POSTOPERATIVE INFLAMMATORY DISEASE
OF LUMBAR DISCS*

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The surgical treatment in certain cases of sciatica by removal of the protruding portion of a lumbar disc has become a fairly uniform procedure. Criteria for operation, the technical procedure, and the functional end-results are much the same from one medical center to another. In contrast to the banal appearance of this group as a whole, I will describe 3 patients whose courses deviated a long way from the beaten track. These are instances of presumed infection that developed within the disc after operation. They are described as presumed infection because culture of the causative organism was not obtained in any of them, although a psoas abscess was drained in one, and purulent fluid drained from the disc of another. The 3 cases are dissimilar in several ways, but parallel in respect to their long morbidity, their evidence of severe toxemia, and their end-result.

It is necessary to relate that in my experience with the surgery of lumbar discs (about 300 operations) there has been no instance of major postoperative infection arising in the soft tissues. I have not enjoyed the same immunity from gross soft-tissue infections in cases in which a combined disc and fusion operation was performed. But I am speaking of cases in which the disc alone was disturbed, and in these, healing of the wound has been the rule.

Acute infection of intervertebral discs is usually classified with osteomyelitis of the vertebrae. In 1940, Ghormley, Bickel and Dickson4 of the Mayo Clinic reported 20 cases in which a diagnosis of infectious spondylitis had been made. They were focussing attention on a special group of cases in which there was very little osteomyelitis of vertebral bodies, but evidence of severe infection within a disc. Three of their patients showed clinical evidence of irritation of the central nervous system—a point in common with my cases. None was related to operation.‡

Certain anatomical and pathological features of intervertebral discs influence the course of infection. Within the normal disc after the third dec-

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Submission for publication was postponed in expectation of a more illuminating discussion from some neurosurgeon of greater experience with disc surgery. This tardy appearance may stimulate such a report.
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‡ Since writing this paper brief references to postoperative infections have been noted in two publications by Armstrong.1,2
ade, there are no blood vessels or lymphatics.\(^3\) This situation is altered following injury. When there has been a rupture through the posterior margin of a disc the process of healing involves an intrusion of blood vessels into the disc along with the invading fibrous tissue.\(^4\) If the rupture has been very gross the protruding lump of fibrocartilage may have to be pulled out at operation. Then there will be even greater vascularization of the interior of the disc. In either case, a blood-borne infection might find a satisfactory locale for activity. In the patient who has had operation an adjacent extradural infection would find an easy path of entry. The poorly healed hole in the posterior spinal ligament and fibrous cover of the disc is like a weak trap door.

With these pathological postulates in mind, I will describe briefly the clinical stories of 3 middle-aged British Columbians—a Finn, a Swede, and a Czecho-Slovak. They are all grateful for surgical relief from severe sciatica, but badly shaken by the morbid events that followed.

**Case 1. G.H., Finnish workman, aged 46.**

In July 1947 the patient presented a typical clinical syndrome of protruded intervertebral disc with sciatica, which had been initiated by injury. The signs were characteristic of involvement of the left 1st sacral root. Myelogram disclosed a rather indefinite defect on the left side at L5-S1. Disc interspaces were normal.

**Operation.** A small, firm protrusion was found at L5-S1 and pulled out as one chunk. The interior of the disc was not curetted.

**Course.** His initial postoperative progress was uneventful. Temperature was normal by the 3rd day. On this day a sore throat and cough developed, which continued for a week. He was out of bed on the 10th day and left hospital 3 weeks after operation.

Three weeks later he returned, looking miserable and complaining of severe headache with vomiting, which had troubled him for about 3 days. He was re-admitted to hospital. His neck was very stiff and he had bilateral Kernig sign. Cisternal puncture was done; the CSF contained 525 WBC, 44 per cent polymorphs, 56 per cent lymphocytes, and 115 mg. protein per cc.

He was started on large doses of penicillin. CSF obtained by lumbar puncture 3 days after admission showed 1150 WBC, 28 per cent polymorphs, 72 per cent lymphocytes, and 200 mg. protein. Successive lumbar punctures disclosed a gradual improvement in the CSF which coincided with a gradual clearing up of his meningitic signs and symptoms. His temperature had remained normal and his pulse rate in the 60’s from the beginning. Two months after admission the CSF cell count was 33, with 90 per cent lymphocytes, and 105 mg. protein.

As the headache cleared up increasingly severe low backache developed, with pain radiating to both legs. He was discharged from hospital after 3 months, still complaining of pain in his lower back.

A month later he looked very miserable, so shaky that he had to lie down to take off his low-back belt, depressed and unnerved. He returned to his home in a small town up the B.C. coast. Up there, under the direction of a physiotherapist he made a slow, but excellent recovery. He returned to his regular work, about 1 year after the original operation.

Two years later he was still working steadily with only occasional stiffness in his back after a hard day. There was moderate limitation of flexion of the lower
back. Roentgenograms showed apparent solid fusion at the lumbosacral level with generalized calcification and considerable new bone production anteriorly.

Case 2. N.B., Swedish workman, aged 50.

He was first examined on Jan. 7, 1949, complaining of sciatica in the left leg, with typical story and clinical signs of a protruded disc. There was no pre-operative myelogram. AP and lateral X-ray films before operation showed nothing remarkable.

1st Operation. The interlaminar spaces on the left side between L4 and S1 were explored. At the L4–L5 level a rupture and protrusion of the disc was disclosed. The usual stringy chunk of fibrocartilage was pulled out and further soft cartilage was removed from within the disc with curette.

Course. We expected a good recovery, but he did very poorly from the start. He complained of headache for about 4 weeks, but after the 1st week the headaches were overshadowed by severe pains in his lower back. For about 5 weeks he had a daily rise in temperature from normal to 100° or 101° and a persistent, high sedimentation rate.

He was a very thin man before operation and during the course of the illness the loss of weight became extreme. Six weeks after operation myelography was performed. The CSF showed no cells but a protein of 50 mg. There was a large filling defect at the L4–L5 level, occupying the anterior part of the canal above and below the level of the disc. There appeared to be some destruction of bone on the inferior surface of the body of L4.

2nd Operation. On entering the disc, a thin purulent fluid poured out. A large amount of soft degenerated cartilage was removed. This cartilage was heavily infiltrated with polymorphs, lymphocytes and some plasma cells. No organisms, tuberculous or otherwise, were found on smear and culture showed no growth.

Course. Two months after the 2nd operation, roentgenograms showed some destruction of the inferior portion of the body of L4, and slight narrowing of the L4–L5 disc space. New bone formation was beginning to bridge lateral margins of the space. After the 3rd month his temperature remained normal. Four months after the 2nd operation X-ray films demonstrated further consolidation of new bone around the L4–L5 space. His general condition remained so poor that it was not until the end of 6 months that he was fit to leave hospital. Two months later he was getting along well and returned to his home in the country.

Case 3. J.M., Czecho-Slovak farmer, aged 44.

The patient was first seen in July, 1946, complaining of recurrent right-sided sciatica of 6 years’ duration. The history was typical of protruded disc. The spinal fluid was normal. Plain X-ray films appeared normal. Myelography demonstrated filling defects at both the L5–L4 and L4–L5 levels.

1st Operation. Both levels were explored. A characteristic protrusion of disc was encountered at the L4–L5 level. The protruding loose mass was pulled out and further cartilage was removed from within the disc by curette.

Course. Recovery was rapid and uneventful.

Three years later (March, 1949) he returned complaining of sciatica in the opposite (left) leg, which had troubled him for 3 months. He was grossly disabled. Again the symptoms and signs were typical of disc protrusion. X-ray films demonstrated that the L4–L5 interspace had been well maintained. There were no gross adjacent degenerative changes.

He was admitted to hospital for myelography. To our surprise the spinal fluid
removed prior to this test showed a cell count of 77, with 30 per cent polymorphs, 70 per cent lymphocytes and protein of 50 mg. The myelogram showed some irregularities, but nothing that was considered clearly diagnostic. Blood counts were normal but sedimentation rate was elevated to 38 mm./hr. (Westgren). During the subsequent 3 weeks spinal fluid obtained by cisternal puncture was checked three times. The cell count remained in the 60's with the same relative percentage (30) of polymorphs. There was a daily temperature rise to 100°. He had no headache or stiff neck. The sciatic symptoms gradually diminished and he was discharged to continue rest at home.

He returned 3 months later complaining of return of severe pain in his lower back and left leg and was re-admitted to hospital. He had lost 30 lbs. in weight since the development of symptoms, and was having occasional severe night sweats. There was marked restriction of forward bending and bilateral hamstring spasm. He had an irregular low-grade fever. The spinal fluid was normal but WBC was 10,000 and sedimentation rate 84 mm./hr.

2nd Operation. The L4–L5 disc was re-explored. On the left side—the side of his radiating pain—the disc did not bulge and was not opened. On the right side, from which a protrusion had been removed 3 years before, the former hole was re-entered. No loose chunks of fibrocartilage were found within the disc and nothing was removed. There was no indication of infection.

Course. Pain in his back and low-grade fever and high sedimentation rate continued. Massive doses of penicillin and later large doses of streptomycin had no apparent effect. Two months after admission there was swelling of the operative area.

3rd Operation. Exploration revealed a small, deep abscess which did not seem to extend deeper than the lamina. Smear and culture were negative.

Course. After a further month the wound was healed. He was free of pain and had no fever but still showed a sedimentation rate around 50 mm. He was discharged from hospital with a plaster body cast, 4 months after re-admission.

He returned to hospital 2 months later (9 months after 2nd admission). Now he had a large, left psoas abscess and clearcut evidence by X-ray of a destructive process of the vertebral bodies adjacent to the L4–L5 disc which was markedly narrowed. Pus aspirated from the psoas abscess was negative on smear and culture. The abscess was drained and healed. He eventually returned home, 1 year after the delayed recurrence of sciatica. He was symptom-free, though still wearing a Taylor brace.*

DISCUSSION

One might enquire where the infection originated, or how it was introduced. Was it blood-borne, introduced by myelography, by spinal anesthesia or by operator? The data do not support a detailed argument. It is my opinion that in all 3 cases the infection was introduced at operation and was primarily a localized infection in the extradural space. That would account for the phase of meningeal irritation. In the first 2 cases the pathway into the disc was wide open from recent operation. The third case, with the 3-year interval, might be explained by a chronic, silent extradural infection

* One year later he returned for observation. Clinical examination was negative. He did not show up for recheck X-ray films.
which was stirred up by the second injury, and then provided with a fresh pathway of entry by the second operation. It is noteworthy that in Case 1, in which the curette had not been introduced into the disc at operation, there was no apparent acute infection of the adjacent vertebrae, whereas in the other 2 cases, in which the cartilage plates had been scraped, there was evidence of invasion of infection into the vertebral bodies.

CONCLUSIONS

This has been a report about a statistically insignificant group of cases. They illustrate the severity and chronicity of infection that may occur within a lumbar disc after an operation. The development of a clinical state that resembles tuberculous meningitis suggests the onset of this complication. My experience has not indicated how to treat it, except by expectant, conservative measures.

REFERENCES