coccal antibody titre; and (d) clinical signs of meningococcal meningitis or septicaemia.

Of the 109 patients, 92 were admitted to hospital by their general practitioner, 13 were admitted through the accident and emergency department, three were found dead at home, and one was admitted after a domiciliary visit by a consultant paediatrician. Parenteral penicillin was given to 23 patients (21%) either by their general practitioner or by a casualty officer. A further seven patients had been taking oral antibiotics before presentation. During 1989, after the chief medical officer's recommendation about early use of penicillin¹ and widespread media coverage of a cluster of cases in this district, 14 of 27 patients (52%) received penicillin before admission. Excluding the patients who had been taking oral antibiotics before presentation, the overall case fatality rate during our study was 12% (one of the 23 patients (4%) given early penicillin died compared with 11 of the 79 (14%) not given it).

The frequency of early use of penicillin in suspected cases of meningococcal disease in our study was similar to that found by Strang and Pugh and Cartwright and colleagues. Early parenteral penicillin was associated with reduced mortality, and, although this was not significant, it is consistent with the trend observed by these other workers. General practitioners need to be continually reminded of the value of early treatment with parenteral penicillin whenever meningococcal disease is suspected.

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Giant cell arteritis

EDITOR,—Justin C Mason and Mark J Walport suggest that C reactive protein concentration may be a more effective marker of disease activity in giant cell arteritis than the erythrocyte sedimentation rate.¹ The study they cite was of 13 patients with polymyalgia rheumatica, and C reactive protein concentration reflected disease activity more accurately over the first two weeks of treatment.² We carried out a prospective study of 74 patients and found that the erythrocyte sedimentation rate correlated better than C reactive protein concentration with disease activity.³ The C reactive protein concentration fell more rapidly in the first two weeks after treatment, but assessing response at this stage is rarely difficult. Thereafter the erythrocyte sedimentation rate was a better marker.

I suggest, therefore, that in most circumstances the erythrocyte sedimentation rate could be safely used to monitor disease activity in polymyalgia rheumatica and giant cell arteritis.

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EDITOR,—By considering giant cell arteritis from a rheumatologist's viewpoint Justin C Mason and Mark J Walport make several statements in their editorial that are probably not applicable generally and certainly not applicable to giant cell arteritis causing visual loss.¹ The sensitivity of temporal artery biopsy is much higher than 60%: it correctly predicts the need for corticosteroid treatment in 94% of cases² and should be regarded as crucial to management.

Doctors have recourse to diagnostic methods other than "mystery and mystique." In ischaemic optic neuropathy both ocular pneumoplethysmography and appreciably delayed choroidal filling on fluorescein angiography are extremely useful in differentiating ischaemia due to giant cell arteritis from that due to non-arteric causes.^{3,4} Systemic markers of the underlying immunological disturbance can also be sought: both raised levels of IgG anticardiolipin antibodies and selective depletion of CD8+ lymphocytes in the peripheral blood are reliable.^{5,6}

The authors' advice on treatment, particularly for patients with visual loss, should be treated circumspectly. Graham *et al*'s recommendation of 250 mg of intravenous hydrocortisone at presentation coupled with a starting dose of 80 mg of prednisolone orally, reducing to 30 mg over the first 10 days, is the standard regimen.⁷ Regimens of lower steroid doses are more suited to the treatment of polymyalgia rheumatica, especially as inadequate steroid treatment has been identified as a possible factor in death in the acute phase of giant cell arteritis.⁸ Although use of inadequate steroid doses is to be deprecated, recommendations to use high dose intravenous pulses of steroid in acute visual loss are not based on convincing evidence.⁹ Monitoring the withdrawal of steroids by measuring the erythrocyte sedimentation rate is unreliable,¹⁰ and a better approach is to use a fixed withdrawal regimen.⁷ During a fixed withdrawal regimen the recurrence of symptoms or signs should be monitored (and the dose of steroids increased to the dose that previously controlled the recurrent features) and CD8+ lymphocyte counts measured serially.¹¹

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AUTHORS' REPLY.—Referral bias means that ophthalmologists, rheumatologists, and general practitioners each see a different part of the large clinical range of severity of giant cell arteritis. Ophthalmologists see a higher proportion of patients presenting with visual loss and therefore requiring more aggressive treatment. As far as

diagnosis is concerned, we do not question the importance of temporal artery biopsy. Such biopsies, however, yield negative results in up to one third of patients with symptoms suggestive of giant cell arteritis, with false negative rates of 10-61% reported.¹

We question the value of raised levels of anti-cardiolipin antibodies and reduced CD8+ lymphocyte counts both as diagnostic tools and as a means of monitoring disease activity in giant cell arteritis. Anticardiolipin antibodies may be a marker of a small subset of patients with ischaemic optic neuropathy as part of the antiphospholipid syndrome.^{2,3} There is no evidence that the CD8+ lymphocyte count is any more than a very expensive indirect correlate of the acute phase response.⁴

The prescription of high dose corticosteroid regimens in patients with giant cell arteritis presenting with visual loss is not in dispute, though no "gold standard" regimen exists. There is, however, evidence to support lower initial doses of corticosteroids in patients without visual loss, and these do not seem to be associated with an increased risk of relapse.⁵ There is no reliable method of predicting which patients will relapse while taking steroids,⁶ and recurrence of severe disease in patients taking stable doses of steroids is well described.⁷ Too rapid a reduction in the dose of corticosteroids is probably the main risk factor. We therefore favour a gradual reduction of treatment, not confined to a fixed regimen but dictated by careful and frequent monitoring of the patient's symptoms and acute phase response.

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Removal of central venous catheter and venous air embolism

EDITOR,—I share Peter Mennim and colleagues' concerns about air embolism occurring when a central venous catheter is removed and agree with their recommendations for preventing this complication.¹ I first witnessed a death due to venous air embolism via a central venous catheter in 1973.² The postmortem examination was carried out by the late Professor Keith Simpson, who coincidentally reported the first episodes of venous air embolism via transfusion equipment.³ He mentioned that he had seen several other deaths due to air embolism via central venous catheters and criticised the integrity of the devices. Unfortunately, little has changed during the past 20 years.

The incidence of important air embolism associated with central venous catheters is, however, much higher than the authors suggest. I have been carrying out a regular search of published work for several years: to date 151 cases in which cardiorespiratory distress or considerable neurological morbidity occurred have been reported and

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