of patients with the syndrome when a sensitive but technically demanding technique was used.¹⁰ This assay detected IgM antibodies that seemed to react with an antigen in myelin, called the Forssman antigen, and fell in titre in the first few weeks of the neuropathy. A separate IgG antibody which reacts with gangliosides G_{MI} and G_{DIB} occurs in about 15% of patients.11 These antibodies seem more frequent in patients with severe disease and are associated with previous infection with campylobacter. Such antibodies could be secondary to whatever process is causing the demyelination, and international collaborative efforts are underway to clarify their role.

Research into the role of specific treatment for the syndrome has been more rewarding. Trials of plasma exchange have shown convincing benefits in the more severe cases of the syndrome, particularly in those patients who become bedbound and receive plasma exchange within one to two weeks of the onset of their disease.^{12 13} Patients with milder disease who receive plasma exchange later in the course of their illness may benefit, but the relevant studies have not been performed. Plasma exchange is expensive and, although morbidity is small in experienced hands, other treatment options are needed.

Steroids were a popular treatment until an influential but small controlled trial in 1978 failed to provide evidence of benefit.14 A trial conducted by the Medical Research Council is in progress to assess a possible role for methylprednisolone, but preliminary results suggest that it is ineffective.¹⁵ Gammaglobulin is the latest treatment option, and interim results of a Dutch trial were encouraging (FCA Van der Meche et al, Peripheral Neuropathy Association meeting, Oxford 1990). Further studies will be needed to define its role.

Such treatment is crude, and a better understanding of the pathogenesis of the disease may eventually lead to more specific modes of treatment. Until then there is no doubt that excellent intensive care is vital to reduce morbidity and mortality. Autonomic complications are difficult to manage and may rarely lead to sudden death. It is worrying that mortality in a British survey could be as high as 13%³ whereas centres specialising in neurological intensive care quote a rate of 1.3%.¹⁶ Such figures are clearly influenced by many factors, including selection bias, but they are understandably used by those who favour increased specialisation in the management of this fascinating and challenging condition. JOHN WINER

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Preventing infection in laboratories

A new code promotes even safer practices

Before 1974 only limited parts of health care premises were subject to health and safety inspection. When the Health and Safety at Work Act 1974 extended occupational and safety legislation to all areas of health care the need for guidance covering clinical laboratories became clear. The original code of practice on preventing laboratory acquired infections, the Howie code,¹ was issued in 1978, but since then it has been modified, added to, and partially superseded. Now there is a completely new code of practice.²⁻⁵

The original Howie code was based on a report of a working party set up by the Department of Health and Social Security to consider ways of preventing laboratory acquired infection. The report was never published—only the code, which immediately prompted some controversy. Although it had been widely circulated beforehand for consultation, its publication was followed by considerable discussion about the real and apparent cost implications of the code.67 It was always clear that it would require reviewing and updating, and this has now been done by technical subcommittees of the Health and Safety Commission's health services advisory group.

The new code does not have the force of law, but, as with its predecessor, health and safety inspectors (and courts and tribunals) are likely to use it as the benchmark for what is reasonable and acceptable in the laboratory. Nevertheless, one of the original criticisms of the Howie code was that it was sometimes overinterpreted by overzealous safety inspectors, and overinterpretation remains a danger. Strict application of any code may so restrict activity that it defeats the object of the exercise. Too tight restrictions on laboratories (or other health care departments) may prevent them from fulfilling their main task of contributing to health care.

In essence, the prevention of infection in clinical laboratories requires the application of common sense. It does not require large amounts of money, but it does mean that all staff coming into contact with biological specimens should be trained about the dangers of inappropriate activity both to themselves and to others. Furthermore, planners will have to include adequate provision for safety in new laboratories, and these measures must not be adversely affected by considerations of economy. Collins summarised the problem as follows: "Diseases cost money, but good techniques cost no more than bad ones; good equipment and premises cost no more, and in the long run much less, than bad ones."8

Within clinical laboratories the laboratory manager is responsible for ensuring that safety policies are written down and practised by all who work there. There is an increasing trend, however, to have equipment available in other clinical areas to provide rapid results or for emergency testing-for example, reflectometers in outpatient clinics and blood gas analysers in intensive care units. These areas should be regarded as extensions of the clinical laboratory, and it must be made perfectly clear who is responsible for the apparatus and for training and supervising the staff using it.⁹ The further extension of side room testing to community based clinics and general practitioners' surgeries implies that the dangers of infection from biological specimens need to be widely known. It also needs to be made clear who is responsible-and thus answerable for accidents-in these settings.

One aspect of the code that does require emphasis and wide dissemination is the onus it places on the person who takes specimens and sends them to the laboratory to make sure they are safe to handle and that any known risk is identified. This does not mean that all risks will be identified as new bloodborne infections continue to be discovered.

Despite the caveats about overrestrictive interpretation safety at work is of paramount importance in the health service and the updated code is a welcome contribution. The code takes the form of three documents-on clinical laboratories, on the mortuary and postmortem room, and model rules for staff and visitors. Health authorities are responsible for seeing that laboratories and mortuaries conform to the new code, but the model rules should be essential reading for all health service staff.

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Using tympanometry to detect glue ear in general practice

Overreliance will lead to overtreatment

The most common use of tympanometry is to show the negative middle ear pressure associated with otitis media with effusion. It is non-invasive, rapid to perform, and well accepted even by young children and infants. With the introduction of portable and, more recently, hand held equipment tympanometry has become widely available and cheap to perform. Should general practitioners be using tympanometry to detect otitis media with effusion?

Tympanometry measures the proportion of an acoustic signal transferred from the external to the internal ear and compares the absorbed and reflected components to indicate middle ear pressure. Comparison with findings at myringotomy shows that the technique has a high sensitivity (about 90%) and specificity (about 75%)¹: most children with otitis media with effusion show a flat, low compliance trace (type B) and very few the normal symmetrical peaked curve (type A). Nevertheless, these results may be no better than those obtainable by a well trained clinician using pneumatic otoscopy to examine the ear. Some training in using the tympanometer is necessary because an inadequate seal in the meatus may produce a flat trace which is then wrongly interpreted as showing middle ear fluid.

At present tympanometry is used both for screening and for diagnosis by audiological technicians. Since tympanometry cannot distinguish marginal from mild to moderate hearing loss² it is usually combined with an assessment of hearing loss. Our district's pattern of referral for otitis media with effusion may be universal: most cases are first suspected by parents, and the children are referred to hospital after a period of observation with otoscopy, tympanometry, and hearing assessment by senior clinical medical officers in audiology. de Melker suggests that general practitioners using a hand held microtympanometer can also reliably diagnose otitis media with effusion (p 96).

The incidence and clinical course of otitis media in childhood are reasons for caution about wider, uncontrolled use of tympanometry. The incidence of glue ear peaks during the second half of the first year of life and declines gradually thereafter.⁴ Twenty to thirty per cent of children under 6 develop the condition, but prevalence rates vary according to the diagnostic techniques used. Ninety per cent of effusions after otitis media resolve within three months. In a study of bilateral disease 322 children referred to hospital were assessed to produce a cohort of 150 in whom the disease had remained constant in each ear for three months before surgery.⁵ Even in the unoperated ears almost 30% of the effusions had resolved within a year after surgery to the contralateral ear. Thus even in severe cases there is a high spontaneous resolution rate that is mainly age related.

Nevertheless, in 1985 in England and Wales about 60 000 children were treated for otitis media with myringotomy or grommets. A further 16000 underwent adenoidectomies, some of which may have been for ear disease. A recent survey showed that 64% of otolaryngologists waited for a period to confirm the persistence of middle ear fluid before listing the child for surgery-71% of them for two months or more.⁶ Eighty five per cent of surgeons used tympanometry for diagnosing otitis media with effusion, as well as otoscopy and audiometry. A recent study from Oxford, however, showed that 34% of children undergoing surgery for otitis media with effusion were found not to have middle ear fluid.⁷

The danger of using tympanometry more widely in general practice is that its high sensitivity for detecting otitis media with effusion together with the high rate of spontaneous resolution may lead to over-referral of mild cases. Failure to wait and retest at each stage may lead to overtreatment.8 Tympanometry can undoubtedly help general practitioners improve their diagnostic ability in middle ear disease, but it