We need to know more about the natural history of scoliosis, however: we do not even know how many people with obvious deformity are performing quite satisfactorily in society.

Thus screening as currently performed uncovers vast numbers of inconsequential spinal asymmetries and a few patients with benign scoliosis of late onset. Moreover, it is ethically questionable to detect a cosmetic deformity of which the patient is usually unaware, to inform the patient that the problem stems from a spinal curvature of which the natural history is virtually unknown, and then to prescribe a "standard treatment" which is generally ineffective. On the rare occasions when screening in adolescence picks up a patient with untreated infantile progressive scoliosis the deformity may be so severe as to have caused irreversible organic damage already.

What then should be done? Firstly, we need to learn more about the epidemiological features of idiopathic scoliosis throughout life, and where screening is performed it should be directed to this end.³⁷⁻⁴⁰ It would not unduly tax existing arrangements, at least in Britain, to include an examination of the back during first immunisation at 3½ months and at the school medical examination at age 5 years to exclude the sinister scoliosis of early onset, for which prompt action is essential.⁴¹ Meanwhile, clinical research must be fostered in relation to the three dimensional nature of the deformity to produce more effective corrections for those who present later with a deformity.

Fortunately, there is one ray of real hope from apparent changes in natural history. The infantile progressive form has become much less common. When the condition was first described progressive curves were more prevalent than resolving ones.⁴² The next 30 years saw a dramatic reversal,⁴³ and now (possibly because babies are kept prone in their cots) the incidence of both varieties has considerably diminished.⁴⁴ A similar trend is seen in scoliosis of late onset, and where most screening has been performed the need for treatment has been less obvious, suggesting an even more benign course.⁴⁵

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Acute self limited colitis

Changes are present in most rectal biopsy specimens taken from patients with acute diarrhoea due to the known common infectious agents—campylobacter, salmonella, and shigella.^{1,2} Many patients have bloody diarrhoea with tenesmus and tenderness over the colon, providing clinical as well as histological evidence of "colitis." Most patients with acute transient diarrhoea have negative stool cultures, but the histological appearances seen in rectal biopsy specimens from these patients are similar to those from patients with positive cultures.^{1,3} The terms "acute infective type" or "transient" colitis have been introduced to describe the disorder in which cultures are negative and "acute self limited colitis" to encompass both the conditions in which cultures are positive and those in which they are negative.^{3,5} In time, currently unrecognised bacterial or viral pathogens may well be found to account for most of the group in which cultures are negative, but acute self limited colitis—as defined by some authors—also includes ischaemic, antibiotic associated, and pseudomembranous colitis.⁵

Ulcerative colitis of acute onset or even Crohn's disease—classed together as idiopathic colitis—may be indistinguishable clinically from an acute self limited colitis. Conversely, severe salmonella colitis may mimic idiopathic inflammatory bowel disease closely, even to the extent of causing toxic dilatation of the colon. The histological features of acute infective type colitis or acute self limited colitis are therefore of considerable interest, as they may be crucial in differentiating between this condition and an early case of ulcerative colitis or Crohn's disease.

Mandal and coworkers described the histological features which they found of value in distinguishing what they called

"transient colitis" from active ulcerative colitis and Crohn's disease.⁴ Their experience was based on 238 patients admitted to an infectious diseases unit in Manchester, all of whom underwent biopsy soon after admission. The patients included 53 with proved acute infection, 42 with a similar clinical picture but negative stool cultures ("transient colitis"), and 65 with ulcerative colitis or Crohn's disease. It was not possible on histological grounds to distinguish the specimens from patients with different types of infective colitis from those with culture negative colitis, the histological features being identical with those described in greater detail by the same authors in salmonella colitis.¹ There was mucosal oedema but preservation of the architecture of the crypts, little or no mucus depletion in goblet cells, and focal collection of polymorphonuclear leucocytes in the mucosa and submucosa. True crypt abscesses were seen but were often more localised than in ulcerative colitis. In both ulcerative colitis and Crohn's disease mononuclear cells were predominant, and in ulcerative colitis depletion of mucus or distortion of the crypt architecture was usually striking. Even so, in a few patients with colitis of the infective type the histological changes could not be distinguished from those of ulcerative colitis. Similar conclusions were reached in the study of Kumar, Nostrant, and Appelman, but they emphasised the crypt distortion and basal plasmacytosis unique to ulcerative colitis.³

A recent histological study carried out in Seattle comparing biopsy specimens from 44 patients with acute self limited colitis with those taken from 104 with ulcerative colitis and Crohn's disease is of interest for two reasons: not only were all the biopsy specimens evaluated initially with no clinical information but 44 histopathological features were defined and their presence or absence recorded in each specimen.⁵ The predictive probability of self limited colitis or chronic idiopathic inflammatory bowel disease being the true diagnosis was subsequently calculated for each of the 37 histopathological features which could be recognised reproducibly by different observers. These authors confirmed that rectal biopsy may be of great value in the early diagnosis of patients with acute diarrhoea, though the admitted weakness of their study was that most of the patients with idiopathic inflammatory bowel disease had had symptoms for several weeks before biopsy was undertaken, whereas specimens from patients with acute self limited colitis were obtained within days of the onset.

Seven histological features favoured the diagnosis of idiopathic inflammatory bowel disease rather than acute self limited colitis: distorted crypt architecture, increased cellularity of the lamina propria with both round cells and neutrophils, a villous surface (due to separation of crypts by oedema and inflammatory cells), epithelioid granuloma, crypt atrophy, basal lymphoid aggregates, and one or more basally located giant cells. Each of these features carried a predictive probability of idiopathic inflammatory bowel disease of between 87% and 100%, and one or more was present in 83 of the 104 patients with that diagnosis who had biopsy specimens taken. Distorted crypt architecture and an increase in both round cells and neutrophils were particularly valuable signs as they never occurred in acute self limited colitis. Features such as normal crypt architecture, superficial giant cells, and superficial crypt abscesses favoured acute self limited disease, but in general this diagnosis was better made by the absence of features specific for idiopathic inflammatory bowel disease. The Seattle study also confirmed suggestions that "cystic" crypt abscesses reported in salmonella¹ or campylobacter⁶ infections are often morpho-

logically different from the typical crypt abscesses of idiopathic inflammatory bowel disease, but as they may occur in acute ulcerative colitis they may merely represent an early version of the same phenomenon.

This objective validation of histological criteria that help the differentiation of transient, infective type, or acute self limited colitis from idiopathic inflammatory bowel disease should encourage pathologists to try to give more help to their clinical colleagues.⁵ The main advantage of differentiating acute self limited colitis from ulcerative colitis and Crohn's disease is that patients will not be labelled wrongly as having a chronic disorder and given unnecessary and prolonged treatment with sulphasalazine. The features seen in the biopsy specimen alone may be adequate for firm diagnosis if the characteristics of idiopathic inflammatory bowel disease are present, but the clinician will still have to remain prepared for those patients—far from rare—who have both this and a superimposed infection.^{1,7} Continued observation and repeat biopsies will always resolve the true diagnosis in due course,² but meanwhile the occasional very sick patient (for example, with invasive salmonella infection) may have to be treated with both antibiotics and corticosteroids.

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Surgical waiting lists

Concern to improve provision of surgical services within the National Health Service has encouraged the growth of publications on waiting lists, their interpretation, and their management. With so much advice on a chronic and apparently unremitting problem the cynic might be forgiven for calling to mind the clinical dictum which suggests that the greater the number of remedies for a particular condition the less the likelihood that any is really effective. Waiting lists show the characteristic pattern of a rich tradition of folk remedies combined with insufficient understanding of pathogenesis, and publications which give further insight are to be welcomed. Among recent papers of this type are official reports from multidisciplinary working groups, offering informed advice on waiting list management^{1,2}; papers by sympathetic management scientists, largely directed towards non-clinical readers, urging circumspection in interpreting information on waiting lists^{3,5}; and useful guidance on the statistical material available for monitoring and reviewing waiting lists.^{4,7}

The increasing use of management sciences in the NHS has led to growing appreciation that waiting lists lengthen because of disturbances in a delicate balance of factors. The importance of physical resources (hospital beds and