

antibody at about six to eight weeks after the onset of illness.¹⁶ During reinfection, however, there is often no complement fixing antibody response. Cross reactions between chlamydial species—even on “type specific” microimmunofluorescent tests—may give confusing results, and detailed prolonged serological investigations may be needed to identify the causative organism.⁴ Techniques such as DNA amplification by the polymerase chain reaction may prove useful,¹⁹ but much of our current knowledge of chlamydial respiratory infection is based on microimmunofluorescent serological tests.

Tetracycline and erythromycin have formed the basis of antibiotic treatment of *C psittaci* and *C trachomatis*, and these antibiotics are also effective against *C pneumoniae*, although prolonged treatment for 10–14 days with doses of 2 g/day may be necessary.^{13 20} Ofloxacin and clarithromycin may be effective alternatives, although this supposition is based on initial laboratory tests and limited clinical experience.²⁰

The explosion of knowledge resulting from the discovery of *C pneumoniae* has revolutionised concepts of chlamydial respiratory infection. Research suggests that this organism may account for many cases of respiratory infection in which no pathogen is identified. Further preliminary reports suggest that *C pneumoniae* also plays a part in asthma,²¹ sarcoidosis,²² and ischaemic heart disease.²³ The limitations of current diagnostic tests, however, mean that the precise role of chlamydial infections in respiratory disease in the United Kingdom remains to be defined.

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1 Grayston JT, Kuo C, Wang SP, Altman J. A new Chlamydia psittaci strain, TWAR, isolated in acute respiratory tract infections. *N Engl J Med* 1986;315:161–8.

- 2 Schachter J. Chlamydia psittaci—“re-emergence” of a forgotten pathogen. *N Engl J Med* 1986;315:189–91.
- 3 Grayston JT. Chlamydia pneumoniae, strain TWAR. *Chest* 1989;95:664–9.
- 4 Bourke SJ, Carrington D, Frew CE, Stevenson RD, Banham SW. Serological cross-reactivity among chlamydial strains in a family outbreak of psittacosis. *J Infect* 1989;19:41–5.
- 5 Weiss SG, Newcomb RW, Beem MO. Pulmonary assessment of children after chlamydial pneumonia of infancy. *J Pediatr* 1986;108:659–64.
- 6 Wheeler WB, Kurachek SC, Lobas JG, Einzig MJ. Acute hypoxemic respiratory failure caused by Chlamydia trachomatis and diagnosed by flexible bronchoscopy. *Am Rev Respir Dis* 1990;142:471–3.
- 7 Wang SP, Grayston JT. Population prevalence antibody to Chlamydia pneumoniae, strain TWAR. In: Bowie WR, Caldwell HD, Jones RP, Mardh PA, Ridgway GL, Schachter J, et al, eds. *Chlamydial infections*. Cambridge: Cambridge University Press, 1990:402–5.
- 8 Darougar S, Forsey T, Brewerton DA, Rogers KL. Prevalence of antichlamydial antibody in London blood donors. *Br J Vener Dis* 1980;56:404–7.
- 9 Forsey T, Darougar S, Treharne JD. Prevalence in human beings of antibodies to Chlamydia IOL-207, an atypical strain of chlamydia. *J Infect* 1986;12:145–52.
- 10 Bourke SJ, Carrington D, Frew CE, McSharry CP, Boyd G. A comparison of the seroepidemiology of chlamydial infection in pigeon fanciers and farmers in the UK. *J Infect* 1992;25:91–8.
- 11 Fryden A, Kihlstrom E, Maller R, Persson K, Romanus V, Ansehn S. A clinical and epidemiological study of “ornithosis” caused by Chlamydia psittaci and Chlamydia pneumoniae (strain TWAR). *Scand J Infect Dis* 1989;21:681–91.
- 12 Ghosh K, Frew CE, Carrington D. A family outbreak of Chlamydia pneumoniae infection. *J Infect* 1992;25(suppl 1):99–103.
- 13 Myhra W, Mordhorst CH, Wang SP, Grayston JT. Clinical features of Chlamydia pneumoniae, strain TWAR, infection in Denmark 1975–1987. In: Bowie WR, Caldwell HD, Jones RP, Mardh PA, Ridgway GL, Schachter J, et al, eds. *Chlamydial infections*. Cambridge: Cambridge University Press, 1990:422–5.
- 14 Grayston JT, Diwan VK, Cooney M, Wang SP. Community and hospital-acquired pneumonia associated with Chlamydia TWAR infection demonstrated serologically. *Arch Intern Med* 1989;149:169–73.
- 15 Sundeloef B, Gnarpe J, Gnarpe H, Darougar S. Chlamydia pneumoniae pneumonia in Sweden. In: Bowie WR, Caldwell HD, Jones RP, Mardh PA, Ridgway GL, Schachter J, et al, eds. *Chlamydial infections*. Cambridge: Cambridge University Press, 1990:426–8.
- 16 Marrie TJ, Grayston JT, Wang SP, Kuo C. Pneumonia associated with the TWAR strain of chlamydia. *Ann Intern Med* 1987;106:507–11.
- 17 Augenbraun MH, Roblin RM, Chirgwin K, Landman D, Hammerschlag MR. Isolation of chlamydia pneumoniae from the lungs of patients infected with the human immunodeficiency virus. *J Clin Microbiol* 1991;29:401–2.
- 18 Tack KJ, Rasp FL, Hanto D, Peterson PK, O’Leary M, Simmons RL, et al. Isolation of Chlamydia trachomatis from the lower respiratory tract of adults. *Lancet* 1980;i:116–20.
- 19 Gaydos CA, Quinn TC, Eiden JJ. Identification of Chlamydia pneumoniae by DNA amplification of the 16S rRNA gene. *J Clin Microbiol* 1992;30:796–800.
- 20 Fenelon LE, Mumtaz G, Ridgway GL. The in-vitro antibiotic susceptibility of Chlamydia pneumoniae. *J Antimicrob Chemother* 1990;26:763–7.
- 21 Hahn DL, Dodge RW, Golubjatnikov R. Association of Chlamydia pneumoniae (strain TWAR) infection with wheezing, asthmatic bronchitis, and adult-onset asthma. *JAMA* 1991;266:225–30.
- 22 Groenhagen-Riska C, Saikku P, Riska H, Froese B, Grayston JT. Antibodies to TWAR—a novel type of Chlamydia in sarcoidosis. In: Grassic C, Rizatto G, Pozzi E, eds. *Sarcoidosis and other granulomatous disorders*. Amsterdam: Elsevier Science, 1988:297–301.
- 23 Saikku P, Leinonen M, Tenkanen L, Linnanmaeki E, Ekman MR, Manninen V, et al. Chronic Chlamydia pneumoniae infection as a risk factor for coronary heart disease in the Helsinki Heart Study. *Ann Intern Med* 1992;116:273–8.

Patients with brain injuries

A national rehabilitation service is needed to lift the burden from carers

In a regional health authority with a population of 3.5 million, each year around 140 people survive brain trauma of moderate or worse severity. These join a population in Britain of up to 70 000 disabled survivors, most of whom have a normal life expectancy.¹ These estimates exclude patients with brain damage associated with cerebral tumours, vascular accidents, metabolic disorders, and other causes; and there are many more victims of minor brain injuries. These last may be declared fit yet have undetected and disabling cognitive deficits.

Injury to the brain may be accompanied by severe physical problems that require intensive early treatment and extended rehabilitation. At first these may overshadow important cognitive sequelae such as amnesia, disorientation, and perceptual disorder.² The most troublesome long term morbidity, however, is caused by behavioural and emotional consequences, including sexual disinhibition, aggression, apathy, anxiety, and lability of mood.³ Patients with these symptoms cannot participate in, and are usually excluded from, conventional rehabilitation programmes. They may languish in acute beds—we know of one patient who occupied a surgical bed for 10 years because of his physical dependency and disordered behaviour.

The development of NHS services for patients with brain

injuries has so far been haphazard. Voluntary self help organisations such as Headway (the National Head Injuries Association) and Amnass (the Amnesia Association, now part of Headway) were set up—at least in part—because services were so poor. For example, Headway Houses provide continuing care in the community for some of the disabled people with brain injuries. By contrast with the NHS, the independent sector in Britain has been responsible for much innovation, research, and service development for patients with brain injuries. The main health insurers are not, however, prepared to finance years of rehabilitation, so these facilities are available only to those patients who are paid for by insurance settlements or health authorities.

These problems have been recognised for 20 years or more, and reports have been produced by the Royal College of Physicians,⁴ the Medical Disability Society (now the British Society for Rehabilitation Medicine),¹ and the Royal College of Psychiatrists.⁵ These have spelt out the size and complexity of the problem and the need for a properly coordinated national strategy and effective training of professional staff. Rehabilitation requires a multidisciplinary approach, incorporating nursing; psychology; occupational, speech, music, and art therapies; physiotherapy; clinical engineering; dietetics; oral hygiene; and social work. The problem for the

medical profession is which specialty should train doctors in the rehabilitation of patients with brain injuries. Physicians, neurologists, neurosurgeons, and psychiatrists can contribute, but none of these is trained to provide all the skills required. In practice the setting up of a brain injury rehabilitation service requires a consultant in one of these specialties to acquire the full range of skills through strong interdisciplinary cooperation and mutual learning. A broad medical training makes a consultant the ideal team leader, but he or she must be prepared to learn continually from colleagues.

Recovery after brain injury is slow, and its mechanisms are poorly understood.⁶ The process of rehabilitation is correspondingly lengthy and as yet only partially evaluated.⁷ Intensive behaviour modification benefits some patients, but such resources are scarce.⁸ In these times of financial constraint what service developments can we reasonably recommend?

We believe that the needs of patients with brain injuries would be best met by a network of resources. Multidisciplinary teams based in regional specialist centres with 20 beds would deal with the most complicated cases, but they could not provide the whole service for a large area. Each district should have a head injury coordinator with access to the relevant local professionals and a case register. Coordinators should give advice on the care of patients during early recovery and help to prevent the development of avoidable behavioural problems. They should liaise between regional and district facilities and follow up patients in the community. Coordinators would need to be enthusiastic, energetic, and willing to learn; they might be recruited from several professional backgrounds.

Most patients with brain injuries live at home, cared for by their relatives and friends.⁹ This may be preferable to institutional care, but it is often achieved only at enormous emotional, physical, and financial cost to the carers. Families need much emotional and practical support from health, local authority, and social services.¹⁰

What are the prospects for the future? We have been encouraged by the Department of Health's initiative "Model services for the brain injured." This should result in a

systematic evaluation of specially funded projects ranging from acute aspects of management to long term consequences. The change to a purchaser-provider system of funding will present both opportunities and threats. Regional health authorities will no longer purchase this type of service on behalf of districts—which should see a compensatory increase in their funding. In the next few years weighted capitation will redistribute resources between districts and some will be losers. We fear that the purchase of brain injury services might be given a low priority by such authorities. This would prevent the establishment of a comprehensive national service for rehabilitating patients with brain injuries. Doctors can and should influence this process by taking every opportunity to impress on the Department of Health and purchasers the importance of providing for this large disadvantaged group and their carers.

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- 1 Medical Disability Society. *The management of traumatic brain injury*. London: Development Trust for the Young Disabled, 1988.
- 2 Brooks N. Cognitive deficits after head injury. In: Brooks N, ed. *Closed head injury: psychological, social and family consequences*. Oxford: Oxford University Press, 1984:44-73.
- 3 Thomsen IV. Late outcome of very severe blunt head trauma: a 10-15 year second follow up. *J Neurol Neurosurg Psychiatry* 1984;47:260-8.
- 4 Royal College of Physicians of London. Physical disability in 1986 and beyond. *J R Coll Phys Lond* 1986;20:3-37.
- 5 Working Group of the Research Committee of Royal College of Psychiatrists. Services for brain injured adults. *Bulletin of the Royal College of Psychiatrists* 1991;15:513-8.
- 6 Gloag D. Needs and opportunities in rehabilitation: rehabilitation after head injury. I. Cognitive problems. *BMJ* 1985;290:834-7.
- 7 McClelland RJ. Psychosocial sequelae of head injury—atomy of a relationship. *Br J Psychiatry* 1988;153:141-6.
- 8 Eames P, Wood R. Rehabilitation after severe brain injury. *J Neurol Neurosurg Psychiatry* 1985;48:613-9.
- 9 Bryden J. How many head injured? In: Wood R, Eames P, eds. *Models of brain injury rehabilitation*. London: Chapman and Hall, 1989:17-27.
- 10 Brooks N, Campsie L, Symington C, Beattie A, McKinlay W. The five year outcome of severe blunt head injury: a relative's view. *J Neurol Neurosurg Psychiatry* 1986;47:764-70.

Treating hyperhidrosis

Endoscopic transthoracic sympathectomy may be the best treatment

Primary palmar and axillary hyperhidrosis—sweating that exceeds that needed for thermoregulation—affects between 0.6% and 1% of the population.¹ Usually no cause is found, although it may be secondary to an underlying endocrinological abnormality (such as hyperthyroidism, pheochromocytoma, diabetes mellitus, acromegaly, hyperpituitarism, or the carcinoid syndrome) or inflammatory condition (such as chronic infection, autoimmune neuropathy, or gout). It may easily be overlooked, especially in men. Most patients are young women, some of whom suffer severe and obvious dripping of the hands. The condition brings psychological, social, educational, and occupational problems.

Medical regimens should be tried,² but these are rarely effective when the hyperhidrosis is severe. In mild cases topical saturated aluminium chloride hexahydrate in absolute alcohol has been used with some success. Mild reaction from such treatment may be reduced by a weak topical steroid. Anticholinergic drugs are helpful in some cases but in others

troublesome dry mouth and blurring of vision prevent their use. Iontophoresis with plain tap water³ or with induction of anticholinergic agents into the skin may be used with relief for four to six weeks, but the treatment is messy and time consuming. Calcium channel blocking with diltiazem has been recommended for hereditary emotional hyperhidrosis.⁴

In the 30% of patients who remain appreciably disabled only surgery can provide permanent improvement. For hyperhidrosis affecting only the axilla subcutaneous curettage or excision of the skin bearing the eccrine glands at the apex of the axilla has been used for years, but these procedures fail in one fifth of cases⁵ and may cause permanent scarring and loss of arm mobility.

When the condition affects the hand and axilla upper dorsal sympathectomy is now the treatment of choice. A clear understanding of the distribution and severity of the sweating is required by both surgeon and patient if a satisfactory outcome is to be achieved. The operation may be preceded by