Degenerative lumbar spondylolisthesis with an intact neural arch (pseudospondylolisthesis)

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Twenty patients treated for degenerative spondylolisthesis with an intact neural arch principally at the L4-5 interspace had neural compression caused by dislocation of the vertebral bodies and intrusions of lamina and enlarged, arthrotic facets into a stenotic spinal canal. The resulting "pincer" effect caused complete or partial block demonstrable on myelography, with nerve root and cauda equina compression. Most of the patients were women aged 45 to 84 years. Seven had neurogenic claudication. The majority had unrestricted straight-leg raising, and no signs of acute neural entrapment were seen as in patients with a herniated disc. Absent ankle reflexes, and weakness and atrophy of the anterior tibial muscle group were common, while sensation was relatively undisturbed. Treatment consisted of liberal laminar decompression including foraminotomy and medial or total facetectomy. Good-to-excellent results were obtained, and no patient was made worse by the procedure.

KEY WORDS: spondylolisthesis · nerve root · cauda equina · compression · intervertebral disc

Spondylolisthesis, originally described by Kilian in 1854 as a subluxation of the lumbosacral joint, was attributed to the superimposed body weight slowly displacing the last lumbar vertebra forward. It was recognized as a source of obstruction in labor. In 1855 Robert demonstrated by anatomical dissection that slippage could not occur if the neural arch was intact. Discontinuity in the pars interarticularis was demonstrated by Lambl in 1858. Hartmann, in 1865, observed that in certain circumstances the spinous process was not displaced when the vertebral body moved ventrally. At that time, it was not fully apparent that two distinct pathological conditions existed, namely, spondylolisthesis with and without a defect in the pars interarticularis.

The clinical significance of spondylolisthesis with an intact neural arch was given prominence by the reports of Junghanns in 1931, and McNab in 1950. They were the first to emphasize female dominance in this condition as compared to male dominance in congenital spondylolisthesis with a defect in the neural arch. Degenerative disc changes
preceded the development of severe apophyseal arthrosis which ultimately was followed by subluxation with the entire vertebra moving forward as a unit. Initial symptoms of back pain progressed to eventual nerve root and cauda equina compression.

Junghanns first suggested the term “pseudospondylolisthesis” to differentiate patients with an intact neural arch from those with a congenital defect in the neural arch. McNab preferred the term “spondylolisthesis with an intact neural arch” as a more accurate description of the pathology, since vertebral sliding is correctly called spondylolisthesis whether or not it is associated with a neural arch defect. Yet even this term is somewhat inadequate, since spondylolisthesis with an intact neural arch may occur in conditions other than degenerative disc disease, such as agenesis of the articular facets.

Wiltse and Hutchinson indicate that spondylolisthesis may follow fracture or dislocation of the lumbar spine due to trauma as well as stress fracture through the pars interarticularis. Diseases such as tuberculosis, cancer, or Charcot’s disease may also cause vertebral displacement. Newman and Stone suggested the name “degenerative spondylolisthesis” as more descriptive of the pathology in the present context. To be inclusive, the term, “degenerative spondylolisthesis with an intact neural arch” appears to be most appropriate.

Twenty such patients with nerve root and cauda equina compression are presented with a discussion of diagnostic methods and the results of treatment by decompressive laminectomy, foraminotomy, and facetectomy. Three have been discussed in an earlier presentation on lumbar nerve root compression caused by osteoarthritis of the posterior facets.

Summary of Cases

Between 1965 and 1974, we treated 20 patients with signs of nerve root or cauda equina compression caused by degenerative spondylolisthesis with partial or complete block of the spinal canal. Of these, three were men and 17 women. They ranged from 45 to 84 years of age. Five patients were between 45 and 46, eight between 64 and 69, and seven between 74 and 84 years of age. Trauma was a possible cause in two patients. Back pain had been present in all patients, appearing from 1½ to 20 years before the onset of significant radicular pain and neurological changes. Evidence of nerve root or cauda equina pathology had been present for less than 1 year in most patients. Pain was severe during walking, and in a few it was increased by coughing and straining. Neurogenic claudication was present in seven patients, with both pain and weakness increasing after standing and walking, and relieved only by sitting or lying down. Subjective sensory changes were noted in nine. Weakness was most evident distally, especially the extensor hallucis longus and peroneal muscle groups. In five patients with bilateral symptoms there was delay in urination and episodic incontinence. Only one had numbness over the anagenital area. In 14 patients, pain was bilateral with one side always dominant. Remissions were rare once radicular pain occurred. Acute tilt of the spine was noted twice. A small step-like defect could be palpated at the level of the spinous processes of L4–5 in six patients.

Neurological changes included absent or reduced ankle reflexes in 17 patients. In only three were reflexes intact. Patellar reflexes varied from 1 to 2+. Objective sensory changes were present over the L-5 dermatome in six patients, and in the anagenital areas in two. Atrophy of the pretibial muscle group was present in all, and most advanced in individuals with longstanding symptoms. In only five was the Lasègue sign positive. There was no evidence of peripheral vascular disease. The clinical findings strongly suggested a motor neuropathy, primarily at the L4–5 level.

Plain x-ray examination of the spine disclosed involvement of the L4–5 interspace in 14 patients. The L3–4 interspace was involved in four, and the L2–3 and L5–S1 levels in one each. Anterolisthesis of no more than 1 cm was present in all cases. All had associated spurring of the vertebral margins, narrowing of the affected interspaces, and severe arthrotic changes in the posterior facets. Relative stenosis of the spinal canal accounted for the frequency of complete block on myelography in these patients. Myelography disclosed evidence of a complete block in 11 patients, and partial to nearly complete block in nine.

Laboratory studies were unremarkable.
Protein content in the spinal fluid was rarely above 50 mg%.

In 15 patients operation consisted of laminectomy of both laminae of the vertebrae involved, followed by medial facetectomy and liberal foraminotomy. Neurolysis was also necessary since the red, inflamed nerve roots often adhered to the soft tissue structures in the lateral recesses and beneath the arthrotic facets. The nerve roots were wrapped in Gel-foam. In five patients, hemilaminectomy with removal of approximately half of the suprajacent and infrajacent lamina was sufficient to decompress the stenotic spinal canal.

In 10 patients, spontaneous fusion between the vertebral bodies had occurred. In nine, a degenerated, bulging disc was removed. The interspace was curetted and all accessible disc tissue that could be safely removed was excised. The step-like edge in the floor of the canal was either tamped down or removed in nine patients if it contributed significantly to neural compression. In two patients, 45 and 46 years of age, interbody fusion was performed by packing the intervertebral space with bone chips removed at the time of laminectomy. One patient (Case 1) had a posterior and lateral fusion (Fig. 1). All demonstrated firm union after 6 months.

Five patients either died of unrelated causes or were lost to long-term evaluation. The remainder were available for follow-up study of 1 to 9 years. No one required additional surgery for further decompression or fusion. Radicular pain was significantly relieved in all, usually within the early postoperative period. Two with residual complaints were still able to care for themselves without assistance. Early rehabilitation and physical therapy resulted in sustained improvement in motor power. Once the critical radicular pain was relieved, whatever backache remained was readily controlled with non-narcotic analgesics.

There were no major postoperative complications. Patients were out of bed by the second to third day after operation and were discharged after approximately 2 weeks. No patient depended on a back support. Beta-dexamethasone (Decadron) was used during the past 2 years in diminishing doses for the week after surgery, beginning with 4 mg, four times daily, with definite reduction in the immediate operative pain.

Two patients aged in their forties were not fused, yet they fared as well as the three who had stabilizing procedures. Fusion should be considered in the younger individual with back pain as a prominent complaint. In older patients with advanced spondylosis and arthrosis, spontaneous fusion has probably already occurred, and further arthrodesis is unnecessary. In one patient, follow-up myelograms performed 3 months after laminectomy and facetectomy for symptoms of cervical cord disease showed excellent filling of
the thecal sac in the former area of stenosis (Fig. 2).

Illustrative Case Reports

Case 1

This 46-year-old woman was injured in a rear-end automobile collision 2½ years before admission and complained of increasing low-back pain with right sciatica. During the past year, she developed urinary frequency with intermittent incontinence. She showed no reflex or sensory deficit; straight-leg raising was unrestricted. Paravertebral spasm in the lumbar region was associated with loss of normal mobility. Serial x-ray films (Fig. 1) showed collapse of the interspace at L4–5 with progressive anterior dislocation of the L-4 vertebra to a maximum of 1 cm where it remained fixed (Fig. 1 center).

Rudimentary, arthritic posterior facets were present at the L4–5 level bilaterally (Fig. 1 right, arrow), and there was a partial block at this interspace (Fig. 1 left and center). Complete laminectomy of L-4 and L-5 with liberal foraminal decompression and medial facetectomy was followed by spinal fusion across the transverse processes from L-4 to the sacrum. The radicular pain subsided rapidly, and 3 years after surgery the patient was participating in all of her normal, vigorous activities without restriction.

Case 2

This 66-year-old woman presented with a 1-year history of back pain with radiation down her legs associated with numbness and dysesthesias spreading into the dorsal surfaces of her feet. Weakness and numbness of her legs increased after standing and walking (neurogenic claudication). There were no objective sensory changes. Ankle reflexes were depressed or absent, but patellar reflexes were slightly hyperactive and equivocal plantar responses were noted. Straight-leg raising was unrestricted. X-ray films of the spine disclosed anterolisthesis at L4–5 of approximately 1 cm with advanced degenerative spondyloarthritis involving multiple lumbar segments. Spinal stenosis was evident on plain films, confirmed by myelography. The changes in the posterior articulations at multiple levels cause serial defects in the myelogram, most severe at L4–5, where hypertrophic facets indented the terminal portion of the column of oil above the block (Fig. 2 upper).

Because of stenosis and multiple-level disease, laminectomy of L3–5 was done with foraminotomy and medial facetectomy, most extensive at the L4–5 level where the canal was obliterated. Postoperatively, the patient was relieved of her pain, but paresthesias remained. Three months later, her gait became spastic and she developed sensory changes up to the C-6 area with poor boundaries. Plantar responses were extensor bilaterally. Myelography (Fig. 2 lower) showed excellent filling of the dural sac in the lumbar region at the level of the decompression. However, there was evidence of cervical spondylosis and stenosis. The patient refused further surgery.

Case 3

A 46-year-old woman with back pain for 3 years complained of increasing weakness and numbness in both legs for 1 year. Symptoms increased with standing and walking, and were relieved only by sitting or lying down (claudication). There was unrestricted straight-leg raising bilaterally, absent ankle reflexes, and profound weakness and atrophy of both anterior tibial muscle groups. Sensation was intact. Spine films showed osteoarthropathy of the facets at L4–5 with anterolisthesis of 1 cm and a narrowed interspace.

A partial block was noted on myelography with the inferior articular facet playing a prominent role in causing the obstruction (Fig. 3 upper right).

Bilateral laminotomy and foraminotomy with hemifacetectomy and undercutting of the facets was done at L4–5. A degenerated disc was removed along with the vertebral plates and the interspace was packed solidly with bone fragments.

Recovery was uneventful and fusion was demonstrated at 6 months. Weakness and atrophy improved rapidly; the patient was without pain after 3 months, and has continued to improve for 3 years.

Discussion

Erect posture provides a constant forward thrust to the lower lumbar vertebrae, counteracted by the presence of facets, intact

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Fig. 2. Case 2. Upper: Preoperative myelograms. Anteroposterior view shows severe stenosis (left, arrows) at L4-5. Lateral view shows dorsal dislocation (right, arrow) of dural sac by facets. Lower: Postoperative myelograms, anteroposterior (left) and lateral (right) views, show excellent filling of dural sac (arrows).
FIG. 3. Case 3. Upper: Preoperative myelograms. Lateral view (left) showing interruption of column dorsally (white arrows) by facet encroachment, and ventrally by the step deformity created by listhesis (black arrows). Oblique view (right) showing tapering of column above the L4-5 interspace. Arrows point to arthrotic facets. Lower: Lateral (left) and posteroanterior (right) diagrams show how posterior articularizations cause the stenosis and block seen on the myelograms. Arrows show superior facets, dotted circles show level of pedicles, and shaded areas show oil column.
pedicles, neural arches, normal bone structure, and soft tissue investments. A deficiency in any of these allows forward displacement to occur. In the normal lumbar spine, the axis of flexion and extension passes through the nucleus pulposus. With degeneration of the intervertebral discs, this axis moves posteriorly through the apophyseal joints causing abnormal movement. The resulting arthrosis with hypertrophy of the joints contributes to stenosis and foraminal nerve root compression.  

Allbrook has shown that L-4 has the highest range of mobility, with L-5 next. In addition, the facets between L-4 and L-5 are most susceptible to trauma because they are placed obliquely to the transverse plane. When more sagittally disposed, slippage in a ventrodorsal direction is facilitated. This fact may account for the unusual frequency of degenerative spondylolisthesis at this level. Because the facets at the lumbosacral level present in a coronal or transverse plane, forward dislocation of the vertebral body is possible only in the presence of a defect in the neural arch or an anomaly of the facets. Newman and Stone described arthritic inferior facets that slipped forward, with the progression stopped only by the bone bars on the anterior hooks of the superior facets of the infrajacent vertebra which resisted further displacement (Fig. 3 lower).

In no case of degenerative spondylolisthesis is the dislocation of the vertebral body more extensive than a quarter of the diameter of the vertebra; the measurements provided by McNab averaged 0.6 cm with a maximum of 1 cm. These measurements are remarkably constant both in the present report and in the literature. Complete spondyloptosis never occurs. Stenosis of the spinal canal predisposes such individuals to neural compression, permitting anterior dislocation of only 1 cm or less to obstruct the vertebral foramen.

Disc degeneration by itself produces a downward and backward dislocation of the vertebral body because of the normal forward inclination of the superior articular facets. This results in a minor degree of retro-spondylolisthesis, which, in our experience, has not been a cause of neural compression. Subluxation at the lumbosacral joint in juveniles is a basic feature of congenital spondylolisthesis with spondylolyosis. Vertebral slippage without an interarticular defect rarely occurs at this level. The marked infrequency of cauda equina syndromes in the presence of congenital spondylolisthesis is explained by the fact that the defect in the isthmus may result in the lamina being left behind as the vertebral body slips forward, causing less stenosis of the spinal canal. With the lamina intact, however, nerve roots and the cauda equina are more readily trapped in a pincer mechanism similar to that encountered in the cervical region where degenerative spondylolisthesis may cause severe myelopathy.

Evidence that degenerative spondylolisthesis is an acquired disorder was provided by Newman and Stone. Five of their patients between 30 and 40 years of age with backache showed no slip in their original x-ray films, but subsequently developed the typical findings of anterolisthesis, with an intact neural arch.

A long history of back pain usually precedes the development of nerve root or cauda equina compression in patients with degenerative spondylolisthesis. During this interval, fusion is indicated for the mechanically unstable spine. Unfortunately, there are no published reports of long-term results. Since the nerve roots or cauda equina may be ultimately caught in the stenosis caused by the lamina and inferior facets of the displaced vertebra slipping forward and approaching the posterior elements of the infrajacent vertebra, decompressive laminectomy must be considered. The spondylosis and associated arthrosis of the facets in older individuals may result in severe foraminal constriction, which makes total facetectomy mandatory (Fig. 4).

In the 22 cases described by McNab, there were signs of nerve root compression in 11. There were no cases of cauda equina involvement. The average age was 60 years and the usual radiographic finding was an osteoporotic, osteoarthritic spine with slight anterior displacement of L-4 and no defect in the neural arch. He presented seven male and 15 female patients. This ratio is similar to that reported by Junghanns and by Newman and Stone.

Adkins described eight cases occurring at the L4-5 level where ventral displacement of the lamina constricted the thecal sac. The forward slip was limited only by impingement of the displaced inferior facets against the

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THE cephalic surface of the vertebra shows extensive degenerative disease of the facets and indicates how far medially the inferior portions of the superior articular facets can extend (arrows). Narrowing of the lateral recesses is evident. Right: The caudal surface, arthrotic inferior articular facets narrow the lateral recesses and entry zones to the foramen, more on the left side. This specimen also shows the protective tunnel around the pedicles in which the nerve root is encased beneath the facet as it leaves the spinal canal. The absence of osteophytes on the margins of the vertebral body is noteworthy and is a frequent finding in advanced apophyseal arthrosis. (From the collection of the Department of Anthropology of the American Museum of Natural History in New York City.)

vertebral body. At surgery, the nerve roots were squeezed medially and were densely adherent to the tip of the displaced facets. In one patient, slippage had occurred because of a congenital defect in the posterior facets of the displaced vertebrae, similar to one of the patients in this report (Fig. 1). This is one of the rare cases described with a cauda equina syndrome.

Transverse axial tomography on patients with degenerative spondylolisthesis and stenosis has demonstrated the forward dislocation of the lamina with the inferior articular facets lying ventral to the adjacent sclerotic superior facets.9 Intermittent neurogenic claudication commonly found in developmental spinal stenosis was found in seven of our patients and represents a symptom of diagnostic importance. Relief was achieved in all with proper decompression (Figs. 2 and 3).10

It is not uncommon to find varying degrees of anterolisthesis and retrolisthesis during review of spinal x-ray films in patients with low back disorders. Dislocation of 2 to 3 mm rarely produces symptoms unless an accompanying arthrosis of the posterior facets causes significant narrowing of the intervertebral foramen. After extensive laminectomy with injury to the posterior facets or even excision of these facets, degrees of retrolisthesis and anterolisthesis may reach alarming proportions. Because of the accompanying dorsal unroofing of the spinal canal, however, cauda equina syndromes rarely occur. Nonetheless, one must be aware of the possibility of such deformity during the years subsequent to extensive decompression, especially in patients with spinal cord tumors in whom muscular weakness adds to instability.

Age is no contraindication to decompressive surgery. With proper preparation and medical clearance, in a well-motivated, vigorous individual, restoration of function can be gratifying.

References

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13. Verbiest H: Neurogenic intermittent claudication in cases with absolute and relative stenosis of the lumbar vertebral canal (ASLC and RSLC), in cases with narrow lumbar intervertebral foramina, and in cases with both entities. Clin Neurosurg 20:204–214, 1972

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