psychotherapy work?" is meaningless. The question must be more specific. Are delinquent boys who have group therapy and individual psychotherapy while in prison less likely to offend again and more likely to stay in a job than boys matched for age, background, offence, and as many other variables as possible, who do not have therapy?[13] Or, are university students with "maladaptive anxiety" likely to respond better with treatment by desensitization than to insight-orientated psychotherapy, and does the difference persist at follow-up two years later?[14] In both these cases the answer seems to be Yes. Can psychotherapy sometimes be harmful too, as clinicians who try to piece together the bits of the partly analysed have long suspected?

If psychotherapeutic techniques for specified groups of patients with specified complaints can be shown to work—and a great deal more research is needed—how could help best be given to those who could benefit? One, expensive, possibility is the Wings' estimate that 480 more psychiatrists should be recruited; another, often suggested, is that general practitioners should be encouraged to attend training courses to acquire special skills (though the supply of interested doctors is not unlimited); a third, favoured in the U.S.A., is that the field should be opened to psychologists and trained counsellors, who would not necessarily work under a medical umbrella. This last suggestion is not so heterodox as it may seem. Already a great deal of "psychotherapy" is practised by priests in their pastoral work, by probation officers, and by welfare workers of many kinds. A fourth solution, an effort to shift the emphasis from the psychiatric symptoms and to deal with the social problems that are so often associated, is already the subject of a pilot study.[15]

It looks, then, as if the answer could be a compromise between two alternatives. The recruitment of the extra psychiatrists needed would at present be prohibitively expensive, even if enough doctors could be attracted into the specialty. At the other extreme, and certainly until the worth of psychotherapy has been established beyond doubt, spreading the load might prove the best solution.

Herpesvirus Infection in Burned Patients

Viruses are not the first things which come to mind when the conversation turns to infected burns. Most of these infections are bacterial and hence respond to one antibiotic or another. However, a series of six cases recently published from the U.S. Army Institute for Surgical Research[1] shows that viral infections cannot be entirely ignored.

There is theoretically no reason why at least some viruses should not infect burns. Indeed, these areas of exposed cells are like large tissue cultures inviting infection by viruses, and it is hardly surprising that herpes simplex, with its predilection for the skin, should have been found in these circumstances. What is not clear is whether the infection came from outside (like the unpublished case of Scott, referred to in this paper, of the mother who infected a child's burn by "kissing it better") or whether, as seems more likely, the patients were carriers of herpes, and the infection was lit up by trauma to the skin. An additional factor, perhaps the critical one, is the possibility of an alteration in the immune mechanism of the body caused by the burns, and such disturbances have been reported.[2]

Of the six patients, all of whom were very severely burnt, three died (two of them adult males and one a child of 5). The three survivors were all young adult males. The most interesting feature of the pathology of the fatal cases is that in two of them the liver and adrenals were affected. Herpetic infection of the liver is usually confined to the neonatal period, when it occurs as part of a primary widespread infection with the virus. Herpesvirus hepatitis in the adult is exceedingly rare, and it has been supposed to result from a depressed immunity allowing virus already present to break out. Two cases have been reported recently. One was herpetic hepatitis in a pregnant woman, who recovered.[3] The other was a fatal illness in an elderly man who had had bronchial asthma for many years, and who died of generalized herpes infection arising from a stomatitis.[4] This was considered to be the rare instance of a primary infection in an old patient.

It is not easy to provide pointers to the diagnosis of herpes infection for the guidance of those caring for the badly burned. The occurrence of fever without an obvious bacterial infection as its cause should arouse suspicion, but in some of these cases there was a combined bacterial and viral infection. The local manifestations of the infection are described as being multiple, small, erosive lesions on the healing areas. One of the most important diagnostic features, if it is present, is the occurrence of the vesicular lesions of herpes on the unburnt areas of the skin—though in a seriously injured patient these could easily be mistaken for minor abrasions. In the recently reported series[1] diagnosis was made in the first patient by electron-microscopy of thin sections of the margin of the skin lesion. This showed in the cells the characteristic herpesvirus particles and also Cowdry type A intranuclear inclusions. The virus (*Herpesvirus hominis*) was isolated at necropsy from skin and liver. The other two fatal cases were also diagnosed by histopathology and electron-microscopy, but virus was not isolated. In the three survivors diagnosis was made by biopsy and electron-microscopy of thin sections and in two of them also by rising antibody titres in the blood, particularly of neutralizing antibody.

It is clear from this report, firstly, that medical staff treating extensively burned patients should be on the look-out for infections with viruses, particularly herpesvirus, as a rare but not impossible complication. Secondly, it is interesting that thin sections of biopsy material were used for electron-microscopy but that the technique of negative staining was applied neither to the fluid from the burns nor to the vesicular lesions on the unburnt areas of skin, though it is a procedure which can be helpful in reaching a rapid diagnosis. Again, it would have been interesting to have had more isolations of virus, so as to know whether this was a type 1 or type 2 herpes simplex. A third point concerns treatment. If a diagnosis can be made, is it feasible to treat these patients with iodo-deoxy-uridine or with pooled gammaglobulin, which can be assumed to contain antibodies? Patients with affected liver and adrenals are unlikely to benefit from topical treatment with iodo-deoxy-uridine, and systemic administration of this

[1] Foley, F. D., Greenawald, K. A., Nash, G., and Pruitt, B. A., jun., *New England Journal of Medicine*, 1970, **282**, 652.
[2] Arturson, G., Högman, C. F., Johansson, S. G. O., and Killander, J., *Lancet*, 1969, **1**, 546.
[3] Flewett, T. H., Parker, R. G. F., and Philip, W. M., *Journal of Clinical Pathology*, 1969, **22**, 60.
[4] Diderholm, H., Stenram, U., Tegner, K. B., and Willén, R., *Acta Medica Scandinavica*, 1969, **186**, 151.

drug is still a heroic measure. The administration of specific antibody might even be harmful.

This report underlines once again how ill equipped we still are to treat viral infections. It also reminds us that there is as much clinical virology within hospital walls as there is outside them. Furthermore, as any clinical virologist in a general hospital could confirm, there seems to be at least as much for virologists to do in the surgical wards as in the medical ones.

Neurological Complications of Infective Endocarditis

In 1885 William Osler described the features of malignant endocarditis, stating that "a considerable number of cases come under observation . . . for the first time with symptoms of cerebral or even cerebro-spinal trouble."[1] Recent surveys on endocarditis[2-5] have emphasized the changed characteristics of this disease in the antibiotic era—for example, the frequency with which late perforation of the heart valves is now seen—but neurological complications have received little mention. Nevertheless, three recent studies have now reawakened interest in this important aspect of the disease. Reviewing 116 patients seen at the Radcliffe Infirmary, Oxford from 1955 to 1965, M. J. G. Harrison and J. R. Hampton[7] found that 37 had neurological manifestations and in 33 of the 37 these were present at the time of admission and formed the immediate diagnostic problem. Royden Jones and his colleagues[6] surveyed 385 patients seen at the Mayo Clinic from 1950 to 1964; no fewer than 110 of these patients had neurological involvement, and in 60% of them this was either the chief complaint or one of the major presenting symptoms. Finally, in a comprehensive review[8] I. Ziment has emphasized that the incidence of neurological complications has remained unchanged since the introduction of antibiotics. Failure to recognize the underlying condition, moreover, often leads to a fatal outcome, these patients having a significantly higher mortality rate than those without neurological manifestations.

The neurological complications of infective endocarditis may be divided into four main groups. Firstly, the cerebrovascular features, caused by infarction or haemorrhage, are the commonest, presenting with signs that depend on the location of the lesion. The infarction is presumed to be due to an embolus, while haemorrhage results from rupture of a mycotic aneurysm into either the brain substance or the subarachnoid space. This type of aneurysm is more peripherally situated than the congenital berry one, and this may suggest the diagnosis at angiography if it has been previously missed. M. R. Roach and C. G. Drake,[9] reviewing 191 cases of ruptured intracranial aneurysms, found five cases of mycotic aneurysms, in none of which had the correct diagnosis of infective endocarditis been made before rupture. In young patients especially it is important to consider that endocarditis may be the cause of even apparently straightforward strokes.

The second group comprises toxic encephalopathy, and few reports have emphasized the importance of considering infective endocarditis in its differential diagnosis. Fluctuating confusion, delirium, hallucinations, depression, confabulation, a feeling of unreality, or organic psychoses are features that may lead to admission to a psychiatric unit. The diagnostic problems may be particularly difficult in the elderly.[10]

Thirdly, acute meningitis may be another presenting feature of infective endocarditis, but the cerebrospinal fluid is often sterile with a predominantly polymorphonuclear leucocytosis. The combination of meningitis and heart murmur should make the doctor suspect infective endocarditis and if he fails to make the correct diagnosis the true state of affairs may be learnt only at necropsy.

Lastly, miscellaneous presentations include those patients seen with severe headaches that clear with antibiotic treatment; generalized convulsions, which are occasionally focal; acute mononeuropathy; chorea; pseudobulbar palsy; or a multiplicity of symptoms and signs suggesting demyelinating disease or even hysteria.

Today many of the classical features of infective endocarditis are no longer common, and, with the ageing population, the declining incidence of rheumatic fever, and the increase in cardiac surgery, the spectrum of the disease has changed. Nevertheless, the incidence of neurological complications in this disease remains high, and possibly may even be increasing. Unless the disease is diligently sought for, there will be a commensurate increase in the mortality.

Treatment of Whooping-cough

In the catarrhal stage of whooping-cough, before there is any spasmodic cough or whoop, the mucosa of the upper respiratory tract shows early signs of inflammation. Thin mucus covers the epithelium, some of the epithelial cells show early necrotic changes, and clumps of *Bordetella pertussis*, the organism which causes these changes, can be seen enmeshed in the cilia. If an antimicrobial agent could reach and destroy the organisms at this stage, the inflammatory reaction in the mucosa would probably be brought to an end.

Later in the disease, when the child is coughing and whooping, the damage spreads to the lower respiratory tract and pierces deeply into lung tissue. The basilar layers of the epithelium are necrotic and in places detached from the basement membrane, and there is much peribronchial and peribronchiolar infiltration with lymphocytes and polymorphonuclears, which spreads widely into the lung and produces areas of collapsed, sodden tissue. Here and there are patches of emphysema, where air is trapped behind bronchioles, which are designed for the free flow of air but instead are blocked with viscid mucopus. *Bordetella pertussis* can settle down and flourish in these conditions, which differ so much from the early superficial changes, and it is unlikely that even if an antimicrobial agent could reach and kill the organism the serious damage which it has already caused deep in the respiratory

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[2] Lerner, P. I., and Weinstein, L., *New England Journal of Medicine*, 1966, **274**, 199.
[3] *British Medical Journal*, 1969, **2**, 5.
[4] *Lancet*, 1967, **1**, 605.
[5] Morgan, W. L., and Bland, E. F., *Circulation*, 1959, **19**, 753.
[6] Jones, H. R., Jun., Siekert, R. G., and Geraci, J. E., *Annals of Internal Medicine*, 1969, **71**, 21.
[7] Harrison, M. J. G., and Hampton, J. R., *British Medical Journal*, 1967, **2**, 148.
[8] Ziment, I., *American Journal of Medicine*, 1969, **47**, 593.
[9] Roach, M. R., and Drake, C. G., *New England Journal of Medicine*, 1965, **273**, 240.
[10] Anderson, J. H., and Staffurth, J. S., *Lancet*, 1955, **2**, 1055.
[11] Carpenter, R. R., and Petersdorf, R. G., *American Journal of Medicine*, 1962, **33**, 262.