and the culture result may be negative in some cases. When antibiotics have been given before culturing blood or cerebrospinal fluid, diagnosis may still be confirmed by countercurrent immunoelectrophoresis or latex agglutination (though their value is doubtful in group B infections).^{6 26} In fulminant disease treatment must be started immediately without waiting for results of investigations (or a consultant opinion). A recent DHSS circular (3 February 1988) has suggested that doctors should give penicillin to patients with suspected meningococcal infection before transferring them to hospital; this may be particularly sensible in circumstances where there may be a delay. The antibiotic cover should be broad spectrum and the popular regimen is intravenous benzylpenicillin 3-4 MU (60 000—70 000 units/kg in children) four hourly and chloramphenicol 1 g (20-25 mg/kg in children) six hourly. The chloramphenicol may be withdrawn once meningococcal infection has been confirmed. The recommended duration of treatment varies from one to two weeks, though shorter courses have been successful.²⁷⁻²⁹ An alternative effective drug is ceftriaxone.³⁰

Overwhelming sepsis should ideally be treated in an intensive therapy unit with close haemodynamic, biochemical, and haematological monitoring. The patient may need assisted ventilation, circulatory support, or renal dialysis. The possibility of acute haemorrhagic adrenal failure must be kept in mind and hydrocortisone given if there is any suspicion that the patient might have it. The endotoxaemia may be severe enough to kill despite these measures. It might be treated by plasmapheresis, leucapheresis, and exchange transfusion,^{31 32} but further studies are needed before these measures are recommended.

Though penicillin is almost invariably effective in systemic infection it may not be adequate in clearing the carrier state,³³ and it is generally (though not universally) agreed that after the penicillin the patient should be given rifampicin 600 mg or 10 mg/kg body weight (5 mg/kg for children under 1 year) twice daily for two days.⁴ Chemoprophylaxis is indicated only for close family contacts,³³ especially children and young adults. Sulphonamides, which have been widely used in the past, are not reliable agents for prophylaxis because of the high incidence of resistant strains; they may be used when a strain associated with a specific outbreak is known to be sensitive. Rifampicin is therefore the prophylactic drug of choice (at the dosage mentioned), though rare failures have been reported. Vaccination is not generally recommended except in epidemics, and vaccines are available only against groups A, C, Y, and W135, but not group B.^{4 34} Mortality in this condition remains depressingly high—about 10% in meningitis, rising to as much as 50% in septicaemia without localising signs.^{4 34}

G VENKAT RAMAN

Consultant Physician and Nephrologist,
St Mary's Hospital,
Portsmouth PO3 6AD

- Jones DM. Epidemiology of meningococcal infection. *Br J Hosp Med* 1987;37:383.
- Poolman JT, Lind I, Jonsdottir K, Froholm LO, Jones DM, Zanen HC. Meningococcal serotypes and serogroup B disease in North-West Europe. *Lancet* 1986;ii:555-7.
- Abbott JD, Jones DM, Painter MJ, Young SEJ. The epidemiology of meningococcal infections in England and Wales 1912-1983. *J Infect* 1985;11:241-57.
- Anonymous. Report from the PHLS Communicable Disease Surveillance Centre. *Br Med J* 1986;292:1447-8.
- Weinstein L. Bacterial meningitis. *Med Clin North Am* 1985;69:219-29.
- Christie AB. *Infectious diseases: Epidemiology and Clinical Practice*. 3rd ed. London: Churchill Livingstone, 1980:607-10.
- Petersen BH, Lee TJ, Snyderman R, Brooks GF. Neisseria meningitidis and neisseria gonorrhoeae bacteremia associated with C6, C7, or C8 deficiency. *Ann Intern Med* 1979;90:917-20.
- Ellison RT, Kohler PF, Curd JG, Judson FN, Reller LB. Prevalence of congenital or acquired complement deficiency in patients with sporadic meningococcal disease. *N Engl J Med* 1983;308:913-6.

- Stiehm ER, Damrosch DS. Factors in the prognosis of meningococcal infection. *J Pediatr* 1966;68:457-67.
- Niklasson P, Lundbergh P, Strandell T. Prognostic factors in meningococcal disease. *Scand J Infect Dis* 1971;3:17-25.
- Toews WH, Bass JW. Skin manifestations of meningococcal infection. *Am J Dis Child* 1974;127:173-6.
- Greenwood BM, Onyewo II, Whittle HC. Complement and meningococcal infection. *Br Med J* 1976;ii:797-9.
- Wagge A, Halstensen A, Espevik T. Association between tumour necrosis factor in serum and fatal outcome in patients with meningococcal disease. *Lancet* 1987;ii:355-7.
- Powars DR, Rogers ZR, Patch MJ, McGehee WG, Francis RB. Purpura fulminans with acquired deficiencies of proteins C and S. *N Engl J Med* 1987;317:571-2.
- Kaplan JM, Gronvik KO, Murgita RA. Selective impairment of B cell function by neisseria meningitidis. *Cell Immunol* 1986;100:247-59.
- Sotto MN, Langer B, Shimizu SH, De Brito T. Pathogenesis of cutaneous lesions in acute meningococemia in humans: Light, immunofluorescent and electron microscopic studies of skin biopsy specimens. *J Infect Dis* 1976;133:506-14.
- Mier AK, Havard CWH. Meningococcal septicaemia presenting as drug hypersensitivity. *J R Soc Med* 1986;79:428-9.
- Conly JM, Ronald AR. Cerebrospinal fluid as a diagnostic body fluid. *Am J Med* 1983;75(1B):102-8.
- Mahida Y, Noone M. Atypical presentation of meningococcal meningitis. *J Infect* 1986;13:277-9.
- Schaad UB. Arthritis in disease due to Neisseria meningitidis. *Rev Infect Dis* 1980;2:880-8.
- Rompalo AM, Hook EW, Roberts PL, Ramsey PG, Handsfield HH, Holmes K. The acute arthritis-dermatitis syndrome. *Arch Intern Med* 1987;147:281-3.
- Ramsdale DR, Kaul TJ, Coulshed N. Primary meningococcal pericarditis with tamponade. *Postgrad Med J* 1985;61:1067-8.
- Sacks HS. Meningococcal pneumonia and empyema. *Am J Med* 1986;80:290-1.
- Givan KF, Thomas BW, Johnston AG. Isolation of neisseria meningitidis from the urethra, cervix and anal canal: Further observations. *British Journal of Venereal Diseases* 1977;53:109-12.
- Greenwood BM, Whittle HC, Bryceson ADM. Allergic complications of meningococcal disease II-Immunological investigations. *Br Med J* 1973;ii:737-40.
- Martin WJ. Rapid and reliable techniques for the laboratory detection of bacterial meningitis. *Am J Med* 1983;75(1B):119-23.
- MacFarlane JT, Anjorin FI, Cleland PG, et al. Single injection treatment of meningococcal meningitis: I. Long-acting penicillin. *Trans R Soc Trop Med Hyg* 1979;73:693-7.
- Wali SS, MacFarlane JT, Weir WRC, et al. Single injection treatment of meningococcal meningitis: II. Long-acting chloramphenicol. *Trans R Soc Trop Med Hyg* 1979;73:698-702.
- Viladrich PF, Pallares R, Ariza J, Rufi G, Gudiol F. Four days of penicillin therapy for meningococcal meningitis. *Arch Intern Med* 1986;146:2380-2.
- Martin E. Once daily administration of ceftriaxone in the treatment of meningitis and other serious infections in children. *Eur J Clin Microbiol* 1983;2:509-15.
- Schwartzmann WB, Tillotson JR, Taft EG, Wright E. Plasmapheresis for meningococemia with disseminated intravascular coagulation. *N Engl J Med* 1979;300:1277.
- Bjorvatn K, Bjertnaes L, Fadnes HO, et al. Meningococcal septicaemia treated with combined plasmapheresis and leucapheresis or with blood exchange. *Br Med J* 1984;288:439-41.
- Smith H. Chemoprophylaxis of meningitis. *Arch Dis Child* 1986;61:4-5.
- Anonymous. Defences against meningococcal infections. *Lancet* 1985;ii:929-30.

After the horror

Post-traumatic stress disorder is formal terminology for a common condition, the psychological problems that follow exposure to events such as the Bradford football stadium fire, the Zeebrugge ferry disaster, the Vietnam war, or some less well publicised and more private horror such as assault or rape. These psychological problems may be severe, prolonged, and disabling, and they include intrusive memories, flashbacks, anxiety, numbing of feelings, and irritability. The account of a survivor of the Bradford soccer stadium fire illustrates phenomena associated with the condition.

It's hard for me to talk about it. I go hot and cold and I can't stop me hands trembling. I didn't used to be able to talk about it at all . . . I thought I was beginning to get over it but then I went to the memorial service and that brought it all back . . . I didn't feel I could go on, I wanted to talk to somebody because I was feeling very disturbed. . . . One of my main things was I felt very lonely and couldn't talk to anyone properly and I was feeling very scared and vulnerable all the time and having bad dreams. And I did have very bad guilt feelings. Everyone in the stand had been rushing about, it was every man for himself and I felt very bad about that. I was all the time thinking why was it me that had got out, had I pushed others aside and just saved myself? . . . I've only been back to the ground once since, so far . . . I forced myself to go . . . I did it as a test. I came through it and so I know now I'm getting stronger in myself.¹

The past decade has seen a blossoming of interest in such experiences, but they have been recognised for well over a century. Attempts to find meaning in the folly that was the Vietnam war and growing acceptance of the Vietnam veteran as a victim rather than a perpetrator have added new

legitimacy to the diagnosis of post-traumatic stress disorder caused by the trauma of combat. An early description was given by Hammond based on clinical observations during the American civil war,² while DaCosta in 1871 erroneously attributed to an "irritable heart" a syndrome among soldiers characterised by palpitations, chest pain or heaviness, shortness of breath, sweating, and gastrointestinal disturbances.³

After complaints about the harsh way Austrian military neurotics were treated in the first world war, Freud implied that if the external threat was great enough any individual would develop neurotic symptoms.⁴ He described trauma as occurring when the ego is overwhelmed "as a consequence of an extensive breach being made in the protective shield against stimuli."⁵ Traumatic neurosis, he acknowledged, led to attempts to deal with the trauma by remembering, repeating, and fixating; and the defensive reactions of avoidance, inhibition, and phobia.⁶ Some of the best recent work on this subject has been done by Horowitz, who has studied peacetime trauma such as bereavement, accident, and the loss of a limb.⁷ He evolved the impact of events scale to measure such phenomena.⁸

The American Psychiatric Association lists the following criteria for diagnosing post-traumatic stress disorder⁹:

- (a) exposure to a recognisable trauma or stressor;
- (b) the trauma is persistently re-experienced in at least one of the following ways: recurrent intrusive and distressing recollections or dreams, re-experiencing in actions or feelings (or traumatic play for children), or extensive distress at reminders;
- (c) persistent avoidance of stimuli associated with the trauma or numbing of general responsiveness; these were not present before;
- (d) Persistent symptoms of increased arousal not present before as shown by irritability, sleep difficulties, excessive vigilance, startle responses, or reactions to trigger stress;
- (e) These criteria (b, c, d) have occurred during at least one month. Special note is made of effects in children and that onset may be delayed.

Brett and Ostroff suggested modification to the association's model by hypothesising that there are two distinct dimensions to the disorder: one having to do with repetitions of ideational, affective, and somatic aspects of the trauma; and the other having to do with efforts to avoid or defend against such repetitions.¹⁰ Other commentators still doubt that post-traumatic stress disorder is a valid syndrome.¹¹

Clinically the disorder may present some time after onset with intrusive phenomena (nightmares, flashbacks, and panic) or repressive phenomena (numbing of feelings and withdrawal) predominating. Why exposure to trauma should lead to these responses in some but not others is uncertain, but an important factor may be the severity of the stress—particularly in terms of gruesomeness and encounters with death, destruction, and violence.¹²⁻¹⁵ Traumatic experiences may induce reactive phenomena in many if not most people in the days immediately afterwards but they usually fade within a few weeks. Rising or persisting anxiety towards the end of the first week may indicate a risk of a serious disorder developing and suggests the need for early intervention.

The course of the disorder may be acute, subacute, chronic, or fluctuating and it may have a delayed onset. Studies have suggested that 15-35% of Vietnam veterans developed post-traumatic stress disorder.¹⁶ About 30-59% of people may do so after natural disasters^{17 18} and even more may do so after man made disasters.¹⁹ Weisaeth showed a clear relation between the incidence of disorder and the

degree of exposure to traumatic threat in his study of a paint factory fire: 40% of the most severely exposed developed the disorder, 25% of those with medium exposure, and 10% of those who were at a distance.²⁰ Even rescue workers may be affected.¹² Children may also suffer from post-traumatic stress disorder and may show their responses in traumatic games or dreams.²¹

The disorder is often difficult to diagnose, especially when the symptoms are not clear cut and complicated by time, experience, failure of interpersonal relationships, litigation, or the presence of other disorders such as depression, substance abuse, or personality impairment. Furthermore, people are often hesitant to talk about their condition because the reawakened experience brings distress. There is considerable evidence that post-traumatic stress disorder is poorly recognised and treated after some major disasters.^{22 23}

Treatment is notoriously difficult, especially with established disorder. There is an urgent need for effective preventive programmes. Facilitating abreaction of the experience through psychotherapy has been a cornerstone of intervention,²⁴ but other approaches have included cognitive²⁵ and behavioural techniques,¹⁶ and using drugs for associated features—for example panic disorder. In war treatment has been offered at the front, with adequate sedation and expectation of return to duty as soon as possible. It is safe to say, however, that there is a great need for systematic and controlled trials of treating post-traumatic stress disorder. Systematic attempts at prevention must also be made with those at risk, for the disability and distress associated with the disorder are substantial.

BEVERLEY RAPHAEL

Professor of Psychiatry,
University of Queensland,
Royal Brisbane Hospital,
Herston,
Queensland,
Australia 4029

WARWICK MIDDLETON

Staff Psychiatrist,
Royal Brisbane Hospital,
Herston,
Queensland,
Australia 4029

- 1 Parker A. The scars left by the flames. *Sydney Morning Herald Magazine*. 1986 June 21;34-42.
- 2 Hammond WA. *A treatise on insanity in its medical relations*. London: H K Lewis, 1883.
- 3 DaCosta JM. On irritable heart: A clinical study of a form of functional cardiac disorder and its consequences. *Am J Med Sci* 1871;61:2-53.
- 4 Freud S. Psycho-analysis and war neuroses. In: Strachey J, ed. *Collected papers Vol 5*. New York: Basic Books, 1959.
- 5 Freud S. Beyond the pleasure principle. In: *Complete psychological works, Vol 18*. London: Hogarth Press, 1953:31.
- 6 Freud S. Moses and monotheism. In: *Complete psychological works, Vol 23*. London: Hogarth Press, 1964.
- 7 Horowitz M. *Stress response syndromes*. New York: Jason Aronson, 1976.
- 8 Horowitz M, Wilner N, Alvarez W. Impact of event scale: A measure of subjective stress. *Psychosom Med* 1979;41:209-18.
- 9 American Psychiatric Association. *Diagnostic and statistical manual of mental disorders, 3rd ed*. Washington DC: American Psychiatric Association, 1987.
- 10 Brett EA, Ostroff R. Imagery and post-traumatic stress disorder: an overview. *Am J Psychiatry* 1985;142:417-24.
- 11 Breslau N, Davis GC. Post-traumatic stress disorder. The stressor criterion. *J Nerv Ment Dis* 1987;175:255-64.
- 12 Raphael B. *When disaster strikes*. New York: Basic Books, 1986.
- 13 Garb R, Kutz I, Bleich A, Solomon Z. Varieties of combat stress reaction: an immunological metaphor. *Br J Psychiatry* 1987;151:248-51.
- 14 Raphael B, Middleton W. Personal disaster. In: Howells JG, ed. *Perspectives in psychological pathology*. New York: Brunner Mazel Inc (in press).
- 15 Wilson JP, Smith WK, Johnson SK. A comparative analysis of PTSD among various survivor groups. In: Figley CR, ed. *Trauma and its wake*. New York: Brunner/Mazel, 1985:142-72.
- 16 Keane TM, Fairbank JA, Caddell JM, Zimet RT, Bender ME. A behavioural approach to assessing and treating post-traumatic stress disorder in Vietnam veterans. In: Figley CR, ed. *Trauma and its wake*. New York: Brunner/Mazel, 1985:257-94.
- 17 McFarlane AC. The longitudinal course of post-traumatic morbidity: The range of outcome and their predictors. *J Nerv Ment Dis* (In press).
- 18 Madakasira S, O'Brien K. Acute post-traumatic stress disorder in victims of a natural disaster. *J Nerv Ment Dis* 1987;175:286-90.
- 19 Tichenor JL, Kapp FT. Family and character change at Buffalo Creek. *Am J Psychiatry* 1976;133:295-9.
- 20 Weisaeth L. *The study of a factory fire*. Oslo: University of Oslo, 1983. Dissertation.

- 21 Garnezy N. Children under severe stress: Critique and commentary. *J Am Acad Child Psychiatry* 1986;25:392-4.
- 22 McFarlane AC. Psychiatric morbidity after a natural disaster: On the implications for disaster planners and ongoing services. *Med J Aust* 1986;145:11-12.
- 23 McFarlane AC. Psychiatric morbidity after a natural disaster: on the implications for disaster planners and ongoing services. *Med J Aust* 1986;145:561-3.
- 24 Fairbank JA, Nicholson RA. Theoretical and empirical issues in the treatment of post-traumatic stress disorder in Vietnam veterans. *J Clin Psychol* 1987;43:44-55.
- 25 Horowitz M. Stress response syndrome: character style, and dynamic psychotherapy. *Arch Gen Psychiatry* 1974;31:768-81.

Cyclists need helmets

Cycling has many attractions. It provides independence, needs no licence, has an extremely high mechanical efficiency, produces no pollution, and seldom causes injury to others. Injuries to riders are, however, all too common—about 300 deaths and over 5000 serious injuries in Britain annually. Pedal cycling is second only to motorcycling as the most dangerous form of travel per mile.

The Transport and Road Research Laboratory has shown that many of the cycle accidents that include a second vehicle occur at T junctions, and often the cyclist is travelling straight ahead and therefore should have priority.¹ As the report states, "In over half of these accidents the other vehicle was turning, suggesting that a significant number of these vehicles either did not see the cycle, saw it but misjudged its speed, or saw it but unreasonably expected it to give way."

Many injuries to cyclists could be prevented if other road users gave them a better opportunity to ride safely. For instance, stones and other rubbish tend to find their way into the sides of the road—but it is here that cyclists are often forced to ride by the bulk of other vehicles. The bicycle wheel is particularly vulnerable to irregular surfaces, and the rider may easily be thrown off and injured. The speed of the cycle may contribute little to the impact, but the height of the fall from the riding position is important. Injuries to the head are an important cause of death and are present in about three quarters of seriously injured cyclists. In many accidents the cycle is struck by the front of a car, and experiments show that modifying the front of cars to reduce their "aggressivity" to pedestrians also helps prevent serious injuries to cyclists.

A recent study from a large accident and emergency department provides some striking comparisons between cyclists' and motorcyclists' injuries²: the cases of 506 injured pedal cyclists and 456 injured motorcyclists were reviewed together with necropsy findings on fatal cases from the same catchment area. The study showed that pedal cyclists "were more likely to suffer a head injury than motorcyclists and that those dying suffered, on average, more severe head injuries than motorcyclists who died." Motorcyclists who died with head injuries usually suffered other major injuries, which is consistent with their higher impact velocities and the protection afforded by helmets. A study from Oxford published today (p 1161) also compared injuries to cyclists and motorcyclists and showed that head injuries were significantly more common in the cyclist. The authors also produce data to show that cycle tracks reduced the number of accidents.

The medical evidence for cyclists to wear head protection is strong. The comparative efficacy of different designs of helmet has been studied in Australia in a follow up of accidents to cycling club members: the best protection was from helmets with a hard shell and firm energy absorbing linings.³

There is now a British Standard specification for such helmets (BS 6863:1987); this tests for performance on impact, strength of strap, extent of permitted vision, and ear clearance. Many helmets now available are imported, which should be a challenge to British manufacturers to meet the British standard and add refinements such as better ventilation and a buckle that can easily be fastened with the gloved hand.

The medical case for helmets has been restated by an American doctor in family medicine.⁴ He concludes: "We have a tremendous opportunity to diminish the death toll from childhood bicycling accidents. Simple educational measures are likely to be effective. All we have to do is to act."

JOHN P BULL

MRC Research Group, Birmingham Accident Hospital, Birmingham B15 1NA

- 1 Department of Transport. *Road Accidents Great Britain*, 1983. London: HMSO, 1984;34.
- 2 Waters EA. Should pedal cyclists wear helmets? A comparison of head injuries sustained by pedal cyclists and motorcyclists in road traffic accidents. *Injury* 1986;17:372-5.
- 3 Dorsch MM, Woodward AJ, Somers RL. Do bicycle safety helmets reduce severity of head injuries in real crashes? *Accident Analysis and Prevention* 1987;19:183-90.
- 4 Weiss BD. Preventing bicycle-related head injuries. *NY State J Med* 1987;87:319-20.

What sort of "health checks" for older people?

The recent white paper *Promoting Better Health* tantalisingly suggested that the government might be prepared to encourage "health checks" for older patients within primary care.¹ "Regular and frequent health checks . . . for some elderly people" are to be discussed with relevant professions. There might even be special payments for such procedures: "The government shall, through changes in the remuneration system, encourage doctors to provide comprehensive regular care for elderly people."

Though this encouragement of prevention for older patients is welcome and long overdue, we must be quite clear what is meant by "health checks." Medical screening, especially that using multiple laboratory tests and measurements ("multiphasic screening"), has been shown by controlled trials not to reduce morbidity and mortality and not to improve use of services.^{2,3} Enthusiasm for screening for precursors of disease in older people was severely restricted by these negative results, and in the past few years it has become clear that the emphasis in prevention for this age group should be not on earlier detection of disease but rather on assessing loss of function.⁴

The intervention should aim at helping older people to avoid the adverse effects of established disabilities by planned programmes of case finding. Functional assessment should encompass physical, mental, and social function, and since the well being of many older people depends on the morale and competence of informal carers the welfare needs of the carer also need to be assessed. The broad scope of such case finding means that it is best carried out as a multi-disciplinary activity within primary care.

Many older people are fit and well, and it is wasteful for all those over 65 to be visited and subjected to a detailed assessment. Thus programmes of case finding need two stages: the first stage simply to identify those who are at high risk and likely to benefit from the second stage, the detailed assessment of functional capabilities.