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Screening for scoliosis

Teenagers are still being seen with idiopathic scoliosis, bent double at presentation and with such severe respiratory dysfunction as to preclude surgery[1]—apparently lending powerful support to those who have enthusiastically introduced school screening programmes to detect such cases at an earlier stage.[2-5] Such distressing tragedies evoke powerful emotions, but is the argument so simple, and is screening a suitable solution?

The cause of idiopathic scoliosis is unknown, but the pathogenesis of the three dimensional deformity has been established.[6-11] The typical idiopathic deformity, a lateral curvature with rotation, is secondary to a primary deformity in the sagittal plane. There is lordosis in the thoracic region, where kyphosis should exist, and the deformity is the opposite of Scheuermann's kyphosis.[10] A lordosis is rotationally unstable as its axis lies posterior to the vertebral bodies. Therefore, all scolioses with rotation are lordoscolioses and not kyphoscolioses.

Recognition of the presence of the lordosis is crucial to treatment. Just as Scheuermann's kyphosis needs extension to improve it—and this is most effective, as the deformity is rotationally stable[12]—so the idiopathic lordosis might appear to need flexion. Unfortunately, on flexion the lordosis rotates to the side to produce the scoliosis (just as a piece of paper twists on being flexed towards one of its edges).[8] The deformity cannot, therefore, be treated conservatively.[13] What braces do primarily is to prevent flexion, though some temporary improvement may occur if reduction of the lumber lordosis below produces thoracic extension above.[10] Even if bracing were effective it would have to be continued until spinal maturity,[14-16] which occurs 10 years after general skeletal maturity.[17 18] Curves in the spine tend to get worse until this time, but an unusual degree of compliance would be required for a 10 year old to wear a brace for 15 years.[19]

If, then, conservative treatment is of little value is surgical treatment much better? The crucial factor is the potential for progression of the deformity. The traditional operation —posterior Harrington instrumentation and fusion—is addressed to the secondary scoliotic deformity. At best the only correction it gives is around 50% of the component in the coronal plane; it has no effect on the rotational prominence with which every patient presents.[20-23] From the patient's viewpoint a lot of surgery seems to produce little gain. Moreover, the underlying lordosis implies that the back of the spine is too short, and a posterior fusion may add insult to injury. Thus posterior surgery may indeed prevent progression in patients with the more benign, late onset variety of scoliosis, but for those with early onset with much more growth ahead quite the opposite may happen—surgery may facilitate accelerated progression.[8 10] With bigger curves the outlook is even more gloomy. Preoperative traction of any kind has no corrective effect,[24 25] and the surgical attack needs to be more aggressive.[26 27] On the brighter side, newer forms of instrumentation are more promising,[28 29] provided that attention is directed to the sagittal plane.[10]

The key to untreated scoliosis is the age of onset. The high morbidity and mortality rates from cardiopulmonary compromise quoted as strong arguments by screeners[30] are relevant only to patients with an early onset, when chest deformity may be present at the time the pulmonary parenchyma is developing.[31] Late onset deformities, by contrast, even if they become severe, are not associated with any substantial reduction in chest function,[30 32 33]—though patients with this type of scoliosis may fare less well socially[34] and psychologically.[35]

In practice, while the serious organic problems tend to occur in patients with early onset disease most screening has been done in adolescence. The data are difficult to interpret because of differences in terminology.[36] Most screening has relied on a quick visual examination, when about 15% of adolescents show evidence of asymmetry of the trunk.[37] When the surface profile of the back is measured the rate of asymmetry rises to about 25%.[38] When adolescents are *x* rayed almost all may be shown to have a measurable scoliosis, but in half this is secondary to a tilted pelvis.[37] About 2% of children screened have a curve measuring 10° or more, and 0·2-0·5% have a curve measuring 20° or more.[2-4 37] The few longitudinal studies show that scoliosis due to pelvic tilt has no potential for progression, and that while 10% of the remainder may deteriorate twice as many improve—and the rest stay the same.[2 4 37 39]

True idiopathic scoliosis is, then, probably made up from the small fraction that progress. Girls with right thoracic curves show the greatest progression potential.[2 4 37]

The benign course of most patients with scoliosis of late onset has obscured the real efficacy of conservative treatment: a brace that obliterates the lumbar lordosis and prevents flexion ought to check progression if it was known that the curve would result in an unacceptable deformity.[10]

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