THE LUMBAR FACET ARTHROSIS SYNDROME

CLINICAL PRESENTATION AND ARTICULAR SURFACE CHANGES

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We describe a lumbar facet syndrome in which disabling symptoms are associated with normal or near-normal plain radiographs. Local spinal fusion relieved symptoms in 12 patients; the excised facet joint surfaces showed some of the histological changes seen in chondromalacia patellae and in osteoarthritis of other large joints.

The most frequent change was focal full-thickness cartilage necrosis or loss of cartilage with exposure of subchondral bone, but osteophyte formation was remarkably absent in all specimens. We suggest that there are both clinical and histological similarities between the facet arthrosis syndrome and chondromalacia patellae. Facet arthrosis may be a relatively important cause of intractable back pain in young and middle-aged adults.

The designation "non-specific low-back pain" implies failure to establish the pathological changes in many of the patients who present with disabling low back pain and normal or near-normal radiographs. We suggest that, among these patients, there are at least two syndromes, each with a recognisable pattern of pain; these are the "facet arthrosis syndrome" and the "instability syndrome". Our investigation aimed to provide evidence that pathological changes in the articular cartilage of the lumbar facet joints may be related to the facet arthrosis syndrome.

PAIN SYNDROMES

Facet arthrosis syndrome. In this syndrome pain is aggravated by rest in any posture, including recumbency, and is relieved by repeated or continuous gentle movement. Rising in the morning is difficult because of pain and stiffness, which ease as physical activity increases. When rest is unavoidable, pain is commonly reduced by a position of lumbar flexion. Backward bending is restricted by pain; forward bending is usually of normal range and character, with little or no pain. We have called these patients the "uppers" because of their need to be up and about or constantly altering posture in order to reduce their pain.

This type of pain is recognisably similar to that seen in degenerative or inflammatory arthritis in other synovial joints, including the hip, knee and those of the hand. Ankylosing spondylitis in its early stages provides one clinical model for this syndrome; in a young adult with known disease of the spinal synovial joints, the presentation may be much as described above.

Lumbar instability. In this syndrome, by contrast, the patients are "downers", whose pain is relieved by rest and recumbency and increases throughout the day. Forward bending is restricted by pain and characterised by swaying or jerking movements. This description fits those patients who have had some sprain or strain of spinal soft tissues, the result of unrecognised or long-forgotten minor injuries. This clinical pattern may provide a better definition of lumbar instability than the more objective definitions which have failed in practical clinical application (Nachemson 1985). One clinical model for this syndrome is symptomatic spondylolysis, in which a similar pattern of symptoms and signs result from the ununited fracture.

The differentiation of these two patterns is important and helpful in both conservative treatment and pre-operative investigation, but elements of both patterns may be found in one patient. The "combination" syndrome must be recognised and not allowed to cause confusion. It reflects the fact that an intervertebral segment may fail in more than one of its parts at the same time. Both pain patterns may be associated with some referred pain in the lower limbs, but this can readily be

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distinguished from the major disabling pain produced by nerve root compression.

We have investigated 12 patients with the facet arthrosis syndrome in an attempt to relate this to pathological changes.

PATIENTS AND METHODS

Of a very large number of patients seen for low back pain, 12 patients with characteristic facet arthrosis syndrome and significant disability were fully investigated. Nine of the patients had had symptoms for an average of 15 months before referral, and three for 12 to 20 years. All had failed to respond to conservative treatment given for an average of four months after referral. Four patients had some lower limb pain but of a lesser degree than their low back pain.

There were 11 women and one man with ages ranging from 24 to 60 years. The average age was 40; only one patient was under 30 and one over 50. Pain and tenderness were localised to the general area of the lumbosacral junction in all cases. Two patients had had previous spinal operations: one an L5 laminectomy and one a lumbosacral discectomy.

Investigations. Plain radiographs of the lumbar spine were helpful only in excluding other causes of backache. In four patients they were normal, in three there was a detectable decrease in the joint space of the lumbosacral facet joints. Mild reduction of intervertebral disc height was seen at one or more levels in six patients, and one patient had a lumbar scoliosis.

Osteophytosis of the lumbar facet joints, commonly associated with lumbar "spondylosis", was not seen.

Computerised tomography failed to show any additional pathological change in the facet joints and showed no other segmental sources of pain. More specific localisation of the cause of symptoms was achieved by facet arthrography or by diligent palpation for points of maximum tenderness followed by radiographs with skin markers. Arthrography was considered to be positive only when the injection reproduced some or all of the usual symptoms, and when some relief was provided by subsequent infiltration with lignocaine. Several of the patients had negative lumbar myelography and discography in the search for other causes of their pain.

Operation. All 12 patients had posterolateral and intertransverse fusion operations. Both L4–5 and L5–S1 were fused in seven patients, L5–S1 alone in four patients, while fusion from L2 to the sacrum with Harrington instrumentation was required for a 32-year-old woman with progressive scoliosis and intractable facet pain at the lumbosacral junction.

During the operations the facet joints were excised and preserved for histological examination. Sections were cut perpendicular to the plane of the joint and stained with either haematoxylin and eosin or toluidine blue. The facet joint capsules had necessarily been damaged or destroyed and could not be studied histologically.

To provide some control material without too much postmortem change the low lumbar facet joints were excised from four fresh cadavers whose kidneys were being taken for transplantation. Death had occurred at ages ranging from 17 to 48 years and the specimens were examined in the same way as those of the patients. It could not be established whether or not these subjects had suffered backache.

RESULTS

Facet joints. There was some evidence of early damage to articular cartilage in the facet joints of all 12 patients. The most frequent finding was a focus of full-thickness cartilage necrosis, but we also saw ulceration, fibrillation and eburnation (Figs 1 to 4). We suspect that the cartilage "ulcer" is the result of sloughing of a plug of necrotic cartilage.

Chondrocyte clusters, foci of fibrocartilage (Fig. 5) and increased perichondrocyte metachromasia provided evidence of repair. The only noteworthy change in the subchondral bone was early subchondral cyst formation (Fig. 2). No specific part of the facet surface appeared to be particularly involved and, strikingly, there was no osteophyte formation in any specimen.

The common feature of all specimens was the exposure of subchondral bone, sometimes in an ulcer, or else potentially present in an area of full-thickness cartilage necrosis.

The control specimens were completely normal in three subjects aged 17, 17 and 26 years, but in a 48-year-old man killed in a motor vehicle accident there was surface fibrillation of the articular cartilage and minor peripheral osteophytosis in all lumbar facet joints, without evidence of any focal cartilage necrosis.

Clinical results. One patient required revision of his fusion for pseudarthrosis, but all patients achieved gratifying pain relief in an average of 3.5 months after operation.

DISCUSSION

Many of the histological changes which we found have been described in classic and standard texts as those of osteoarthritis or arthritis deformans (Ayers 1935; Leubner 1936; Oppenheimer 1938; Badgley 1941; Putti and Logròscino 1952; Lewin 1964; Schmorl and Junghanns 1971; Vernon-Roberts 1980). All these studies are anatomical descriptions only and therefore cannot relate the abnormalities to the causes of low back pain. Ayers (1935) describes what is probably the first examination of a lumbar facet joint excised at operation, but the histology suggests inflammation rather than degeneration.

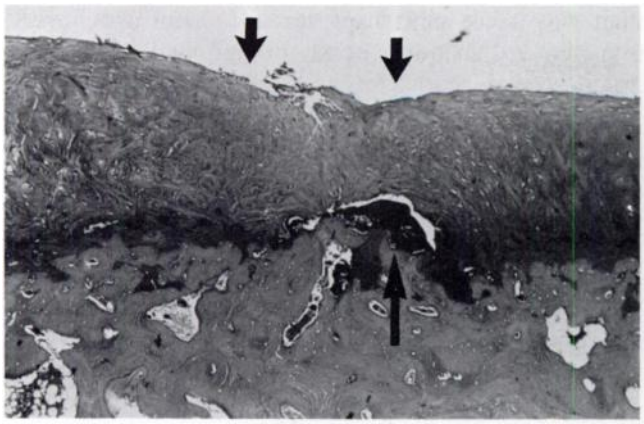


Fig. 1

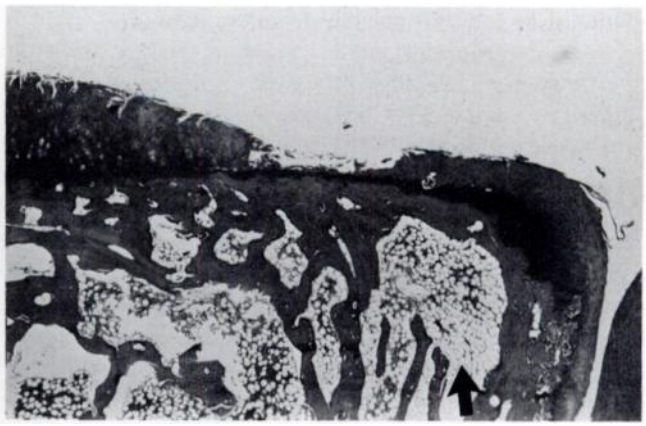


Fig. 2

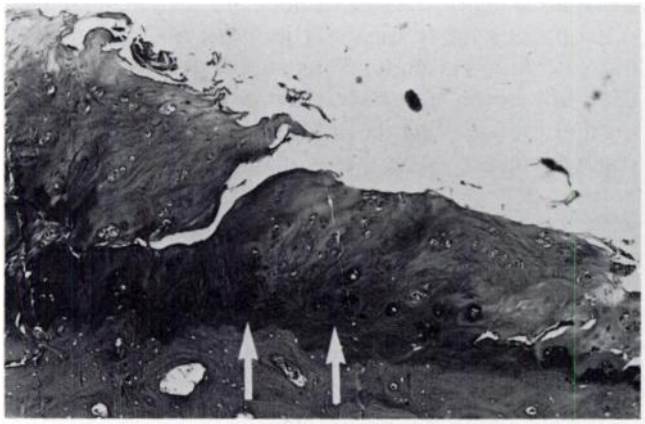


Fig. 3

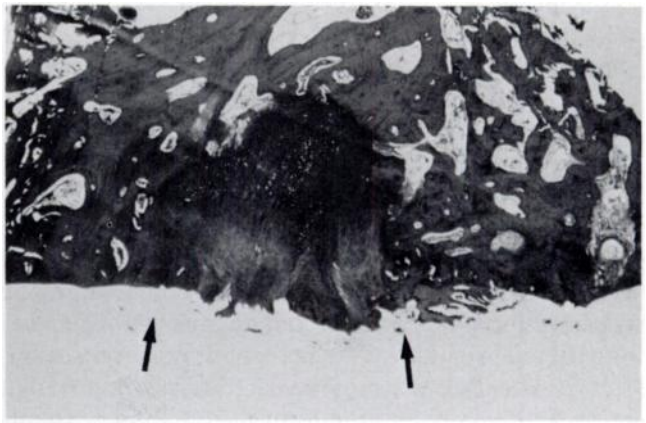
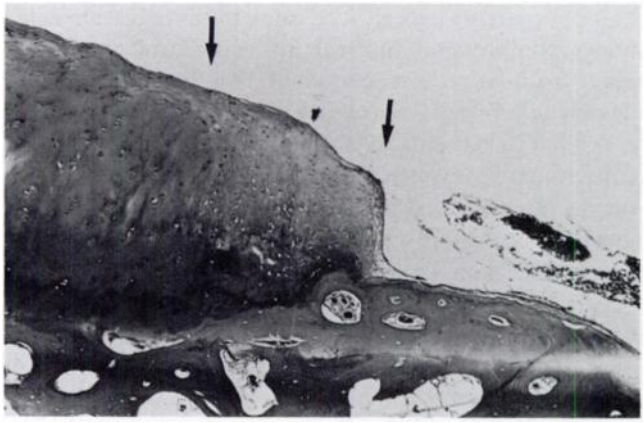


Fig. 4



Histological sections of facet joints excised from patients with facet arthrosis syndrome. Figure 1 – Full-thickness cartilage necrosis, between the short arrows. This shows lighter staining and no viable chondrocytes. There is some separation at the cartilage–bone junction (long arrow) and the space is filled with exudate (toluidine blue, × 7). Figure 2 – An articular cartilage ulcer which exposes bone. This is presumed to represent a stage beyond the "necrosis-in-situ" in Figure 1. An early cyst in subchondral bone is arrowed (toluidine blue, × 3). Figure 3 – A fibrillation cleft with adjacent cartilage necrosis down to bone. Chondrocyte clusters are arrowed (toluidine blue × 12). Figure 4 – To show grooved eburnation exposing subchondral bone. A fibrocartilaginous plug (between arrows) fills a cyst (toluidine blue × 3). Figure 5 – Full-thickness fibrocartilage (between arrows) at the edge of an ulcer which exposes subchondral bone (toluidine blue, × 12).

Fig. 5

These studies do, however, all emphasise osteophytosis or bony spurring as an important feature of the pathology of osteoarthritis. A finding which does not appear to have been described previously is the full-thickness "necrosis-in-situ" shown in Figure 1, but this is not associated with osteophytes and resembles the "intermediate stage destruction" which Meachim (1980) reported in his study of excised femoral heads, and the "basal degeneration" described by Goodfellow, Hungerford and Woods (1976) in chondromalacia patellae.

The atrophic features we found in the articular cartilage of our relatively young patients raises the question which is currently exercising the minds of those engaged in the study of chondromalacia patellae (Goodfellow et al. 1976; Insall 1982; Bentley and Dowd 1984; Bentley 1985): whether this is merely a stage in the course of "classic" spondylotic osteoarthritis (Outerbridge 1961) or a peculiarly symptomatic variant of it. There are similarities between the facet arthrosis syndrome and chondromalacia patellae. These similarities are found not only in the histological changes of full-thickness cartilage necrosis, separation of cartilage from bone, chondrocyte clusters and metachromasia (Goodfellow et al. 1976) but also in the clinical presentation; in both conditions relatively young patients may present with severe disability from pain, associated with local

tenderness and normal plain radiographs.

As in chondromalacia patellae, the relationship between the histology and the symptoms in facet arthrosis is not clear. Clinicians may feel intuitively that the degree of pain in both conditions is disproportionate to the physical changes which can be demonstrated. We have proposed the term "chondromalacia facetae", if only to confer a degree of respectability upon those patients who do not qualify for a diagnosis of "spinal arthritis" and are sometimes unjustly classified as psychologically suspect. Fifty years ago Hugo Leubner (1936) appealed to colleagues to consider a diagnosis of "early arthritis deformans" in patients presenting with low back pain but normal radiographs. We suggest that this appeal is now supported by a link between symptoms and pathology. We also suspect that a similar syndrome may present in the thoracic spine, that it can be distinguished from myofascial pain and that it may similarly require spinal arthrodesis if other treatment fails.

The causes for the articular changes we have described are obscure, but no less so than those conjectured for chondromalacia patellae, which include variations of normal biomechanics, trauma and genetic predisposition. It is possible that asymmetric angulation of left and right facet joints could produce stresses sufficient to cause early articular cartilage injury, but asymmetry was not a prominent feature in our patients and yet is so common (Badgley 1941) that it may be considered to be a variation of normal anatomy. Putti (1927) originally described these anomalies of facet angulation as a possible cause of nerve root compression and sciatic pain rather than low back pain. Loss of height of an intervertebral disc can be expected to produce increased pressure on the facet joint surfaces posterior to it (Dunlop, Adams and Hutton 1984; Yang and King 1984) but in most of our patients the disc spaces were of normal height or only slightly reduced. We found little change in the subchondral bone of the facet joints, certainly nothing like the patellar osteoporosis described by Darracott and Vernon-Roberts (1971).

The obvious argument against an attempt to relate minor changes in articular surfaces to major pain symptoms is that these changes are probably almost universal in middle-aged adults yet few have disabling lumbar pain. The purpose of our limited study of cadaver material was to attempt to discover if the described articular changes were indeed universal. The results so far are unsatisfactory; most of the few renal transplant donors available for study are young adults and no accurate history of spinal pain is available. We have to fall back on the findings of Putti and Logròscino (1952) that subjects under 30 years of age had normal joints and the vast majority of those under 40 had only mild arthritic changes.

The mechanism whereby these pathological changes may produce pain is not known. The concept that increased joint pressure is transmitted to pain-sensitive subchondral bone through foci of necrotic cartilage, as described for the patella by Goodfellow et al. (1976), is plausible. Any attempt to explain major pain by relatively minor changes confined to articular cartilage is confronted by the fact, well known to clinicians in this field, that many patients present with minor symptoms in the presence of advanced joint destruction, sclerosis and osteophytosis. Explanations for this opposite situation are also conjectural. It is possible that widespread loss of cartilage allows a relatively even diffusion of joint pressure into the subchondral bone, producing less pain than that resulting from high concentrations of pressure acting through small areas of cartilage loss.

"Facet syndromes" have been described previously, but with different features on each occasion. Ghormley (1933) pioneered the association of low back pain with radiographic evidence of advanced degenerative changes in the facet joints. He did not distinguish between arthritis and instability, but ventured to suggest, with some diffidence, that spinal arthrodesis produced symptomatic relief. Mooney and Robertson (1976) also failed to make this distinction but made a major contribution by describing joint injection for the identification of symptomatic facet joints and for treatment of pain.

Our patients most closely resemble the "responders" to local infiltration described by Fairbank (1981) except that our patients experienced more pain with their joints under compression (lumbar spine in extension), and relief with joint surfaces separated (lumbar spine in flexion).

For a patient disabled by pain refractory to conservative measures who is facing operation for spinal fusion, diagnosis of the responsible segmental level or levels is crucial. Computerised tomography offers no more than plain radiographs, unless there is advanced degeneration (Carrera et al. 1980). Facet arthrography, while invasive and painful, remains the best pre-operative investigation by virtue of the provocation of pain in the affected joints (Park and McCall, personal communication 1976; Fairbank et al. 1981). The arthrographic abnormalities described by Dory (1981) are of secondary importance but may provide useful confirmation of a positive pain response.

CONCLUSIONS

We have described links between a clinical syndrome of low back pain, localisation of the source of pain in facet joints, histological abnormalities in the excised joint surfaces and clinical relief obtained through fusion of the affected segments.

The causes for the facet joint abnormalities remain unknown and the association between these articular surface changes and pain has not been proved; it remains a matter of conjecture.

At this stage, it is impossible to be dogmatic as to whether the facet syndrome we have described ("chondromalacia facetae") is a distinct non-osteophytic arthrosis, or merely a stage (possibly reversible) in the progression of age-related hypertrophic osteoarthritis. It is important that the condition should be recognised so that patients who are disabled by the syndrome may receive appropriate treatment rather than be considered neurotic.

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REFERENCES

Ayres CE. Further case studies of lumbo-sacral pathology with consideration of involvement of intervertebral discs and articular facets. *N Engl J Med* 1935;213:716–21.

Badgley CE. The articular facets in relation to low-back pain and sciatic radiation. *J Bone Joint Surg* 1941;23:481–96.

Bentley G, Dowd G. Current concepts of etiology and treatment of chondromalacia patellae. *Clin Orthop* 1984;189:209–28.

Bentley G. Articular cartilage changes in chondromalacia patellae. *J Bone Joint Surg [Br]* 1985;67-B:769–74.

Carrera GF, Haughton VM, Syvertsen A, Williams AL. Computed tomography of the lumbar facet joints. *Radiology* 1980;134:145–8.

Darracott J, Vernon-Roberts B. The bony changes in "chondromalacia patellae". *Rheumatol Phys Med* 1971;11:175–9.

Dory MA. Arthrography of the lumbar facet joints. *Radiology* 1981;140:23–7.

Dunlop RB, Adams MA, Hutton WC. Disc space narrowing and the lumbar facet joints. *J Bone Joint Surg [Br]* 1984;66-B:706–10.

Fairbank JC, The anatomical sources of low back pain with particular reference to the intervertebral apophyseal joints. Thesis, University of Cambridge, 1981.

Fairbank JC, Park WM, McCall IW, O'Brien JP. Apophyseal injection of local anaesthetic as a diagnostic aid in primary low-back pain syndromes. *Spine* 1981;6:598–605.

Ghormley RK. Low back pain, with special reference to articular facet, with presentation of an operative procedure. *JAMA* 1933;101:1773–7.

Goodfellow J, Hungerford DS, Woods C. Patello-femoral joint mechanics and pathology. 2. Chondromalacia patellae. *J Bone Joint Surg [Br]* 1976;58-B:291–9.

Insall J. Current concepts review. Patellar pain. *J Bone Joint Surg [Am]* 1982;64-A:147–52.

Leubner H. Die Arthritis deformans der kleinen Wirbelgelenke. *Z Orthop* 1936;65:42–52.

Lewin T. Osteoarthritis in lumbar synovial joints: a morphological study. *Acta Orthop Scand* 1964;Suppl 73.

Meachim G. Ways of cartilage breakdown in human and experimental osteoarthrosis. In: Nuki G, ed. *The aetiopathogenesis of osteoarthrosis.* Tunbridge Wells: Pitman Medical, 1980:16–28.

Mooney V, Robertson J. The facet syndrome. *Clin Orthop* 1976;115:149–56.

Oppenheimer A. Diseases of the apophyseal (intervertebral) articulations. *J Bone Joint Surg* 1938;20:285–313.

Outerbridge RE. The etiology of chomdromalacia patellae. *J Bone Joint Surg [Br]* 1961;43-B:752–7.

Putti V. Lady Jones Lecture. On new conceptions in the pathogenesis of sciatic pain. *Lancet* 1927;2:53–60.

Putti V, Logròscino D. Anatomia dell'artritismo vertebrale apofisario. In: Putti V, ed. *Scritti medici.*Vol II. Bologna: Edizioni Scientifiche Instituto Rizzoli, 1952:53.

Schmorl G, Junghanns H. *The human spine in health and disease.* 5th German edition by Junghanns H. Translated and edited by Besemann EF. New York etc: Grune & Stratton, 1971.

Vernon-Roberts B. The pathology and interrelation of intervertebral disc lesions, osteoarthrosis of the apophyseal joints, lumbar spondylosis and low back pain. In: Jayson MIV, ed. *The lumbar spine and back pain.* 2nd ed. Tunbridge Wells etc: Pitman Medical, 1980:83–114.

Yang KH, King AI. Mechanism of facet load transmission as a hypothesis for low-back pain. *Spine* 1984;9:557–65.