IN June 1981, an interinstitutional orthopedic and neurosurgical study of the “failed-back-surgery syndrome” was published (1), identifying the primary factors causing such failures as:

Lateral spinal stenosis 58.5%
Recurrent or persistent disc herniation 14%
Adhesive arachnoiditis 11%
Central spinal stenosis 10.5%
Epidural fibrosis 7%
(All other factors were less than 5% each.)

This identification of causes has made subsequent monitoring possible. In the article published here, the causative factors are reviewed and their status in 1991, a decade after the original study, is described.

The cost of surgery on the spine, particularly the cost of failure of surgery on the lower back, is high. The need for understanding and decreasing the incidence of such failures is imperative given the challenge to reduce health care costs. In 1980, Kane (2) estimated that 250,000 Americans underwent lumbar laminectomy each year. A survey of the published literature on the subject suggests that the overall poor results of laminectomy have been about 30% over the years, but have been less than 10% in modern comprehensive spine care centers. Although the number of laminectomies being performed each year appears to have progressively decreased over the past decade, this trend has been offset by a myriad of invasive procedures, including chemonucleolysis, percutaneous nucleolectomy, arthroscopic microdiscectomy, and now laser microdiscectomy.

Unlike some underdeveloped countries, in which the rate of postoperative sepsis is as high as 20%, in the United States infection is not a significant factor in producing failures of surgery. The overall incidence is estimated at 2.5%, but the incidence is less than 1% at spine centers. A significant decrease over the past decade may be attributed to the advocacy of Malis (3) for the routine use of prophylactic antibiotics.

The study of past failures of surgery on the lumbar spine provides the opportunity to potentiate our prowess in the future. Surgery is a significant and expensive endeavor. Neurosurgeons can no longer afford those failures which are preventable.

Lateral Spinal Stenosis

Failure to adequately identify or treat lateral spinal stenosis was identified in the interinstitutional study as the primary cause of failures of surgery. The reasons are many but begin with continued confusion over nomenclature. For example, what could be more basic than the spelling of the word “disc” (disk?) or “anulus” (annulus?). These terms appeared 702 times in the 1989 International Society for Study of the Lumbar Spine abstract book, spelled as follows:

disc, discs 614
disk, disks 12
annulus, annular 72
anulus, anular 4

Although this tabulation indicates a clear-cut preference of society members, there has never been general agreement on usage. On the other hand, spelling of the term “lateral spinal stenosis” is well established, but understanding of it is typically lacking.
Central, Foraminal, and Extraforaminal Zones. Many surgeons confuse lateral spinal with spinal recess stenosis, sometimes referred to as “subarticular stenosis.” I have found the concept of central, foraminal, and extraforaminal zones helpful (Fig. 1). With enlargement of the facet joints, there is narrowing of the most lateral portion of the central zone, referred to as narrow spinal recess. When a traversing spinal nerve is compressed, spinal recess stenosis is then said to be present. This tendency may be aided by a congenitally small (trefoil-shaped) spinal canal or by an anterior osteophyte.

Fig. 2 is a three-dimensional representation of classic “up-down” lateral spinal stenosis in grade I isthmic spondylolisthesis. From the surgical standpoint, the foraminal zone is a three-dimensional rather than a two-dimensional structure (Fig. 3). It may not be evident that the area of nerve compression may extend beyond the foramen to the extraforaminal zone, in what is called the “far-out” syndrome (Fig. 4).

Appropriate deciphering of the anatomy requires quality spinal imaging. Although magnetic resonance imaging (MRI) has opened up new vistas of information, it is sometimes used in place of computed tomography (CT), which remains the single most reliable anatomic map for the surgeon. MRI is not optimal in demonstrating bone spurs and osteophytes and may miss dense mass lesions, such as synovial cysts producing nerve compression. Adequate knowledge of the “geography of nerve entrapment” is the basis for effective surgical decompression. Primary decompression alone may not be fully adequate to restore normal nerve function.

“Secondary” Surgical Decompression. Some very important lessons have been learned during the past 10 years from patients with adequate decompressions who have not shown clinical improvement. Although some of these patients have permanent nerve injury, the majority of these compressed spinal nerves have not returned to optimal physiologic function. Many patients wit
seemingly “adequate” anatomic decompressions have not achieved improvement of neurohumoral transport, axoplasmic flow, arterial supply, and venous return due to continued nerve stretch over a foraminal uncinate spur or osteophyte. Elimination of this impairment can be referred to as the “secondary” surgical decompression involved in lateral spinal stenosis surgery.

Experimental studies have shown that when a spinal nerve is compressed or stretched, the most sensitive system is venous return rather than arterial supply. This observation correlates well with the nerve swelling seen at surgery and the transformation of the distended dorsal root ganglion into a hypersensitive pain generator. “Secondary” decompressive surgery eliminates the dorsal root ganglion as a pain generator.

Preserving Periradicular Spinal Nerve Veins. I recently had the pleasure of meeting with Harry Crock and asked his opinion as to why apparently adequate primary and secondary decompression for LSS sometimes fails. He chided me for the obsessive manner in which neurosurgeons, with their microsurgical technique and bipolar coagulation, routinely cauterize periradicular spinal nerve veins and thus create insufficient venous return of the nerve root. I now assiduously attempt to preserve these structures and control local bleeding with other means, such as the use of thrombin-soaked patties or local thrombin/powdered Gelfoam mix.

After 10 years, lateral spinal stenosis continues to be the most common reason for failure of surgery on the lumbar spine.

Recurrent or Persistent Disc Herniation

In many patients with an L4–5 disc herniation, it is necessary to remove some of the L4 lamina in order to gain adequate surgical access to the disk. This relationship can be appreciated on a plain spine X-ray. The lack of evidence of a surgical defect frequently suggests the source of the problem.

Routine CT cannot differentiate between herniated disc and isodense epidural (or perineural) fibrosis. Intravenous enhancement techniques have not been very impressive and require inordinate amounts of parenteral contrast material. MRI with gadolinium has been recommended by some for the differential diagnosis. The greatest help I have found has been the double echo, spin-echo T2 weighted technique. With the added advantage of being noninvasive, axial studies also permit evaluation of arachnoiditis, which is not imaged by T1 gadolinium technique.

A small percentage of the population with low back pain accounts for the majority of the costs. Webster and Snook (4) recently documented that 25% of cases of low back pain accounted for 95% of the money spent. In my practice, recurrent disc herniations occur frequently in individuals (often quite young) demonstrating multilevel degenerative disc disease on MRI. The pattern of involvement tends to spare the L3 disc and typically includes internal disc disruption, multiple end-plate deformities (that is, Schmorl’s nodes, limbus vertebrae), anular thickening, vertebral molding, facet degeneration, and herniated discs (Fig. 5). Most of the patients have been in the second or third decade of life, and family groups showing these changes have now been identified.

At the Center for Diagnostic Imaging in Minneapolis, Ken Heithoff and his associates surveyed 1,419 MRI scans performed during the first 3 months of 1990 and found multilevel degenerative disc changes in 9% of the patient group; males represented 75% and females 25%. For the present I have given the entity the designation “juvenile discogenic disease.” There is every reason to believe that this population will prove to be a target group for recurrent disc herniation and failure of surgery.

Adhesive Arachnoiditis

The study of foreign-body–induced lumbosacral adhesive arachnoiditis over the past 50 years, and particularly during the past decade, has been fascinating but also frightening. The
The medical profession has not yet succeeded in finding a benign, effective myelographic medium. Thorotrast (stabilized suspension of thorium dioxide) turned out to be a radioactive (beta emitter) contrast medium producing malignancies of the neural axis, often 20 to 30 years later. Lipiodol (combination of iodine in poppyseed oil), introduced by Sicard and Forestier in 1921, was still being recommended by Mixter (5) in 1944 because “it does no harm.”

In 1944, Pantopaque (ethyl iodphenylundecylate) was introduced by Steinhausen et al. (6); based on experimental studies, they “assured” the medical profession that Pantopaque was “safe” for intrathecal injection. The conclusion, in retrospect, does not appear justified by the data. Soon after Steinhausen’s (6) publication, additional trials in Sweden demonstrated toxicity, and the product was banned for human use. Surprisingly, the same level of concern was not exhibited by the remainder of the world’s medical community, and not until the late 1970s did the causal relationship of iophendylate and lumbosacral adhesive arachnoiditis become clearly evident. A 1978 symposium at the International Society for Study of the Lumbar Spine reviewed the pathologic anatomy (7) (Fig. 6). Subsequently, a data base on Pantopaque toxicity was established at the Association of Consulting Toxicologists in Salt Lake City, Utah. By 1981, the role of Pantopaque as the single most significant toxic agent in the production of lumbosacral adhesive arachnoiditis was well established in the literature. Additional studies of the condition, its anatomy and physiology, and its relationship to autoimmune factors were recently reviewed and updated (8).

The disease is an insidious process that has been, and continues to be, a significant cause of failures of back surgery. During the period of maximal use, an estimated 450,000 iophendylate myelograms were performed each year in the United States. In each case, at least a mild meningeal reaction occurred. How many of these patients went on to clinically significant adhesive arachnoiditis is not known, but about 11% of total number of surgical failures were performed to treat that disease.

The condition is a cruel one. Not only is the pain typically constant and unrelenting, and thus particularly disabling, but afflicted individuals have often been considered to have functional disorders. Many physicians still maintain that iophendylate is safe and that they have never seen a case of lumbosacral adhesive arachnoiditis. Iophendylate (as Pantopaque or Myodil) continues to be used worldwide for diagnostic purposes, despite the advent of better myelographic agents, CT, and MRI scanning. As recently as 1987, the Minnesota State Neurosurgical Society passed a resolution supporting the continued use of Pantopaque as a diagnostic medium. To date, no medical, legal, or governmental ban or prohibition has ever been enforced in the United States. It is therefore not surprising that groups of patients with postmyelographic lumbosacral adhesive arachnoiditis have formed self-help groups around the world; their activity has led to the recent initiation, in both the United Kingdom and the United States, of class action legal suits against the manufacturers of iophendylate.

Central Spinal Stenosis

Central spinal stenosis following laminectomy continues to be a cause of failure of surgery because of confusion as to what constitutes the central spinal canal zone and the failure to adequately decompress the spinal recesses at the time of surgery.

Epidural Fibrosis

The actual role of postsurgical scar tissue in producing failures of surgery on the lumbar spine remains unclear. To some extent, epidural and perineural fibrosis is a "normal" sequela of a spine...
nal operation. Yet that local scarring can be a liability and significantly decreasing its presence is worthwhile. Inherent in decreasing fibrosis is also the need to reduce the volume of surgical hematoma and the promotion of a dural seal should the dura be torn. The multiplicity of present approaches to achieve these aims attests to the lack of a clear answer to the problem.

In 1978, I began to explore full-thickness autogenous fat grafts to prevent epidural fibrosis (9). Autogenous fat grafts have proven to be the most satisfactory of the various options available but have a number of drawbacks. The most satisfactory soft fat is obtained from the perigluteal area, but patients often complain of postincisional pain, and the harvesting extends the time of surgery. Too large a graft, overly dense fat, or placing fat over previously compressed dura (CSS) can produce cauda equina compression.

Despite the trial of numerous alternatives, a clearly optimal means of avoiding failures of spinal surgery due to postsurgical scarring has not yet emerged.

Summary

A decade has passed since the primary factors in failures of surgery on the lumbar spine were identified, and the entity is still an expensive, disabling reality for too many patients. The incidence of failure has significantly decreased due to better practices as well as advances in the technology of surgical diagnostic studies and better means of conservative care. The author would like to join his colleagues in acknowledging the many contributions of Dr. Leonard Malis throughout his career toward improving the quality of patient care and helping avoid failure of back surgery in all patients.

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References