hepatitis is especially low. For many years Swedish tourists visiting Mediterranean countries have been advised to obtain gammaglobulin prophylaxis before departure, and estimates suggest that half of the one million Swedish tourists are immunised in this way each year. Numerous studies[5-8] have shown the short-term value of this treatment in preventing hepatitis type A when given before exposure. It may possibly also confer some protection against type B, though hyperimmune globulin containing high titres of anti-HBs is probably better for this purpose.[9] During the 10 year period 1965-74 there were 112 cases of tourist hepatitis, about 20% being HBsAg positive. Only five of these patients had received prophylaxis. Iwarson and Stenqvist estimated that the incidence of hepatitis in tourists not given gammaglobulin was about 1 in 3000, but they did not report on the prevalence of the disease in the different countries visited or the differing standards of hygiene and sanitation encountered by tourists.

The application of new serological techniques for the detection of antibody to hepatitis type A in healthy populations[10] has shown that many people, particularly in the lower socioeconomic classes, are naturally immune and that the degree of immunity in the population rises progressively with age. Since supplies of gammaglobulin are always likely to be limited its use should ideally be reserved for people known to be without immunity; such a policy will become possible when tests for hepatitis A antibody are routinely available. For the present it does not seem justifiable to extend the indications for the prophylaxis of hepatitis type A beyond persons visiting areas such as the tropics or the Far East, where the disease is highly endemic, and those who are likely to encounter poor standards of sanitation. Until we know more of the frequency and causation of tourist hepatitis type B there is no indication for routine prophylaxis with either immune or hyperimmune globulin.

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Henoch-Schönlein purpura

Henoch-Schönlein purpura is a clearly recognisable syndrome. An acute or chronic non-thrombocytopenic purpura of characteristic distribution may be associated with polyarthritis, localised subcutaneous oedema, gastrointestinal manifestations, and acute glomerulonephritis. Both adults[1 2] and children[3] may be affected, but the condition is commoner in children, with a median age of 4 years.[3] Despite the characteristic features the incidence of this form of purpura remains unknown, as does its aetiology—though circumstantial evidence implicates hypersensitivity and allergic reactions to bacteria and viruses.[4-7]

Henoch-Schönlein purpura is essentially an acute vasculitis with perivascular accumulation of polymorphonuclear neutrophils and erythrocytes.[8 9] Immunofluorescent studies have shown variable deposition of IgA, IgG, C3, and fibrin in the glomeruli.[10 11] Deposits of IgA and complement, C3 and C4[12] or only C3,[13] have been shown simultaneously in capillary walls in cutaneous and renal biopsy specimens from these patients. Finding deposits of C3 and properdin in the absence of C1q provides evidence that the C3 in the mesangium of these patients results from activation of the alternative complement pathway.[14] The precise cause of the IgA deposits is uncertain, though it may be deposited as part of an immune complex.[14] The recent finding of circulating cryoglobulins in the acute phase supports this hypothesis.[15]

The only generally agreed approach to management of the condition is that these patients should rest in bed, be given supportive care, and avoid aspirin and phenacetin.[16] Steroids have been used, but there has been no controlled trial to assess their effect. They do not appear to have any effect on the skin lesions, the duration of the illness,[3] or on the development of renal complications,[3 6] but steroid treatment is claimed to reduce localised soft tissue swelling within 24 hours, to give relief of colicky abdominal pain within 72 hours, and even to control melaena, and it has been suggested that steroids may prevent the development of intussusception.[3]

The most serious and sometimes fatal complication of Henoch-Schönlein purpura is a severe glomerulonephritis. Haematuria and proteinuria occur in 20-70% of cases,[3 9 17 18] though progressive glomerulonephritis occurs in less than 10%.[18] At one time the prognosis was thought to be linked with the clinical features (time of onset and degree of haematuria and proteinuria) and the histological features (evidence of glomerulonephritis including epithelial crescents in Bowman's capsule). Nevertheless, long-term follow-up of children with this type of nephritis has suggested that some children in the worst prognosis group may do better than those thought to have a good prognosis. Recent studies do not support the view that adults and older children with renal lesions associated with Henoch-Schönlein purpura have a poorer prognosis than young children.[1]

Uncontrolled observations on the use of cyclophosphamide and azothiaprine in the management of nephritis complicating the illness showed no definite advantage over conservative management.[6] An MRC controlled trial showed that azothioprine and prednisolone had no effect in chronic renal failure in Henoch-Schönlein purpura.[20] Nevertheless, uncontrolled observations on the use of cytotoxic drugs in proliferative glomerulonephritis have suggested that some benefit may be derived from early chemotherapy. At present the value of cyclophosphamide in the management of this form of severe nephritis is being evaluated by the International Study of Kidney Diseases in Childhood, and we await the results with interest.

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Deafness and mental health

The deaf or hard of hearing whose impairment arises after normal speech development are a neglected group. Their needs often go unrecognised and their disability may attract social stigma rather than sympathy.[1] To a greater or less degree they may be isolated from past pleasures and present social discourse.[2 3] Difficulties in communication may result in their being bypassed or ignored even in matters of direct personal concern, so that they are made to feel stupid, inferior, or incompetent. Depression and other neurotic symptoms—particularly preoccupation with tinnitus or other adventitious noises—and various psychosomatic complaints are common sequelae,[4 5] and many authors have drawn attention to the association between deafness and the development of paranoid illness. Kraepelin[6] observed the occurrence of delusions of persecution in the hard of hearing, and more recent authors have emphasised the increased prevalence of deafness in schizophrenic and paranoid illnesses when compared with affective disorders.[7–9] Kay *et al*[10] have shown that the presence of social deafness is one of the premorbid characteristics which discriminate between patients with paranoid and with affective psychoses.

Roth and McClelland[11] contrasted patients with affective disorders with schizophrenics, who were found more often to have deafness, visual defects, or skeletal abnormalities such as dwarfism, hunchback, or amputations. They suggested that in predisposed individuals these disabilities might increase sensitivity, impede social communication and relationships, decrease self-esteem, and aggravate tendencies to withdrawal, solitariness, and paranoid distortion of reality. Misunderstanding or misinterpretation of the outside world may lead any of us to flashes of paranoid thinking, but the testing of these ideas against external reality offers constant readjustment. Where there is a failure of such reality testing an edifice of paranoid disorder may be erected upon this foundation of misunderstanding.[12] It is not difficult to understand how deafness might facilitate such a process. Indeed, it is now apparent that deafness may contribute to the development of mental disorder through its association with sensory deprivation, communication disorder, perceptual distortion, and attention deficit or as a non-specific stress.[13]

Regardless of the actual pathogenesis, then, hearing disorders which are severe, of early onset, and of long duration are important causative factors in paranoid psychoses of middle and later life. In contrast, the hearing losses associated with ageing seem to be of less importance. This suggests that the psychopathological process is one of gradual change and offers some prospect of intervention. The commonest cause of the progression from deafness to paranoia is bilateral middle ear disease originating in childhood or early adult life, and here prevention should be the aim. Chronic suppurative otitis media tends to arise from multiple attacks of acute otitis media and otorrhoea, particularly in socially disadvantaged children.[14] Education needs to be directed to early recognition and effective treatment, and more aggressive measures may be required to identify those at risk. Once deafness is established accurate assessment is essential, as is the prescription (where appropriate) of a flexible, effective, inconspicuous, and simple deaf aid. At present such aids are often cumbersome or unreliable, and the deaf tend to avoid wearing them or spend their money in search of more acceptable devices.[15]

Equally important are efforts to combat withdrawal or isolation by involvement of the deaf not only in organisations concerned specifically with the deaf, but also in more general social activities. Families and employers may need help and guidance, which can often be provided by social workers with special experience or through voluntary agencies. Assessment facilities, audiology services, and social services do, however, vary from one part of the country to another. The general practitioner has a vital part to play in ensuring that his patient receives the best service available and is enabled to remain in the real world.

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Radiation-induced breast cancer

There is no way in which a naturally occurring breast cancer can be distinguished histologically from one induced by ionising radiation. That radiation does induce breast cancer can, indeed, be suggested only by epidemiological data showing an increase in the observed incidence of the tumour in women at risk compared with that expected.

In 1965 MacKenzie[1] reported an increase of breast cancer in women who had had repeated fluoroscopic examinations during treatment by artificial pneumothorax for pulmonary tuberculosis. Myrden and Hiltz[2] continued this investigation among tuberculous women in Nova Scotia. Of 300 women fluoroscoped, 22 developed cancer of the breast compared with 4 of the 483 who had not undergone fluoroscopy. The average age of the women at the time of irradiation was 26, and the cancers developed about 17 years later. Some of the patients had had more than 500 fluoroscopies; the total dose of radiation to the breast varied from 50 rads to 6000 rads delivered over some weeks to years. In a separate type of study Wanebo *et al*[3] noted an excess of breast cancer in women who had survived an