Letters

Prescribing antidepressants in general practice

What is an effective dose?

EDITOR—What should be the advised dosage of a tricyclic antidepressant for major depression? In his editorial Tony Kendrick quotes a recommended dose of a typical tricyclic antidepressant, amitriptyline, as 125 mg daily.[1] This dosage is at the higher end of the range of dosages quoted for all tricyclic antidepressants in the *British National Formulary*. If followed it would have major implications for the prescribing habits of general practitioners.

The recommended dosage for amitriptyline for major depression is quoted from a consensus statement published in the *BMJ* in 1992.[2] The source of this recommendation is a double blind placebo controlled trial of amitriptyline among depressed patients in general practice.[3] In this trial patients given the active drug received amitriptyline 75 mg daily by the end of the first week, amitriptyline 100 mg during the second week, and amitriptyline 125-175 mg, depending on improvement, during the remaining four weeks of the study. The median dose of amitriptyline given over the six week study period was 125 mg. It is the median tolerated dose. Figure 1 in the paper by Hollyman *et al* clearly shows a response to treatment in the first two weeks, when the lower dose of amitriptyline was given.[3]

Kendrick discusses the results of several investigations into the effectiveness of antidepressants at different dosages. Similar investigations have been used to develop the dose range of every prescribable antidepressant—for example, Kerihuel and Dreyfus reviewed 34 randomised clinical trials of lofepramine.[4] In the papers reviewed the concentration of lofepramine giving a clinical response ranged from 105 mg to 210 mg, which is equivalent to 75-150 mg amitriptyline.

The principle that more severe cases of major depression require higher doses of antidepressants is also valid. It also holds true for newer selective serotonin reuptake inhibitors. For example, a study of prescribing patterns across 23 381 prescriptions shows that a prescription for higher than the initial dose was required in 6.8% of patients taking fluoxetine, 21.1% of patients taking paroxetine, and 51% of patients taking sertraline.[5]

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1 Kendrick T. Prescribing antidepressants in general practice. *BMJ* 1996;313:829-30. (5 October.)
2 Paykel ES, Priest RG. Recognition and management of depression in general practice: consensus statement. *BMJ* 1992;305:1198-202.
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5 Donoghue J. Prescribing patterns of selective serotonin reuptake inhibitors in primary care: a naturalistic follow up study. *Journal of Serotonin Research* 1996;4:267-70.

More on what is an effective dose

EDITOR—I think that there is some misconception about the effective doses of tricyclic antidepressants in primary care. The consensus guidelines state that doses between 125 mg and 150 mg have been shown to be effective and doses of 75 mg have not.[1] This statement is widely interpreted to mean that 125 mg or more is the recommended dose and that the minimum effective dose is 125 mg. Much has been made of general practitioners failure to prescribe at recommended doses.[2 3] What is the evidence that this interpretation of the guidelines is justified?

In none of the three studies cited in the consensus guidelines was there any attempt at dose titration. Amitriptyline was compared with either placebo or substantially lower doses. In one of these studies, a double blind placebo controlled trial in general practice, the final dose of amitriptyline achieved in the treatment group was a median of 125 mg and a mean of 119 mg.[4] In other words, at least half of the treatment group were receiving doses of less than those recommended. Doses based on how many tablets were returned were lower still: 113 mg and 111 mg at four and six weeks respectively. The same study data reported elsewhere show that significant differences between the study groups were present after only two weeks, when treated patients were taking only 100 mg amitriptyline. Indeed, the authors concluded that their study had not provided evidence about the optimum dose of amitriptyline.[4]

In conclusion, I suggest that it is unjustified to assume that doses of tricyclic antidepressants below 125 mg of amitriptyline or equivalent are not effective in primary care. I would echo the sentiments of Tony Kendrick, who questions whether general practitioners are really wrong to prescribe at lower doses.[5] I welcome the news of further research into effectiveness of low dose tricyclic antidepressants in primary care.[5]

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1 Paykel ES, Priest RG. Recognition and management of depression in general practice: consensus statement. *BMJ* 1992;305:1198-202.
2 MacDonald TM, McMahon AD, Fenton GW, McDevitt DG. Antidepressant drug use in primary care: a record linkage study in Tayside, Scotland. *BMJ* 1996;313:860-1. (5 October.)
3 Donoghue J, Tylee A, Wildgust H. Cross sectional database analysis of antidepressant prescribing in general practice in the United Kingdom, 1993-5. *BMJ* 1996; 313:861-2.
4 Hollyman JA, Freeling P, Paykel ES, Bhat A, Sedgwick P. Double-blind placebo-controlled trial of amitriptyline among depressed patients in general practice. *J R Coll Gen Pract* 1988;38:393-7.
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Systematic review of all pertinent trials is required to establish guidelines

EDITOR—"Watchful waiting for minor depression, full dose treatment for major depression" is a fairly catchy subtitle to Tony Kendrick's editorial,[1] but it is questionable how far it is based on evidence.

Depression is continuously, not dichotomously distributed in the population, with no point of rarity between major depression and other forms. Intrinsically, non-major depression is likely to respond to antidepressants to some extent if major depression

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does. Such a view gains no support from evidence about the treatment of other continuously distributed health problems such as pain or hypertension, and the trials quoted have conflicting results.

If patients with mild depression benefit from antidepressants they may indeed be responding to a "placebo with side effects," but other options desired by patients such as counselling seem to be ineffective and are likely to be more expensive. If general practitioners believe that they have to do something they know that the hypnotic and anxiolytic properties of antidepressants are often helpful in the mixed neurotic states seen in primary care, irrespective of any direct effect on mood.

The various guidelines advocating high doses for major depression in general practice are based on evidence,[2] but, again, general practitioners know that starting with a low dose is the only way that many patients will take tricyclic antidepressants, and many patients will have recovered or stopped treatment long before their general practitioner has been able to increase the dose anywhere near 150 mg or 225 mg daily. Selective serotonin reuptake inhibitors, another option, are more expensive, lack dose flexibility, and have not been shown to be superior in practice.

Only a systematic review of all pertinent trials from all countries, published and unpublished, can answer these questions. This is one of the main objectives of the recently formed Cochrane Collaborative Review Group for Depression and Neurosis, which welcomes any help towards this aim. Premature guideline development should be avoided.

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2 School of Public Health, University of Leeds. *The treatment of depression in primary care.* Leeds: University of Leeds, 1993. (Effective Healthcare Bulletin No 5.)

Low dose tricyclic antidepressants are effective in treating major depression

EDITOR—T M McDonald and colleagues commented that tricyclic antidepressants used in low doses were unlikely to be of benefit in treating major depression.[1] I disagree on the basis of work that I did with colleagues at Hammersmith Hospital.[2] We studied the effect of low dose lofepramine (a tricyclic antidepressant) in 46 depressed elderly people and found that one third of the recommended dose was sufficient to improve major depression. We also found that placebo was as effective in people with mild depression ($P = 0.04$, 95% confidence interval 0.4 to 7.9). A similar study of another tricyclic antidepressant was conducted in America with low dose doxepin in 24 elderly patients.[3] Low doses of doxepin (10-20 mg) were also effective in significantly reducing depressive symptoms.

I believe that the biological half lives of tricyclic antidepressants do not correlate well with the physiological half lives. This might explain why low doses of these agents are effective. Currently, therapeutic ranges have been developed for amitriptyline and imipramine. In my experience the clinical effects of tricyclic antidepressants do not always parallel serum concentrations. MacDonald and colleagues found that 72% of subjects were given subtherapeutic dosages,[1] but whether this group improved with the lowered dosages is not known.

However, I concur that low doses of tricyclic antidepressants are safe as this was also observed in our study of low dose lofepramine.[2] This is especially important in ambulatory elderly patients as they may not be able to tolerate higher dosages.[4]

Tony Kendrick argues in his editorial that watchful waiting is sufficient for minor depression.[5] We also found placebo to be as effective for people with minor depression. An attentive ear is often more effective than drugs. However, the statement that a full dose is warranted for major depression is challenged by the results of our study.[2]

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Prescription does not mean that major depression is being treated

EDITOR—The study of John Donoghue and colleagues raises the question why general practitioners continue to prescribe low dose tricyclic antidepressants.[1]

They cite my study of people taking long term antidepressants, which showed that 62% of the 177 responders to a postal survey reported moderate to severe depressive symptoms at follow up; prescription of low dose tricyclic antidepressants was widespread even among this group.[2] Patients frequently consulted their general practitioner with psychological complaints, which tends to refute the notion that low doses continue to be prescribed because of a belief in their efficacy. Furthermore, the suggestion that general practitioners avoid raising the dose because of side effects cannot be the whole explanation because the logical solution would be simply to prescribe an alternative antidepressant.

Another explanation is that the general practitioner may have revised his or her original diagnosis. The diagnostic process in primary care takes place over several consultations with the accumulation of information which helps to gain a more complete formulation of the patient's problems. Factors becoming apparent later, such as marital and financial difficulties, social adversity, life events, and even a poor response to drug treatment, could influence the general practitioner to revise the original diagnosis to one of dysthymia, adjustment disorder, or social problems. This may induce a sense of therapeutic nihilism, in which symptoms are perceived as being understandable and appropriate in the given psychosocial circumstances and therefore as unresponsive to antidepressants.

Of course some patients initially diagnosed as being depressed by their general practitioner may not have met standard criteria for major depression in the first place and therefore antidepressants would not be indicated. The problem, however, lies with patients who do meet criteria for major depression, which is treatable. Helping general practitioners to identify these patients and recognise that antidepressants are effective even when the depression is understandable has rightly been one of the aims of the Defeat Depression Campaign.

Low dose tricyclic antidepressants probably continue to be prescribed because depression in primary care comprises a heterogeneous group of major and minor depression, adjustment and anxiety disorders, and dysthymia, in which low dose tricyclic antidepressants are prescribed for insomnia and anxiety while clarification of the diagnosis is awaited. Although major depression may be included in a general practitioner's differential diagnosis, it may be discarded later. The important thing is that patients with a diagnosis of depression from a general practitioner (without a concurrent standardised psychiatric assessment) may be flawed because the concept of depression in primary care is broad and the use of antidepressants in itself does not mean that major depression is being treated.

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2 Ali IM. Depression in primary care: a study of long term antidepressant users [MSc thesis]. Cardiff: University of Wales, 1994.

Tricyclic antidepressants are also used for relief of chronic pain

EDITOR—I consider the paper by T M MacDonald and colleagues and the accompanying editorial by Tony Kendrick to be misleading because they criticise general practitioners for prescribing low dose tricyclic antidepressants, claiming that there will be no therapeutic effect.[1 2] This may be the case in the treatment of clinical depression, but it must be emphasised that many patients suffering chronic pain and mild depression are also prescribed these drugs for their therapeutic effect on neuropathic pain and their evident benefit on night time sedation.

Any therapeutic action on mood is a bonus but is not the primary goal of treatment. Most of this group of patients will titrate the dose of drug against side effects (dry mouth and daytime sedation) and pain modification. Many patients will use these drugs long term after the drugs have been correctly titrated to minimise the above side effects and it has been explained to them that they are not addictive. Also, the newer (cleaner) antidepressant drugs seem to be less efficacious than the tricyclic antidepressants in this respect.

Most pain management clinics prescribe low dose tricyclic antidepressants on a regular basis, and newly referred patients are now commonly already taking these drugs as part of the management of their chronic pain. I think that it would be a retrograde step if general practitioners took away the wrong message from the above articles[1 2] and increased the drug dose for the wrong reasons or mistakenly switched to newer drugs.

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1 MacDonald TM, McMahon AD, Reid IC, Fenton GW, McDevitt DG. Antidepressant drug use in primary care: a record linkage study in Tayside, Scotland. *BMJ* 1996; 313:860-1. (5 October.)
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Prescribing rates vary widely between practices

EDITOR—The information on general practitioners' prescribing is interesting, but John Donoghue and colleagues give no information on the variation in prescribing between practices.[1]

Grampian region had a population of 532 500 in 1994, with 340 general practitioners in 89 practices. Analysis of the prescribing rates per 100 patients by each practice for psychiatric drugs varied from 2.63 to 15.38 for hypnotics and tranquillisers and from 3.50 to 14.84 for antidepressants, whereas prescribing of antipsychotic drugs ranged from 0.36 to 3.81. These ranges were based on the number of drugs prescribed, and they therefore overestimate the numbers of patients treated when several drugs are given to one patient or when frequent prescriptions are given (perhaps because of perceived risk of suicide). Prescribing rates bore no correlation to the proportion of patients in the practice who lived in deprived areas (as measured by Carstairs deprivation category for postcode) or to the rate of referrals to inpatient or outpatient psychiatry. Only three of the 12 practices with the highest prescribing rates for antidepressants were in the city of Aberdeen: the others were in small rural and fishing villages.

More research is needed on reasons for such a wide variation in the prescribing practice of general practitioners.

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Prescribing is short term and follow up poor

EDITOR—In their study of antidepressant prescribing in general practice John Donoghue and colleagues found that selective serotonin reuptake inhibitors were more likely than older tricyclic antidepressants to be prescribed at recommended doses.[1] However, effective treatment of major depression requires antidepressants to be continued for at least four to six months after clinical improvement because treatment for an inadequate amount of time is more likely to result in relapse.[2]

We performed a retrospective review of antidepressant prescribing in two fully computerised, paperless, general practices in Ewell, Surrey. The combined list size is 11 400 patients. All consultations and prescriptions are entered directly on to a record linked practice computer by each doctor. We performed a computer search of all patients who had a diagnosis of depression and who were prescribed an antidepressant between 1 January 1995 and 31 June 1995. Patients prescribed an antidepressant in the previous year were excluded as we wished to examine newly treated patients. We reviewed the notes manually one year after the initial prescription to determine the duration of treatment, the number of follow up examinations by the general practitioner, the rate of relapse, and the reason for stopping treatment.

We identified 78 patients (annual incidence 1.4%), of whom 47 (60%) were prescribed a serotonin reuptake inhibitor and 31 (40%) a tricyclic antidepressant (including four patients who were given lofepramine). The average age and sex distribution of the patients was similar for both serotonin inhibitors (45.3 years, 37 women (79%)) and tricyclic antidepressants (48.6 years, 25 women (80%)). The median length of treatment with a serotonin reuptake inhibitor was only four weeks compared with six weeks for tricyclic antidepressants. Fourteen patients (30%) initially prescribed a serotonin inhibitor had a relapse after stopping treatment compared with five patients (16%) initially prescribed a tricyclic antidepressant. The average number of general practitioner follow up examinations was 2.0 for serotonin inhibitors and 3.4 for tricyclic antidepressants. In most cases (44 patients (56%)) no reason for stopping treatment was recorded, and most patients had only one follow up consultation or none at all (19 (24%) and 24 (31%) patients, respectively).

We found that both selective serotonin inhibitors and tricyclic antidepressants were given for short durations for the treatment of depression and that follow up by general practitioners was poor. In this comparatively small study we were unable to control for confounding by severity or prescribing history. However, we agree with Donoghue and colleagues that research is urgently required to determine the clinical and economic costs of such prescribing.

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2 Paykel ES, Priest RG. Recognition and management of depression in general practice: consensus statement. *BMJ* 1992;305:1198-202.

Costs should have been considered

EDITOR—The survey of John Donoghue and colleagues shows a 133% rise in prescriptions for specific serotonin reuptake inhibitors between 1993 and 1995.[1] If generalisable this represents a major shift in prescribing, the economic implications of which are ignored in the accompanying editorial by Tony Kendrick,[2] who is unable to clinically justify this trend.

The largest absolute difference in total discontinuation rates between specific serotonin reuptake inhibitors and tricyclic antidepressants in a meta-analysis is 2.8%[3] (rather than 5-10%, as quoted by Kendrick[2]). The number of patients who need to be treated with specific serotonin reuptake inhibitors instead of tricyclic antidepressants to prevent one discontinuation is 38. The clinical importance of a small difference in discontinuation rates is hard to judge and has been little studied.

If, however, small increases in compliance are regarded as sufficiently important to achieve, then the cost effectiveness of alternative strategies should be evaluated,[4] rather than presume that this is best addressed by a major shift in prescribing.

For example, the finding of Robert G Priest and colleagues that most of the public believes antidepressants to be addictive[5] is a possible contributory factor to overall treatment discontinuation rates of over 30%, irrespective of what is prescribed. A more cost effective strategy than such a change in prescribing might be to give better information to patients starting antidepressant treatment in order to allay their fears.

It is important that well intentioned national initiatives (such as the Defeat Depression Campaign) designed to extend quality health care to a wider number of people are not used opportunistically by commercial interests to promote a particular product. We were surprised that the fact that Hiram Wildgust, one of Donoghue's colleagues,[1] is an employee of Lilly Industries was not acknowledged as a conflict of interest.

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Shared care of people with major mental illness

EDITOR—Tony Kendrick and Tom Burns (a general practitioner and a psychiatrist) argue that family doctors should take back the sole continuing care of stable and compliant patients with psychotic disorders.[1] However, we stand by our assertion that it is the job of multidisciplinary community teams to oversee the care of almost everybody with a major mental disorder,[2] although always in collaboration with general practitioners. This debate is at least a healthy sign amid concerns about the failure of community care and in the light of recent guidance from the General Medical Services Committee which could greatly limit general practitioners' role in the management of major mental illness.[3]

No matter who takes primary responsibility, the monitoring of seriously mentally ill patients should lead to adequate recognition of their needs and the appropriate responses. Several studies, including our own, indicate that routine psychiatric services often fail in this task.[2 4] Our subjects who had returned to the sole care of their general practitioner also had important unidentified needs, although Kendrick and Burns are right to criticise our failure to study the important group of psychotic patients who have never been in touch with specialists.[1]

Continuing care of patients with major mental diseases is dominated by the fact that many do not seek help when their condition deteriorates. This is why we now insist that even patients whose condition is stable require systematic review, usually in their own homes. Our work in Lanarkshire and an ongoing investigation by one of us (HW) in the Scottish borders suggests that a rolling survey of all patients with identified psychotic disorders can be undertaken without prohibitive additional costs. Several standardised schedules have been developed which could guide this clinical process.[5] Further research is required to show whether this routine needs assessment will help specialists and general practitioners to prioritise mental health care according to need rather than demand. This will probably lead to increased input to the least vocal and most vulnerable psychiatric patients—that is, those with schizophrenia, manic depressive illness, and other brain disorders. This will inevitably divert services from patients with milder acute conditions, but it is for each area to establish comprehensive services within available resources. Potential shortfalls in healthcare provision will not be avoided by leaving general practitioners to provide all community care once patients recover from the acute phase of major psychotic illness.

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People may become psychologically dependent on antidepressants

EDITOR—Robert G Priest and colleagues advocate educating patients that discontinuing antidepressant treatment will not be a problem but remarkably do not cite any evidence to support their recommendation.[1] They also complain that many lay people regard antidepressants as addictive. They suggest that people may be extrapolating from what they have heard about benzodiazopines. This may be, but it is also common sense to believe that discontinuing taking a drug that is thought to improve mood may be difficult. I think that the general public understands this issue better than the Royal Colleges of Psychiatrists and General Practitioners, which are responsible for the Defeat Depression Campaign.

Of course what Priest and colleagues mean is that there is little evidence of physical dependence caused by antidepressants, but this is not what they say. There are, however, case reports of a withdrawal syndrome.[2] Clinical experience is that it can be difficult to withdraw treatment with antidepressants for various reasons. The general public might reasonably expect psychiatrists specialising in disorders of the mind to recognise psychological dependence, base their advice on clinical experience, and use their common sense.

Randomised controlled trials of discontinuation of antidepressant treatment have a relapse rate varying from 92%[3] to 36%[4] in the placebo group. Relapse rate is significantly reduced by continuing antidepressant treatment. Some patients therefore do maintain their therapeutic gains when antidepressants are withdrawn, but the relapse rate is not insubstantial and seems to support the general public's commonsense view rather than the Defeat Depression Campaign's purist scientific statement. Perhaps the public needs to be suspicious of the motives of a campaign that encourages them to seek medical treatment and also tries to help doctors recognise depression. Patronising misinformation is not constructive.

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1 Priest RG, Vize C, Roberts A, Roberts M, Tylee A. Lay people's attitudes to treatment of depression: results of opinion poll for Defeat Depression Campaign just before its launch. *BMJ* 1996;313:858-9. (5 October.)
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Afghanistan: a biased report

EDITOR—Robert Ivker's news story about medical supplies to Afghanistan publicised the good work of the International Red Cross but was factually incorrect and made inadequate reference to the World Health Organisation.[1]

He writes: "In the past four years nearly 50 000 people have died in the conflict between the Taliban authority and a coalition of ethnic minority groups that is fighting against it." Taliban started its comparatively peaceful takeover of large areas of Afghanistan early in 1995, putting an end to the fighting and bringing safety and peace wherever it came; the opposing coalition formed only late in 1996. Statistics for deaths from fighting among the Mujahedin followers before the time of the Taliban are not available.

We at the WHO have staff constantly in nine suboffices throughout the country, and we know of no case of men being executed because they do not have a beard. Women doctors and nurses at all health facilities in Kabul are working normally. The WHO is not only "primarily concerned with basic nutritional support and the immunisation of children." [1] In the past few years staff from the Ministry of Public Health were not able to visit all parts of the country, but the WHO has always been welcomed everywhere because of its strict neutrality; in practice it has acted on behalf of the ministry. As an impartial United Nations agency, we as the WHO provide some medical supplies, tools, and instruments to nearly all hospitals. We have national and international experts; conduct short term training courses, seminars, and workshops for doctors, nurses, health workers, and traditional birth attendants; and sponsor fellowships for Afghan doctors and paramedics all over the world.

At Qandahar in the south, in remote Faizabad in the north east, and in Jalalabad and Ghazni we have reinstated water supply networks. Together with Unicef and non-government organisations we have vaccinated millions of mothers and children. Two batches of medical students from Kabul and Jalalabad have been able to graduate with

financial help from the WHO. We have also funded income generating projects such as soap making and dairy farming to create job opportunities, contributed to the demobilisation of the militia and to peace, and improved the quality of life of the people.

Incidently, we were not hit by a rocket in Kabul but were bombed by the anti-Taliban coalition forces during a three day workshop sponsored by the WHO for health representatives from 22 provinces.

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1 Ivker R. Medical supplies airlifted to Afghanistan. *BMJ* 1997;314:540. (22 February.)

Ratio of abdominal sagittal diameter to height is strong indicator of coronary risk

EDITOR—Margaret Ashwell and colleagues state that the ratio of waist circumference to height is the best simple anthropometric predictor of intra-abdominal fat ($r=0.83$) in men and women.[1] Kvist *et al* measured the abdominal sagittal diameter (the distance between the examination table and the highest point of the abdomen in the supine position) and showed a high correlation ($r=0.92$) with intra-abdominal fat as measured by computed tomography in men.[2] This measurement was therefore used in the collection of baseline data for the French/Swedish Renault/Volvo heart study, a prospective 10 year study of 1000 male employees aged 45-50 at each of the two companies that was initiated in 1993 (A Simon *et al*, unpublished findings). Measurement of the abdominal sagittal diameter was limited to the Swedish (Volvo) population.

It is our experience from research into obesity that the metabolic syndrome through the neuroendocrine axis involving the formation of intra-abdominal fat may have a central role in the atherosclerotic process.[3] We therefore hypothesised that the association between the best predictive indicators of pathological deposition of abdominal fat and other ischaemic risk factors (the Framingham risk index[4]) would indicate the most clinically relevant measurement. This risk index is composed of the following factors: age, smoking habits, diabetes mellitus, systolic blood pressure, the ratio of total cholesterol to high density lipoprotein concentration, and left ventricular hypertrophy.

Table 1 shows that the abdominal sagittal diameter and the abdominal sagittal diameter divided by height had similar correlations with the Framingham index. The correlation to waist circumference divided by height was slightly lower. These correlations disappeared on adjustment for the ratio of total cholesterol to high density lipoprotein cholesterol concentration. The partial correlations in table 1 were close to zero.

We conclude that the sagittal diameter divided by height may be a useful indicator of coronary risk. The cholesterol ratio, however, is a possible confounder and should be calculated during the follow up study.

Table 1 Correlation coefficients (r) between Framingham risk index (unadjusted and adjusted for ratio of cholesterol to high density lipoprotein concentrations) and certain anthropometric variables

	Framingham risk index	
	Unadjusted	Adjusted
Body mass index (weight (kg)/(height (cm))^2 (n=1798)	0.185*	0.025
Waist:hip circumference (n=1829)	0.165*	0.024
Waist circumference:height (n=1833)	0.211*	0.043
ASD (n=866)	0.282*	0.042
ASD:height (n=866)	0.288*	0.055

ASD=Abdominal sagittal diameter.
*P<0.001.

We thank Bo Eriksson, associate professor at the Nordic School of Public Health, Gothenburg, Sweden, for helping us with this letter.

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Baby milk companies accused of breaching code

Code is often disregarded in the United Kingdom

EDITOR—Violations of the World Health Organisation's code of marketing of breast milk substitutes by baby milk manufacturers have recently been reported from Thailand, Bangladesh, South Africa, and Poland.[1] It is worth noting that the code is also frequently disregarded within the United Kingdom.

Posters, parent information leaflets, and calendars marked with the trade names of baby milk companies are commonplace within health service premises; gifts such as pens and notebooks, with baby milk manufacturers' trade names and logos, are frequently presented to health workers. These provide tacit endorsement of the advertisers' products within the health service.

The code requires that manufacturers' product information, including labels, should explain the benefits of breast feeding and the costs and hazards associated with artificial feeding. No more than a token attempt is made by any of the manufacturers to give this information to the public.

There is now clear evidence that artificial feeding of infants is associated with substantial morbidity, some mortality, and much increased health service costs, even in the affluent countries of the West[2 3] None the less, the present government has refused to legislate to make the advertising of baby milks illegal, apparently after pressure from within the baby milk industry.[4]

The Indian Medical Association and the Pakistan Paediatric Association have managed to refuse financial support from the baby milk industry.[5] It is a pity that the Royal College of Paediatrics and Child Health cannot provide a similar lead within the United Kingdom.[1]

Here, only 29% of new mothers leave hospital breast feeding. As part of a joint breast feeding initiative our local hospital and community trusts have launched a breast feeding policy which includes the avoidance of all forms of advertising of breast milk substitutes within the premises of the trusts. A breast feeding training programme has also been initiated for all relevant staff, which includes information on the implementation of the WHO code.

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Why scientists should not become divorced from baby milk companies

EDITOR—Jacqui Wise discusses a report criticising baby milk manufacturers for violating the international code on marketing breast milk substitutes[1]—a code developed to protect and promote breast feeding.[2] Failure to breast feed has been linked to detrimental outcomes in babies[3] and mothers[4] and, in developing countries, to substantial infant mortality. Code violations are rightly offensive to health professionals and the public.

The backlash against baby milk companies has, however, changed the climate for researchers collaborating with industry to advance infant nutritional care. Such scientists may be personally criticised, even though the international code recognises a place for baby milks. Some hospitals—for example, so called baby friendly hospitals—may inhibit clinically indicated research with formula milks. The International Board of Lactation Consultant Examiners recently moved to withhold continuing education credits from people attending lectures by speakers who have a relationship with manufacturers or retailers of products for the artificial feeding of infants. This could exclude lecturers who work with industry on materials to promote breast feeding.

To foster a balanced view in this sensitive area I suggest four reasons why clinical

scientists should not become divorced from the baby milk industry.

Firstly, raw cows' milk is not recommended before 12 months. As most Western mothers stop breast feeding long before then, baby milk is generally the dominant food for Western infants even when they have been breast fed initially. Therefore its safety and efficacy requires careful industrial and scientific collaborative research.

Secondly, products made by baby milk manufacturers may be clinically indicated when lactation fails; breast feeding is inadvisable as in maternal HIV infection, certain drug treatment, and rare inborn errors of metabolism in the infant—or breast milk alone is nutritionally inadequate, as in preterm infants. Research is complex and requires close liaison between industry and science.

Thirdly, research on formula milks is fast moving. Manufacturers cannot and should not operate alone—they must be responsive to clinical scientists.

Finally, the outcome of breast fed infants guides the design of baby milks. As breast and formula feeding cannot be randomly assigned, how a component of breast milk affects outcome is most robustly tested in non-breast fed infants by randomised comparison of formulas; one containing the relevant component. Collaboration between industry and science is essential.

Recent evidence suggests that infant nutrition may have important programming effects on later health.[5] Much of the world's scientific and technological expertise that underpins this rapidly developing field lies in industry. Much key research—including that on breast feeding—results from industrial grants to responsible clinical scientists, who should not be made uncomfortable for necessary contributions to infant care. Nevertheless, researchers should not miss opportunities to influence baby milk manufacturers to adhere to their important marketing code.

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Strategies to reduce dosing errors

EDITOR—Recently, while waiting for an injection, I heard an auxiliary member of staff talking to herself about a dose, from which I realised that she was confused about the difference between 0.1 and 0.01. Also recently, a baby died after having been given a dose of morphine that was wrong by a factor of 100. We cannot any longer assume elementary understanding of decimal numbers by people who are allowed to wield syringes. What should we do? I suggest that dose units for drugs should be rescaled so that they appear as integers. Most people know the difference between 1, 10, and 100. (However, don't make the units too small because many people confuse 10 000 and 100 000.) When doses must be adjusted to per kilogram body weight a simple multiplication table of integer doses times integer weights could be provided. These steps should greatly reduce the chance of such errors in future.

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Evidence to be given to the public must be presented accurately and fairly

EDITOR—Promoting informed choice is commendable provided the evidence is presented accurately and fairly. The case study by S Oliver and colleagues exemplifies how evidence may be biased.[1] The authors studied reactions to leaflets prepared by the Midwives' Information and Resource Centre and the NHS Centre for Reviews and Dissemination, organisations of which four of the authors are employees. We are not told whether these same four investigators were also authors of the leaflets, but the question of conflict of interest must arise.

The leaflet "Informed choice for women" states: "one in 200 babies who were aborted as a result of what a scan showed were in fact normal or had only minor things wrong," and Oliver *et al* quote this figure four times. As an author of the paper from which this statistic is derived,[2] I suggest that the evidence should be interpreted in its true context, otherwise misinformation will be the result.

We studied 2261 pregnancies with a fetal anomaly diagnosed by ultrasonography in Yorkshire during 1989-91. Altogether 369 of these pregnancies were terminated, and in two the anomaly proved less severe than had been predicted on ultrasonography. As Paul Chamberlain and P A Boyd say in their commentary on Oliver and colleagues' paper,[1] one of these fetuses had a gastroschisis (which still carries a considerable perinatal mortality); the other fetus had enlarged hyperreflective kidneys, and, despite normal histological findings, the outlook for renal function in such a fetus remains open to speculation.[3] This 99.5% specificity for the diagnosis of fetal anomalies warranting termination was achieved from a region-wide hospital mix with ultrasonography equipment of varying sophistication and by ultrasonographers with differing levels of experience.

The diagnosis of fetal anomaly has continued to advance since 1991, with developments in ultrasonographic technology and with improved training programmes for sonographers, radiologists, and obstetricians. The specificity of ultrasonographic diagnosis of fetal anomalies is confidently thought to have increased beyond 99.5% already.

It is a pitfall of evidence based medicine that evidence may be misinterpreted or may be presented in a biased manner. There are few diagnostic tests in medicine that perform to such high accuracy, and it would have been appropriate to inform the public in language that conveys this good news rather than in an alarmist manner. I share the concerns of the ultrasound professionals quoted by Oliver and colleagues that these leaflets may do more harm than good, and I suggest that the relevant health professionals are consulted when such leaflets are next prepared.

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Skin scraping is a useful investigation in meningococcal disease

EDITOR—F Andrew I Riordan and colleagues report the diagnosis and treatment of early meningococcal disease in children in Liverpool.[1] It is worth emphasising another aspect of these rashes—namely, that meningococci that are present in the rash may be identified by taking skin scrapings. The lesion is scraped with the point of a sterile needle until blood just starts to appear. The blood is then blotted on to microscope slides, allowed to dry, and examined for Gram negative diplococci. This investigation has the advantage of providing results rapidly, and, unlike examination of blood and cerebrospinal fluid, it is not greatly affected by prior antibiotic treatment.[2] We found that skin scrapings gave a positive result in 80% (24/30) of meningococcal rashes, which is a higher success rate than that reported for aspiration or biopsy of lesions[3]; in 27% (14/52) of cases the positive results obtained with skin scraping were the only positive results obtained with the three investigations of skin scraping, blood culture, and examination of cerebrospinal fluid.

Our house officers now scrape both typical and atypical rashes, and because immediate action is required and the results are obtained rapidly there is a heightened awareness of the need to suspect meningococcal infection even in the absence of classical signs. House officers who have been given their first positive results of skin scraping are eager to add more to their score.

We note that *Neisseria meningitidis* was isolated in only 62% (78/126) of the cases in Liverpool. This is similar to a report of laboratory confirmation in 54% (61/113) of cases of meningococcal infection in Cork.[4] Our laboratory confirmation rate is 83% (53/64), which is significantly higher than that in Cork ($\chi^2 = 14.81$, df = 1, P = 0.0001) and Liverpool ($\chi^2 = 14,81$, df = 1, P = 0.003). When the cases confirmed by skin scraping alone were excluded, however, our laboratory confirmation rate fell to 61% (39/64), which is not significantly different from the results in either Liverpool or Cork. Thus skin scraping makes all the difference.

We regard this procedure as a simple and highly rewarding investigation that should be carried out on rashes when meningococcal infection is suspected. It is included in the investigation list provided in the recent review of the control of meningococcal diseases.

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Mental health should be measured by same instrument in different ethnic populations

EDITOR—Greta Rait and colleagues are wrong to suggest that recent work on elderly people from ethnic groups has ignored mental health problems.[1] My own work among Gujaratis in north London showed high levels of satisfaction with life and similar levels of depressed mood to those in a comparison white British population.[2] More recent work among Bengalis and Somalis from the east end of London has also focused on mental health.[3] In comparison with Gujaratis, Bengalis and Somalis were found to have extremely high levels of depressed mood, with 77% and 24% respectively scoring in the depressed range on a standardised symptoms of depression and anxiety scale. Correlations between satisfaction with life, depressed mood, and physical ability showed good agreement and suggest that these simple mood scales have some value in all populations—which is not surprising, because there are limits to the ways in which symptoms of depression and anxiety can be experienced. Whether better indicators of depressive illness would be obtained by the methods outlined by Rait and colleagues is debatable. Certainly, indicators of mood that enable white British and other ethnic groups to be compared will not be feasible if specific instruments for different ethnic groups are developed.

I agree with Rait and colleagues that methods of assessing cognitive function depend greatly on literacy and educational attainment and thus seem to be culturally biased. Instruments have, however, been produced that overcome this limitation.[4]

Unravelling the triple jeopardy of aging—old age, discrimination, and limited access to care—among ethnic minority groups will require transdisciplinary investigation with a broad focus on physical, mental, social, and economic determinants of perceptions of health and the occurrence of disease.

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Opinions given by medical experts in court are honest and objective

EDITOR—By suggesting that medical experts eschew objectivity in favour of fat fees Liam Farrell displays his ignorance of the way that the legal system operates in both the civil and criminal courts.[1] The adversarial nature of these proceedings means that it is essential that both sides in a dispute have the opportunity to test each other's evidence. It is to this end that the medical expert points out discrepancies in the arguments of the opposing expert and offers alternative explanations when these exist. In my experience, this task is performed with honesty and objectivity. Perhaps Farrell is not aware that experts frequently agree with their opposite number when preparing their reports, the medical evidence effectively being agreed before court.

An adversarial system may not be the best way of adducing medical evidence, but, as things stand, it is essential for the sake of justice and equity that doctors are able and willing to perform the role of medical experts to the courts. Farrell's suggestion that these doctors are medical whores is untrue and disrespectful and does a disservice to the integrity of the profession as a whole.

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1 Farrell L. A professional opinion? *BMJ* 1996;313:1337. (23 November.)

Making the most of self citation

EDITOR—Nick Craddock and coauthors' exposition of Selfcite 2.0 shows the limitations of self citation.[1] Failure to carry out an adequate literature search has resulted in them failing to uncover my own best selling citation software Egocite.[2] This has already reached version 6.0 and comes with an interactive CDROM to allow a weekly updating of your curriculum vitae with the citation number automatically calculated by downloading from the Internet[3]—a valuable feature not available on their own software, and one that no true academic can be without.

There are two other flaws in their argument. They forgot to mention their Spread feature (also available on Egocite), which spreads a one page paper over two pages so it looks like a bigger paper when cited later,[4] and the immediacy index (number of citations within the same year) of their papers is not maximised. By not delaying returning their *BMJ* proofs for the appropriate time as calculated by the add on software Fudgecite 1.0, which we can also supply,[5] they have ended up with a publication in the last issue of the year with virtually zero chance of adding to the immediacy index. How much better to have delayed those proofs for another week so that the paper would have been published in the January or February issues to allow maximum immediacy and citation throughout a whole year.

An authors' reply to this letter is of course necessary so Egocite has automatically added a controversial edge. This will ensure further citations and will automatically send a letter to the corresponding academic in six months inviting him to coauthor a book in which our additional publishing software will automatically insert copious selected references from both academics, ensuring further citation.

Our next software upgrade is ready for 1997 and includes Salami-cite—a novel feature allowing the same paper with small alterations and limited additional data to constitute "different" papers that can be sent to several journals with the minimum of effort.

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