different times.⁸⁻¹⁰ For example, when the transit time through the whole gut was measured five times in as many weeks in healthy women it fluctuated in some of them from under two days to over five days. There was even more variation in the size of individual stools—in one person from a mere 7 g on one day to 330 g on another.⁸ It would be difficult to show that patients with the irritable bowel syndrome fluctuated more than that.

Some patients with spastic colon complain predominantly of diarrhoea, some predominantly of constipation. It should at least be possible to show that these patients differ from each other. Surprisingly, one group of workers has reported no difference in mean stool weight or transit time, but they did not give their data and they collected stools for only 48 hours.¹⁰ A recent study in Sheffield was more thorough and tells a different story.¹¹ In women whose predominant bowel complaint was diarrhoea the average weight of stool passed in 24 hours was 128 g, whereas in those with predominant constipation it was only 61 g, and there was an appropriate difference in whole gut transit time (35 versus 87 hours). Nevertheless, the women with "diarrhoea" passed no more stool than did a control group of normal women, in whom it averaged 112 g a day. So why did they complain? Possibly they were bothered by urgency (due perhaps to the soft consistency of their stools) but urgency has been little studied and is poorly understood. In the Sheffield study the men with predominant diarrhoea had bulkier stools than their healthy controls (230 versus 166 g a day), but even this does not prove that their stools were abnormal. British vegetarians normally pass stools weighing 225 g a day and in India and Africa healthy people regularly pass stools of 300 g to 500 g a day.¹² In Sheffield, like the women, the men claimed that most of their stools were unformed and perhaps this is what bothered them. But were the stools of the patients with the irritable bowel syndrome pathologically loose or were the patients pathologically intolerant of normal variation in stool consistency?

A novel finding in this study was that transit through the small bowel was fast in the patients with spastic colon and predominant diarrhoea and slow in the constipated group. The average time taken for the head of a test meal to reach the caecum (as judged by a sudden rise in breath content of hydrogen) was 4.2 hours in healthy controls, 3.3 hours in the group with diarrhoea, and 5.4 hours in the group with constipation. This was interpreted as evidence that the irritable bowel syndrome affected the small bowel—a reasonable conclusion, but one that does not necessarily imply that the small bowel was functioning abnormally. Indeed, the asymptomatic controls had very variable small bowel transit times (1.5-7.4 hours) and all the patients with the irritable bowel syndrome fell within this range. So it remains true that physiological measurements have not yet shown a definite abnormality of bowel function in patients with the irritable bowel syndrome except perhaps in those with constipation.

The pain of the irritable bowel syndrome seems to be due either to abnormally strong contraction (spasm) of the intestinal smooth muscle or to undue sensitivity of the intestine to distension. Until recently this oversensitivity had been shown only in the rectum and sigmoid,^{6,13} but now the Sheffield group has evidence that the caecum or ascending colon may be oversensitive too. Two thirds of their patients had pain after a test meal, and in three quarters of these the onset of pain coincided with the arrival of the meal residues in the caecum. Why should the caecum hurt when it is given its normal job of work to do? That is just one among many

questions about the irritable bowel syndrome which are waiting for an answer.

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Postviral neurological syndromes

Many different neurological and psychiatric syndromes follow viral infections, but their clinical picture and pathogenesis are poorly understood. They form a heterogeneous group including well known entities such as acute disseminated encephalomyelitis and the Guillain-Barré syndrome; disorders in which the virus probably causes enzyme abnormalities—for example, Reye's syndrome and epidemic myalgic encephalomyelitis; and diseases which are not primarily caused by viruses but are occasionally precipitated or made worse by a viral infection—for example, the genetic disorder Refsum's disease (polyneuropathy, ichthyosis, retinitis pigmentosa, and deafness)¹ or multiple sclerosis.² Some idea of incidence is given by the fact that Reye's syndrome is the second most common cause of death in childhood due to diseases associated with viral infection affecting the central nervous system,³ and that acute disseminated encephalomyelitis accounts for one third of all the encephalitides.⁴

Acute disseminated encephalomyelitis is, indeed, the most common demyelinating disease in the world because of the frequency with which it follows measles and other viral infections. Its clinical presentations range from seizures and coma to isolated neurological defects. The neurological findings may, however, be minimal in patients with florid psychiatric manifestations. The Guillain-Barré syndrome is precipitated by similar viruses and its clinical course is analogous to that of acute disseminated encephalomyelitis. Both disorders are likely to be vasculopathies mediated by immune complexes with the lesions occurring in central nervous tissue myelin in acute disseminated encephalomyelitis

and in peripheral nerve myelin in the Guillain-Barré syndrome.⁵

Reye's syndrome⁶ usually follows varicella, influenza, or Coxsackie virus infection, but other viruses and agents have also been implicated. It presents with repeated attacks of vomiting, rapidly followed by delirium, seizures, and coma. There is diffuse cerebral oedema without any evidence of inflammation, and the liver and other organs show micro-vesicular fatty change. On electron microscopy, neurones and hepatocytes show distinctive mitochondrial changes. Epidemic myalgic encephalomyelitis, or postviral fatigue state, tends to occur after the same viral infections as Reye's syndrome. It has been recognised in sporadic cases or in epidemics for over 50 years. Most patients have increased IgG antibodies to Coxsackie viruses and some with chronic symptoms and signs show increases in specific IgM antibodies.⁷ Electrophysiological analyses of single muscle fibres in our own laboratory, and nuclear magnetic resonance studies of muscle in collaboration with Dr Radda at Oxford, have recently shown distinctive abnormalities. Furthermore, studies of lymphocyte subsets in the peripheral blood have shown increased numbers of suppressor cytotoxic lymphocytes and decreased numbers of helper cells.⁸ The relation in epidemic myalgic encephalomyelitis between immune deficiency, persistent infection by virus, and a possible enzyme defect is under investigation.

Many other neurological disorders, some inherited and some acquired, may be made worse by a viral infection. These include Refsum's disease,¹ hereditary mononeuritis multiplex,⁹ neuralgic amyotrophy,¹⁰ the hereditary ataxias,¹¹ and multiple sclerosis.² Some patients may develop sensitisation to neural antigens before overt clinical disease develops,^{12 13} and the viral infection may act through mechanisms which are related to the Shwartzman reaction.^{14 15}

The postviral syndromes have stimulated many theories to account for their features, the most important being an alteration in the regulation of the immune system. Viruses may induce immunosuppression in several ways: they may persist and multiply in lymphocytes—for example, measles can replicate in T cells and Epstein-Barre virus in both T and B cells; they may activate suppressor lymphocytes; or they may induce the release of suppressor factors, including interferon. They may also be associated with host reactions against self antigens and the development of autoimmune disease. Older explanations for this association centred on fusion of the virus with the cell membrane, release of sequestered antigen, or viral mimicry of host determinants. There is some experimental support for the suggestion that the virus envelope may incorporate host antigen and that this combination may activate B lymphocytes to produce autoantibodies.¹⁶ Possibly viruses may combine with histocompatibility antigens,¹⁷ leading to recognition by T cells and an immune reaction to host tissue. Immune complexes of virus and antibody, especially when formed in situ, are a known cause of tissue damage. Finally, viruses to which the patient is already sensitised may act as the precipitating agent in a Shwartzman type reaction.¹⁵

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Principles for the future of the special hospitals

Nowhere in its new report on the future of the special hospitals¹ does the Royal College of Psychiatrists' special committee tell us what it thinks should be these hospitals' philosophy, but principles can be sifted out. One explicit statement of principle that the report does make is that we will continue to need special hospitals, which is just as well as contracts have recently been signed for the rebuilding of Broadmoor.

The first principle should be that as few people as possible are detained in the special hospitals and that those who are detained should be in for as short a time as possible. Although the special hospitals are not nearly as unpleasant as people imagine, to have been a special hospital patient is the worst stigma that our society has to offer, and nobody should be in a special hospital unless he or she needs to be there. The psychiatrists who work in the special hospitals know that many of their patients do not need to be there, but they cannot know for sure who those patients are. Those responsible for discharges are between the devil and the deep blue sea: currently they are being pilloried for keeping patients who do not need to be there, but just as often they are criticised for releasing patients who then commit violent offences. Clearly continuing research is needed to refine the criteria for both admission and discharge.

What is sure is that "one in eight patients in the special hospital are waiting to go, their discharge or transfer having been agreed," and that one in three of these patients have been waiting to be transferred for over two years. The report insists that this is a problem that requires "priority attention . . . not only from the Department of Health and Social Security but from psychiatrists, nursing staff and their trade unions, and