

Resistance of microbes to antimicrobial agents is rising throughout the world. Indiscriminate use of antibiotics is often to blame and is associated with bacterial super-infections by organisms such as methicillin resistant Staphylococcus aureus, antibiotic associated diarrhoea, and drug specific side effects.

Reduction in the use of antibiotics may not reverse this trend in rising resistance but will attenuate it as well as protecting patients against drug side effects and healthcare system costs against the financial costs. Prudent and appropriate prescribing of antibiotics must be the ultimate goal.

James R Greig consultant microbiologist Derriford Hospital, Plymouth, Devon PL6 8DH jrgreig@yahoo.co.uk

1 Santarius T, Amour NM, Kirolos RW. Minerva. BMJ 2002;325:554. (7 September.)

Epidurals and backache: again?

Editor—Anaesthetists have reason to be grateful to the BMJ for publishing yet another trial, albeit a small one, showing that epidural analgesia is not associated with long term backache.5 On each occasion this outcome seems to come as a surprise, so the finding is worth repeating. The BMJ published with alacrity several retrospective studies that gave the erroneous result that epidurals did cause backache, but it took more persuading to publish prospective studies with negative results,6 and it flatly refused to publish one showing epidurals were good for babies—good news is no news.

Readers may find it useful to know that further clinical details of this same study can be found in an earlier publication by Howell et al, in which 184 women were randomised to epidural and 185 to non-epidural analgesia with primary outcome measures as backache 3 and 12 months after delivery.7 The authors say that crossover between treatment groups is inevitable in such trials. Not so. Researchers in the University of Texas Southwestern Medical Center, Dallas, have published a series of trials in which a total of 3727 women were randomised to receive either epidural or systemic analgesia. They made progressively more successful efforts to improve analgesia in the non-epidural arm, by the use of generous patient controlled analgesia regimens. In the latest study the crossover amounted to only 3.1%.8

The cover picture relating to this article is misleading. The epiduralist should be wearing a mask and, in the United Kingdom, would usually be wearing a gown (intuitively safer, but not evidence based). The caption: “Do epidurals cause long term backache? No more than other forms of pain relief in labour” is also misleading. It is not the pain relief that causes backache—it is having a baby.

Felicity Reynolds emeritus professor of obstetric anaesthesia St Thomas’s Hospital, London SE1 7EH felicity.reynolds@btinternet.com


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Growth hormone in growth hormone deficiency

Ignore the evidence and keep going wrong

Editor—We were surprised that in his editorial to our paper Saenger challenged our conclusions that most patients treated for growth hormone deficiency do not have this condition, and that controlled trials should be organised to evaluate the long term effects of growth hormone in most of the patients currently treated.1 Saenger supports the use of an integrated approach to diagnosing growth hormone deficiency and the wider use of IGF-1 measurements, as suggested by the Growth Hormone


Deficiency cannot be diagnosed solely on the results of stimulation tests

Enrort—We agree with Carel that many children deny the concept of idiopathic growth hormone deficiency do have this condition. We found that most children with idiopathic growth hormone deficiency had a normal growth hormone response in stimulation tests when restated at the attainment of final height. Only patients with growth hormone deficiency whose results on magnetic resonance imaging showed anatomical abnormalities of the hypothalamic-pituitary area had permanent growth hormone deficiency. All the others had normal growth hormone secretion.

Furthermore, we recently found that among 33 prepubertal children with a biochemical diagnosis of growth hormone deficiency (a growth hormone response to two stimulation tests <10 μg/ml, 28 had a normal response when restated after two to six months). Normalisation of the growth hormone response to stimulation occurred irrespective of the time interval from the first evaluation and was not related to puberty since none of our patients had entered puberty before re-evaluation. This finding does not support the concept of a transient growth hormone secretory defect that improves with the pubertal secretion of sex steroids.

We and others have shown that concentrations of IGF-1 and IGFBP-3 are invariably reduced in patients with severe, unequivocal growth hormone deficiency. This indicates that subnormal concentrations of IGF-1 and IGFBP-3 can be considered diagnostic of growth hormone deficiency, provided other causes of reduced IGF-1 or IGFBP-3 such as malnutrition, hypothyroidism, renal failure, and diabetes are excluded. This observation, coupled with the fact that IGF-1 concentrations do not correlate with the results of growth hormone stimulation tests, implies inadequate assessment of growth hormone secretion and emphasises the need for careful evaluation of short children and eventual revision of the criteria for diagnosis.

Although biochemical tests for growth hormone secretion clearly distinguish children with severe growth hormone deficiency of pituitary origin, recognition of more subtle forms of growth hormone insufficiency still represents a diagnostic dilemma. Idiopathic growth hormone deficiency cannot be diagnosed solely on the results of stimulation tests but requires evaluation of multiple biochemical and clinical or auxological variables.

We are convinced that patients with pathological growth hormone responses to provocative tests but normal results on magnetic resonance imaging scans of the hypothalamic-pituitary area should be re-evaluated and followed up before a diagnosis of growth hormone deficiency is firmly established.
Although it is clear that the committee sought the views and opinions of a wide range of experts, we were struck throughout by the lack of published, peer-reviewed evidence to support one of its important conclusions: “On balance, we are convinced that experiments on animals have contributed greatly to scientific advances, both for human medicine and for animal health. Animal experimentation is a valuable research method which has proved itself over time” (page 22, para 4.8).7 We are not suggesting that the Lords did not seek out such evidence (it is clear from the transcripts published on the internet that on many occasions they asked witnesses to supply them with peer-reviewed references and reviews to support their claims about the efficacy of animal experiments).8 Rather, we wish to draw attention to the poverty and paucity of this evidence. Hardly any systematic reviews, meta-analyses, or retrospective, historical evaluations either support or refute the practice of using animals as models of human disease. The Lords’ assertion of the value of animal experimentation rests on the increase in effective human treatments that have arisen at the same time as the expansion of animal experimentation. This correlation does not mean that animals were necessary for the development of these treatments.

The move in medicine to become more evidence based needs to be replicated in research. If uncertainty persists about a particular paradigm or method—in this case the efficacy of using animals as models of human disease—evidence needs to be gathered so that claims about its efficacy can be supported or refuted. If no evidence supports the use of a particular method and only custom and practice sustain it, then that method should be discarded. Currently animal tests are used as the gold standard by which so called alternatives are judged, yet virtually no evidence supports the use of the animal tests themselves. In the few cases where systematic reviews of animal experiments have been conducted, serious doubts have been raised about the methods used.1 Evaluating the practice of using animals as models of human disease is fairly straightforward and practicable when established animal models of diseases exist. The models should be evaluated retrospectively, the key criterion being the productivity of the animal model in terms of producing treatments for human disease.

Pandora Pound research fellow
Shah Ebrahim professor in epidemiology of ageing
Department of Social Medicine, University of Bristol, Bristol BS8 2PR

New heart risk equations do not affect existing guidelines

Entroir—The paper by Nanchahal et al indicates that substantially fewer men and women without overt cardiovascular disease would be eligible for drug treatment if the standard 1991 Framingham risk equation for coronary heart disease risk that is used in the Joint British Cardiovascular Recommendations were to be replaced with the 2000 model.1 2

This would be an important finding for developers of cardiovascular guidelines and budget holders if the newer risk equation was substantially more accurate than the old one. This seems unlikely, however, because the new risk equation is based on a modest number of end points (224 for men and 108 for women), and the equation for men incorporates the same set of risk factors as the 1991 equation.

What guideline developers need is a comparison of the predictions of each risk equation with observed incident cardiovascular events, preferably over a range of thresholds (such as characteristics of receiver and operator). Published analyses of receiver-operator characteristics show that Framingham risk equations are only modestly accurate against observed events of coronary heart disease.3 4 We have also shown that the 1991 and 2000 risk equations have very similar discriminability for coronary heart disease events in a New Zealand cohort (unpublished data).

Fortunately for guideline developers, the interesting findings reported in this paper do not provide a good reason to abandon the 1991 Framingham risk equations in favour of the 2000 version. Cardiovascular guidelines will need to be revised when better risk equations become available.

Richard Milne associate professor
Divisions of Pharmacology and Community Health, University of Auckland, PO Box 60515 Titirangi, 1007 Auckland, New Zealand
richard.milne@hcu.ac.nz

Role of emotional capacity in consent should be clarified

Entroir—Case law does not clearly distinguish between intellectual and emotional capacity. Sensky discussed the dilemma of withdrawing treatment but missed an opportunity to highlight the importance of emotional capacity in decision making.5

What constitutes a mental disorder that impairs capacity, making someone unable to understand emotionally (as opposed to intellectually) the personal relevance of information and weigh it up? The judge’s perspective cited by Sensky is this: refusal by a mentally competent person to give consent is an absolute right even if the reason for this is irrational. But an irrational reason for refusal of treatment suggests that the ability to weigh evidence is impaired.

Value systems lie at the heart of mental disorders. For example, people may become depressed after having been made redundant if they believe that their only value as a person is determined by their productivity. That their depression is in keeping with their value system does not preclude doctors’ attempts to prevent suicide and help them to overcome the depression. Such is enshrined in mental health law, but it also influences the determination of capacity in common law. In a case heard by the Court of Appeal a needle phobia was considered enough to render an individual incapable of giving valid consent.6 In this case the patient probably held a value that the experience of being injected by needles was unbearable. This patient would not be considered as having a mental disorder by people encountering her; yet in the context of incurring surgery she became so severely anxious that she was considered incapable of consenting to the procedure.

In the case reported by Sensky the patient would similarly be considered not to have a mental disorder until faced with a situation that activated her value system. Her value—continuing life in this manner is worth less than death—has impaired her ability to weigh the objective (intellectual) evidence. She thereby expressed hopelessness of living a life that she would consider worthwhile. An alternative would be to work with her to create a value system that would enable her to consider her life worth while.

The role of emotionally held values and beliefs in influencing an individual’s capacity to give consent should be debated directly. This issue could helpfully be resolved in the drafting of the new Mental Health Act. Other patients who are currently deemed to have capacity (presumably intellectual) despite the presence of psychosis or overpowering affects related to adjustment deserve to have their right to life protected in keeping with the Human Rights Act 1998.

Steve Moorhead Consultant in general psychiatry and cognitive therapist
The George, Newcastle upon Tyne NE12 9PN

Doug Turkington senior lecturer in liaison psychiatry
Department of Psychiatry, University of Newcastle upon Tyne, Leazes Wing, Royal Victoria Infirmary, Newcastle upon Tyne NE14LP

2 Re MB (Medical Treatment) [1997] 2 FLR 426.

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Rapid responses
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1 Dobson, R. Lords support animal experiments but call for alternatives. BMJ 2002;325:238.