I have knowledge of 50 deaths (this includes unpublished cases from personal communications). The problem is sporadic and rare; because the consequences are so devastating for the patient, however, the medical and nursing staff managing patients with central venous catheters must prevent air gaining access to the venous circulation.^{1,5}

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EDITOR.—The head down position during removal of a central venous catheter, which Peter Mennim and colleagues recommend, is not without danger.¹ In elderly people and those with a full abdomen the pulmonary functional residual capacity is reduced, and the head down position may worsen this, leading to dangerous hypoxia. One of our patients experienced a cardiac arrest in this situation. The jack knife position (legs up, upper trunk horizontal) increases central venous pressure without this danger.

Removal of a central venous catheter may also be associated with other dangers. If the catheter has been introduced inadvertently through the pleura without having caused a pneumothorax life threatening bleeding into the pleura may occur after the catheter is removed.²

We agree that a central venous line should not be introduced or removed without careful monitoring or by inexperienced staff. Experience in using the catheter is gained after about 40 cases, when the personal learning curve flattens. We suppose that the same time is needed to gain experience in removal. It takes two to four months and liberal indications in a busy central hospital for an anaesthetist to achieve this.³ If the training is monitored and supervised⁴ the complication rate is fairly low.⁵

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Detecting severe silent mitral regurgitation

EDITOR.—John Efthimiou and colleagues suggest that transoesophageal echocardiography greatly facilitates the diagnosis of mitral regurgitation due

to rupture of a papillary muscle.¹ We disagree with two important points and believe that the authors' conclusions should be modified.

Firstly, the authors state that two dimensional transthoracic echocardiography showed no evidence of mitral regurgitation. Mitral regurgitation cannot be diagnosed from two dimensional ultrasound (echocardiographic) images, whether transthoracic or transoesophageal, since these images merely visualise the mitral valve apparatus and its movement. Secondly, although the mitral regurgitation was clinically silent, we suspect that the regurgitant jet would readily have been detected by transthoracic Doppler studies with or without the addition of colour flow. It seems unlikely that the transoesophageal mode was necessary to detect mitral regurgitation.

Rupture of a papillary muscle is an important though rare complication of myocardial infarction, but careful transthoracic Doppler investigation usually establishes the diagnosis.

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- 1 Efthimiou J, Pitcher M, Ormerod O, Harper F, Westaby S, Grahame-Smith D. Severe "silent" mitral regurgitation after myocardial infarction: a clinical conundrum. *BMJ* 1992;305:105-6. (11 July.)

AUTHORS' REPLY.—Besides two dimensional transthoracic echocardiography, which was performed on two occasions by experienced cardiologists, our patient underwent Doppler and colour flow studies on each occasion; neither of these procedures detected the ruptured papillary muscle or severe mitral regurgitation. This may have been because the mitral regurgitation was so severe that the resulting pressure equalisation between the left atrium and left ventricle allowed the development of only a small regurgitant jet (if any at all), which was missed by the Doppler and colour flow studies. In addition, although we agree that these transthoracic techniques are generally useful in diagnosing mitral regurgitation, in critically ill patients they have several limitations, including positioning problems and the increased interference with transmission of ultrasound as a consequence of mechanical ventilation, as we point out in our article. Transoesophageal techniques have the advantage of providing better resolution of the mitral valve apparatus as well as being much less limited in critically ill patients.

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Trends in cerebral palsy in Western Australia

EDITOR.—In their review of cerebral palsy in Western Australia in 1967-85 Fiona J Stanley and Linda Watson did not separate multiple births from singleton births,¹ although it is well known that the incidence is much higher in multiple births. In a study in Avon from 1969 to 1988, to be published later, the incidence of spastic cerebral palsy was 8.0/1000 in multiple births (figures of multiple births not available for 1981) compared with 1.53/1000 in singleton births; the total numbers of cases of cerebral palsy were 8.22/1000 and 1.94/1000 respectively.

It would be interesting, particularly to those of us studying multiple births, and might also give some pointers to the aetiology of cerebral palsy, if

the authors could divide their cases into singleton and multiple births.

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EDITOR.—Fiona J Stanley and Linda Watson's study of trends in stillbirths, neonatal mortality, and cerebral palsy in Western Australia during 1967-85 found large falls in the stillbirth and neonatal death rates but no concomitant fall in the rate of cerebral palsy.¹ Because the rate of cerebral palsy remained roughly the same over the period despite improvements in perinatal interventions aimed at reducing intrapartum asphyxia the authors conclude that birth asphyxia may not be a major cause of cerebral palsy. We do not think, however, that such a conclusion is justified by the data.

The authors note that, for low birthweight infants, the rise in cases of cerebral palsy mirrors the fall in perinatal mortality, possibly because infants now survive who previously would have died. Whether these newly surviving infants develop cerebral palsy because of factors occurring antenatally, perinatally, or postnatally, the observed trends would be the same, provided these factors would previously have led to the infants' deaths between 20 weeks of gestation and 28 days of life. From the data presented in the paper it is not possible to determine the relative contribution of factors operating in each period.

The "replacement" of some deaths by cases of cerebral palsy could be occurring at all weights. The relatively stable rates of cerebral palsy found may be the net result of a decrease in the rates among the infants who were surviving previously (which could be due to recent improvements in interventions) combined with "new" cases of cerebral palsy occurring among the increasing number of new survivors. Among births under 2000 g there is indeed a suggestion of such balancing. Whereas there is a pronounced fall in rates of cerebral palsy in the 1500-1999 g birthweight group, among births under 1500 g there is a rise. The data therefore do not preclude birth asphyxia being a major cause of cerebral palsy. It would be misleading for such equivocal findings to be presented "to lawyers, judges, parents, and obstetricians" as evidence that birth asphyxia is not an important putative cause of cerebral palsy.

Although it is unlikely to alter the results substantially, there are some inconsistencies in the numbers given in the tables of Stanley and Watson's paper (for example, 109 live births of unknown weight cannot give rise to 637 neonatal survivors).

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AUTHOR'S REPLY.—In answer to Ian MacGillivray, I and colleagues have already published a paper on cerebral palsy in multiple births from our Western Australian database.¹ In that paper we documented the rates for 1966-85 as follows: for singletons 2.4/1000 live births (95% confidence interval 2.2 to 2.5), for twins 6.3 (4.5 to 8.0), and for triplets 32.0 (3.7 to 59.5). Though the rates among multiple births are high and more of the infants are of low birthweight, they contributed only a small

proportion (55/932) of all cases of cerebral palsy in that period. The aetiology of cerebral palsy in low birthweight infants in multiple births is probably similar to that in singletons.

In a few cases a cotwin died antenatally, with resulting brain damage in the surviving twin. This is seen in our data and has been known for many years.² As rates of multiple births are rising it is important to keep monitoring the rates of cerebral palsy in these births.

We agree with Judith Glynn and David Leon's comments. It is impossible from our epidemiological data to ascertain when brain damage occurred. We agree that in infants of 1500 g who develop cerebral palsy the sequence could have started antenatally, perinatally, or postnatally and the epidemiological picture would be similar. We recently reviewed this.³

In normal birthweight infants it is also likely that there has been a shift from deaths to survivors with cerebral palsy, and possibly from survivors with cerebral palsy to non-handicapped survivors. We still believe that our data, in conjunction with other data to which we referred in our paper, can be interpreted as suggesting that birth asphyxia is not the main cause of cerebral palsy.

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Juvenile myoclonic epilepsy

EDITOR,—In their editorial P L Timmings and A Richens state that juvenile myoclonic epilepsy occurs in 10 of all cases of epilepsy.¹ This is a gross overestimate. Community based studies have shown a frequency of no more than 2%,^{2,4} and the paper that Timmings and Richens quote in support of their figure of one in 10 actually gives a figure of 4-6%.³ This is not an insignificant issue, as misdiagnosis is rife and may lead to incorrect treatment.

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AUTHORS' REPLY,—Discussion about the incidence and prevalence of juvenile myoclonic epilepsy is important. In our original draft we observed that "interest in juvenile myoclonic epilepsy has increased steadily and the reported incidence of 2-1%¹ among all epilepsy (1957) has risen to 11-9%² (1984)." This was reduced to "accounts for about 1 in 10 cases of epilepsy" in the final version. As S D Shorvon and J W A S Sander observe these figures are derived from hospital based groups, and the figures in unselected community based services may be lower. We also note, however, that

(a) physicians unfamiliar with a condition are less likely to diagnose it, and this clearly applies to juvenile myoclonic epilepsy as the mean time from presentation to correct diagnosis is measured in years^{3,6}; and (b) including paediatric patients in any survey of the prevalence of juvenile myoclonic epilepsy will tend to dilute the figure (the figure of 11-9% is based on an "almost exclusively adult population").

In a review Penry *et al* stated that juvenile myoclonic epilepsy represents 4-6% of all epilepsy³ but did not give any original figures, preferring to refer to Janz and Christian.^{1,2} In another review of juvenile myoclonic epilepsy Dreifuss stated that it affects about 7% of all adolescents and adults with epilepsy, but, again, no new data are cited.⁷ Interestingly, in a recently reported series of 180 patients referred over 21 months to a "new epilepsy" clinic in Britain the prevalence was 8-3%.⁶

We believe that juvenile myoclonic epilepsy makes up 5-10% of all epilepsy in adolescents and adults and that the papers referred to by Shorvon and Sander (with prevalences of around 2%) may be underestimating its frequency because of underrecognition and inclusion of paediatric data.⁸⁻¹⁰

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Selective serotonin reuptake inhibitors

EDITOR,—The correspondence^{1,2} on J Guy Edwards's editorial on selective serotonin reuptake inhibitors³ requires comment. Debbie Harrison chides Edwards for mentioning single reports of adverse reactions,¹ but such reports are vital as postmarketing surveillance and require attention. Harrison and S O Fradd² criticise Edwards for mentioning cost. This is not surprising from Harrison, who is an employee of the main pharmaceutical company in this field, and it is inappropriate for her to reprimand Edwards for having made up his mind already about the role of selective serotonin reuptake inhibitors—no more so than Harrison and her company have.

Fradd's outlook is particularly disappointing. Fradd oversimplifies the problem, implying that selective serotonin reuptake inhibitors are without serious side effects while tricyclic antidepressants, with which we have long experience, are highly toxic. To suggest that doctors should not consider the economic consequences of their prescribing to society as well as to individual patients is an outmoded concept that doctors should abandon. A professional decision to prescribe only selective serotonin reuptake inhibitors would cost £100-150

million a year, with inevitable consequences for other NHS services.

In reality, neither tricyclic antidepressants nor selective serotonin reuptake inhibitors are ideal for depression: both are slow to produce a benefit, and neither is effective in more than about 80% of cases. The selective serotonin reuptake inhibitors are a relatively new class of antidepressant whose full pharmacology and role in treatment are not yet known.^{4,5} To advocate a wholesale move to using them as first choice antidepressant treatment⁶ rather than more established and less expensive drugs, solely because of their safety in overdose, is unreasonable on medical, ethical, and economic grounds.

Should a doctor treat a depressed patient with a tried and trusted remedy or with a less established drug? The family of a patient who commits suicide by any means (and not just drug overdose) might well have a better case if the doctor prescribed the less established treatment; of course, a doctor should weigh each case on its own merits. In a court case the gold standard would probably be what the doctor's peers would reasonably do in the circumstances.

Edward's conclusion that selective serotonin reuptake inhibitors should be reserved for selected patients represents a balanced view of the current state of knowledge, and Harrison's contention that these drugs are an enormous advance in the treatment of depression is unwarranted.

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Value of Dundee coronary risk-disk

EDITOR,—Tony Randall and colleagues report that when they used the Dundee coronary risk-disk on the population of south Bedfordshire it failed to detect high risk subjects.¹ According to the registrar general's figures, the coronary risk for south Bedfordshire makes it, on the basis of crude death rates, the eighth safest health district in the land: Milton Keynes, west Berkshire, Huntingdon, Oxfordshire, west Surrey, Parkside, and north west Surrey have lower crude death rates. Tayside, on the other hand, is 15th from the top of Britain's 220 health districts in terms of coronary risk and is far more typical of the country as a whole.

Of the seven low risk areas, only three (including south Bedfordshire) have low age standardised rates—that is, the populations are untypically young. Thus a schedule for assessing risk such as the Dundee score will miss more of the single risk factors in a young population than in a population of average age distribution because one of the variables contributing to the score is age; in south Bedfordshire this is artificially low. Other factors must operate in Parkside, Berkshire, Oxford, and Cambridge: all have age standardised coronary death rates 63% of Tayside's rate.

A series of cholesterol concentrations measured in this practice gave an average of 6.1 mmol/l, so other factors in southern England must operate to reduce the coronary risk and the Dundee disk does