Bone and Joint Diseases

Backache

JOHN A MATHEWS

British Medical Journal, 1977, **1**, 432-434

In recent years there has been a crescendo of interest in the symptom of "low back pain" among epidemiologists, pathologists, radiologists, rheumatologists, and orthopaedic surgeons. Much research is directed at delineating the actual mechanisms by which much common back pain is produced as a preliminary to more efficient prophylaxis, diagnosis, and treatment. The symptom has many causes, some of which are obviously mechanical and benign and others more serious. Often the nature of the causative lesion is clear but in many others it is open to debate.

Undoubtedly the problem is a huge one. Certified incapacity from back troubles in 1969-70 was 627 days in men and 347 days in women per 1000 insured and accounted for 32·9% and 21·4% respectively of all rheumatic complaints. Although this must be a gross underestimate of the total national morbidity, it represents over three times the amount of work lost through strikes. I will concentrate on describing mechanical forms of backache in the adult emphasising those minor forms in which the aetiology and mechanisms are particularly controversial and indicating points of use in differential diagnosis. The rationale of treatment will also be described.

"Mechanical" backache

The clinician should suspect a mechanical problem when pain, which is far the commonest feature, is related to posture, minor trauma, or injudicious use. Such pain is often episodic and intermittent, aggravated by those actions that would be expected to demand more from a supporting structure and relieved by rest and recumbency. The importance of this history must be emphasised as it weighs heavily against chronic inflammatory spinal diseases, such as ankylosing spondylitis or tuberculosis, and neoplastic conditions. A useful approach is to divide the symptoms and signs of mechanical back lesions into "articular," "dural," and "neurological" (fig 1).

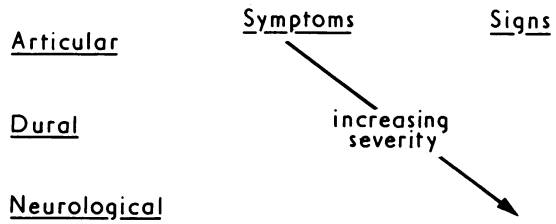

FIG 1—Symptoms and signs of mechanical back lesions. (Reproduced by permission of the editor of *Rheumatology and Rehabilitation*.)

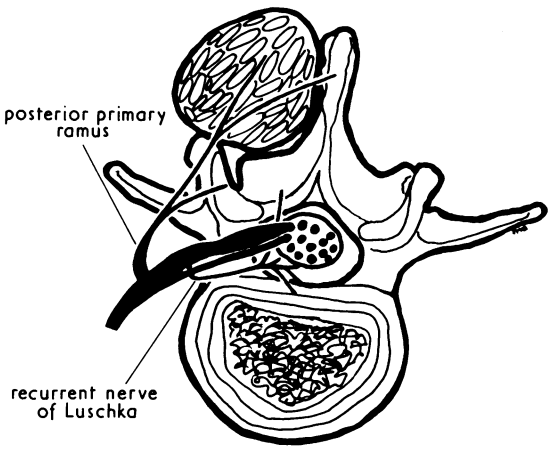

FIG 2—Innervation of intraspinal structures. Reproduced from Taylor (1977), *Recent Advances in Surgery*, No. 9. Edinburgh, Churchill Livingstone.

ARTICULAR

Symptoms only

The innervation of the spine (fig 2) suggests that similar pain may arise from several structures—such as joint, muscle, or ligament. Most spinal pain, however, probably originates from joint disorders. In the absence of objective signs differential diagnosis remains confusing and controversial. In general muscle lesions are characterised by swelling and bruising, both of which are rare in the spine. Muscle tenderness is often referred and misleading as a locating sign, and the best criterion is pain that is maximal on resisted or "isometric" contraction. Primary muscle lesions are rare.

Ligamentous lesions are usually diagnosed by the presence of localised tenderness with pain aggravated by compression or stretching. By these rather imprecise criteria ligamentous lesions are a not infrequent accompaniment of spinal pain. Perhaps the best clue to an articular source of a pain is discomfort that is maximal at the end of a range of movement.

Symptoms with signs

By analogy with more accessible joints elsewhere significant derangement of a spinal intervertebral joint would be expected to produce intermittent "locking"—regardless of whether one believes the usual culprit to be a posterior apophyseal joint or the anterior disc. The lesion is generally asymmetrical, and the accompanying sign

Department of Rheumatology, St Thomas's Hospital, London SE1 7EH

JOHN A MATHEWS, MB, MRCP, consultant physician

is asymmetrical restriction of movement. In severe cases the spine may be held deviated from its normal posture. The controversy concerning the relative responsibility of the apophyseal joint or the disc for spinal derangements is not solved, and as the two are normally linked solidly by bone they can therefore act only as a single unit. All the spinal structures mentioned so far, including disc and apophyseal joints, are innervated either by the posterior primary ramus or the recurrent nerve of Luschka. Pain reference is fairly central and similar so that differential diagnosis depends upon interpretation of symptoms and physical signs. As neither the nucleus of a disc nor the hyaline cartilage of an apophyseal joint has a nerve supply, minor degenerative changes of both may occur without producing pain.

When generalised joint disorders such as ankylosing spondylitis or osteoarthrosis affect the spine the restriction of movement is symmetrical, lateral flexions being reduced particularly early. When an internal derangement occurs the lesion, and therefore the restriction of movement, is asymmetrical. Thus, of the four standard movements of the lumbar spine (flexion, extension, right and left lateral flexion), some, but not all, will be restricted. Disc and apophyseal joint derangement often occur together, and it is not often possible to differentiate the two.

DURAL

Symptoms only

Among the various intraspinal structures innervated by the sinuvertebral nerve (fig 2) are the posterior longitudinal ligament and the adjacent anterior aspect of the dural sac as well as the dural investment of nerve root sheaths. These structures may be impinged on anteriorly by a protruding intervertebral disc, or posteriorly by a swollen apophyseal joint (fig 3). Again it seems unlikely that the actual causative structure can be differentiated by the site or nature of the pain, but a dural pain may be rather less central than an articular pain, although it is also ill-defined. Dural pain may be referred via longitudinal anastomoses to segments other than that of its origin. This might explain the observation that such pain can be felt radiating up to the thoracic spine, down to the sacrum, or round to the abdominal wall or groin.

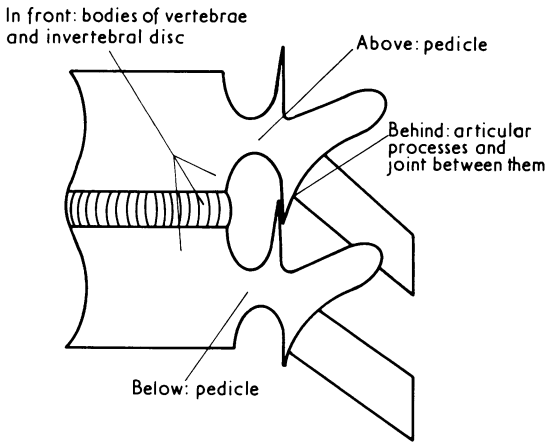

FIG 3—Swollen apophyseal joint. (Reproduced by permission of Dr W Hewitt, Department of Anatomy, St Thomas's Hospital and the editor of *Physiotherapy*.)

Symptoms with signs

Once again the addition of signs clarifies the interpretation of symptoms. Two dural tension signs exist in the lumbar region. The straight leg raise (Lasègue) test is performed with the patient supine, by lifting the leg with an extended knee until painful reflex hamstring spasm prevents further elevation. The spasm is induced by the pressure of the L 5 or S 1 nerve root on the offending structure. The unaffected side should be tested for comparison, and the limiting factor will be merely hamstring length.

Less well known is the femoral nerve stretch test performed by flexing the knee of the prone patient. This manoeuvre tenses the L 3 and L 4 roots and is positive if the patient's pain (rather than vague thigh ache) is reproduced. Even with these signs the apophyseal joint could be responsible for the symptoms.

NEUROLOGICAL

Symptoms only

At this stage the likelihood that a disc prolapse is causative is much greater. The discs most commonly involved are L 5-S 1 and L4-L 5 affecting the S 1 and L 5 nerve roots. Thus the pain is more severe, better circumscribed, and much more distal than articular or dural pain. In addition it can be exquisitely sensitive to alteration in intraspinal pressure so that it is increased by straining.

Less often the L 4 or L 3 roots are affected, and as one ascends the lumbar roots the possibility of alternative non-mechanical causes increases. Root pain may be severe and prostrating but is then usually shortlived to be followed by numbness and weakness; alternatively it may be chronic and relatively mild, even needing some provoking factor to bring it to light. Occasionally the symptoms is numbness or paraesthesiae.

Cauda equina claudication is a variation on the more usual symptom of posture or stress-related pain. The term is used when pain of nerve root distribution is produced on exertion thus leading to possible confusion with limb muscle ischaemia. The actual mechanism of production of the pain is not clear. It seems that either the nerves called into use become ischaemic or the nerve pressure increases with walking. In either case the condition is particularly likely to occur in patients with congenital or acquired stenosis of the spinal canal.

Sacroiliitis is another important differential diagnosis from sciatic root pain. Pain is usually felt in the low back or buttock but occasionally radiates to the thigh or calf. Furthermore it may alternate from side to side—the prerogative of an inflammatory disease rather than a mechanical fault. Immobility stiffness may provide an important clue, and the diagnosis may be confirmed by clinical stress-testing of the sacroiliac joint or later by radiological changes.

Symptoms with signs

The most common neurological deficit due to lumbar disc prolapse is impairment of a tendon reflex. Motor and sensory abnormalities also occur. As S 1 is a common level for the lesion the ankle reflex may be lost. L 5 has no reliable tendon reflex, but an L 4 lesion may lead to diminution of the knee jerk. Lower sacral lesions may lead to loss of bladder and bowel control and the only abnormal neurological sign be sensory loss in the saddle area.

Differential diagnosis and investigations

Laboratory investigations are useful screening procedures. The erythocyte sedimentation rate and alkaline phosphatase are expected to be normal with mechanical disorders but abnormalities help draw attention to inflammatory or metabolic bone disease. Radiographs are virtually uninfluenced by acute mechanical disorders, although long-standing disc degeneration may lead to disc-space narrowing, osteophyte formation, and osteoarthrosis of the apophyseal joints. An important aspect is their help in excluding any destructive pathological processes, although such changes often lag behind history and signs.

Treatment

Recumbency and analgesics remain the basic treatment for most mechanical spinal lesions. When advising a period of recumbent bedrest the patient should be horizontal but in the least uncomfortable position. Occasionally the stress of bedpan usage may make use of a bed-side commode an advantage. Other methods of treatment are least controversial in the more severe conditions, and these will be mentioned first.

SURGICAL TREATMENT

Spinal decompression, laminectomy, or disc removal are strongly indicated when there is serious neurological deficit—that is, when a patient arrives who already has multiradicular root lesions or bladder trouble. Equally, progression of neurological signs despite complete bedrest demands surgical inter-

vention. A lesser indication is persistent pain or recurrent incapacitating episodes.

Chemonucleolysis—This is an alternative when the lesser indications for surgery exist. It is claimed that a proteolytic enzyme injection into the disc may provoke removal of disc material and lessening of pressure effects.

MEDICAL TREATMENT

The unlikelihood of persuading a soft-tissue lesion to heal more rapidly than by rest alone suggests that all active medical treatments are really directed at restoring anatomy and consequent pain relief. Epidural injections of local anaesthetic and corticosteroid are recommended for patients with neurological symptoms or signs. Analgesia is produced, and it is claimed that the recovery time is lessened. Undoubtedly the zenith of pain occurring during root interruption is dramatically lessened.

Traction—This may hasten the recovery of patients with sciatica. The efficacy of treatment depends on the demonstrable possibility of exerting a force sufficient to distract vertebrae, create a negative pressure in a disc, and thus reduce the extent of its protrusion. It also depends upon the notion that some of the resulting improvement will be maintained.

Manipulation—Rotatory or gliding movements are claimed to help patients with articular symptoms and signs and even dural tension signs. The rationale is that the force applied to a relatively intact annular part of a disc may coax a bulge back into shape by a centripetal compression effect exerted through the obliquely radiating fibres. The improvements in the disc contour has been shown radiologically, and it is suggested that some of the improvement might be sustained. In lesser degrees of the syndrome possibly an apophyseal derangement is being corrected.

Sclerosant injections—When ligamentous pain and tenderness seem to dominate, "sclerosant" mixtures of phenol, dextrose, and glycerine may be injected into the tender area. It is claimed that these injections taughten ligaments but the evidence for increased stability is poor. An alternative explanation of pain relief might be an effect of phenol on sensory innervation.

Rhizolysis—By subcutaneous incisions, injections, or cautery rhizolysis is claimed to be effective in "deafferenting" a structure whose mechanical lesion cannot be lastingly corrected. Definite evidence of benefit, however, is difficult to obtain.

Prophylaxis

Undoubtedly much spinal pain is related to posture and the resulting elevation of the intradisc pressure. Advice on maintaining low disc pressure falls into three categories:

Posture—Bending and lifting produces maximum pressure followed by sitting with a bent back; standing straight, and lying down produce the lowest. The high-pressure postures must be avoided.

Lumbosacral belt—This helps in two ways. If correctly fitted it maintains a slight lordosis (avoids flexion) and thus a lower pressure. In addition it may help posture by increasing the intra-abdominal pressure.

Exercises—Any back movement is likely to increase pressure and thus should be avoided. It is claimed, however, that isometric (resisted) posterior spinal and abdominal wall muscle exercises may increase their strength and thus achieve the same two benefits as a lumbosacral belt.

This rather mechanistic approach to the problems of backache does not discuss the many practical problems of referred pain and depressive illness. It is designed to provide a rationale for diagnosis and active treatment of many common back lesions.

Letter from . . . Canada

The Quebec election

PETER J BANKS

British Medical Journal, 1977, **1**, 434-435

Quebec, to separate or not to separate?

This month nobody in Canada is talking about anything else. Superficially the problem is simple. Quebec has elected René Levesque and his Parti Quebecois, and part of his platform was to take Quebec out of Canada. He has pledged to hold a referendum on the separation issue within two years. At present the polls say that few Quebeckers favour separation, but within the two years this could change.

So far the rest of Canada is reacting parochially, province by province. Newfoundland is worrying about the fate of Labrador. The Maritimes are concerned with their electricity supply and their lines of communication. Ontario is pompously adopting a strictly "no-panic in our house" stance. The left-wing prairie provinces see the whole Quebec election as a triumph of the Quebec working class against the Liberal elite. British Columbia and Alberta say it's all Ottawa's fault anyway and think that it will help to get more power into the hands of the provinces.

Nobody is talking about that great big beautiful polyglot country Canada. The French were here first. Deserted by the fleshy myopic Bourbons, they developed their own society. Defeated by the Anglos, soon themselves to split into Americans and British North Americans, the French withdrew from the Mississippi Valley and became Lower Canada—Catholic, rural, French-speaking, and different. Waves of immigration put them into a minority of 5 to 1 and the Quebec birthrate, in spite of vaunted expectation, began to fall. A sense of isolation in a sea of Anglophonic North Americans set in, helped by some economic disadvantage at the hands of English Canada.

The Quiet Revolution took place weakening the authoritarian domination of the church and a growing sense of frustration found expression in books, plays, universities, and the French media. Recognising this, Lester Pearson coined the dictum, "All Canadians must be able to communicate with the organs

Victoria, British Columbia
PETER J BANKS, MD, FRCP, consulting physician