The Failed Back Surgery Syndrome
Pitfalls Surrounding Evaluation and Treatment

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- Failed back surgery syndrome • Myofascial pain • Chronic neuropathic pain
- Interventional pain management techniques • Interdisciplinary pain management

INTRODUCTION

FBSS is persistent or recurring low back pain with or without lumbosacral radiculopathy after 1 or more spine surgeries. The incidence of FBSS is reported as between 10% and 40% but ranges between 5% and 50% have been quoted for microlaminectomy alone. The incidence is known to increase with more complex surgeries and has not improved with the development of less-invasive advanced surgical techniques. The failure rate for lumbar fusion is reported between 30% and 46% based on previous reviews whereas the failure rate for microdiscectomy is thought to range between 19% and 25%. The financial costs are considerable.

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RISK FACTORS FOR FAILED BACK SURGERY SYNDROME

Chan and Peng\(^1\) provide an excellent review of the risk factors for the development of FBSS. Specific psychosocial risk factors that have been found to result in poor outcome for spinal surgery are significant levels of depression, anxiety, poor coping, somatization, and hypochondriasis.\(^1,8\) The presence of a worker’s compensation claim is consistently cited in the literature as a risk factor associated with poor surgical outcomes and is often dismissed as due to secondary gain.\(^1\) It is important to differentiate true secondary gain from symptom magnification imposed by inability to obtain timely diagnosis and treatment. In addition, FBSS patients often pursue disability claims after job loss to retain insurance coverage for ongoing treatment. FBSS is, therefore, a biopsychosocial problem where indirect and intangible costs play a significant role in defining morbidity.\(^4\)

A major difficulty in preventing FBSS is that the ideal time to operate is not well defined in the literature.\(^1,3\) Surgical decision making is clear when there is progressive motor loss or cauda equina syndrome. But the timing and indications for surgery when pain is the primary complaint are not well defined. A general dictum is that 6 to 12 weeks of conservative care is reasonable prior to surgery.

It has been stated that the 2-year outcome for patients treated with either laminectomy or microlaminectomy is the same as for those treated with conservative care.\(^1,9–11\) The time to pain improvement is faster, however, with the surgical groups. If considerations for lost productivity and lifestyle compromise are openly discussed, surgery may be a reasonable option for refractory pain as an earlier option.

Earlier surgical intervention for low back pain may make sense in selected cases on a physiologic basis. Since the 1970’s it has been accepted that untreated pain promotes persistent pain patterns in the central nervous system in as little as 3 months. This is often attributed to “wind-up phenomena” or central sensitization at the levels of the spinal cord and central nervous system. Once chronic pain patterns develop treatment because more complicated and the likelihood of a successful outcome diminishes.

Deciding on the ideal time to operate is further complicated by disuse atrophy and chronic inflammation promoting physical deconditioning,\(^3\) making restoration of function and pain control more difficult after surgery. Prolonged pain and distress also may exacerbate preexisting psychosocial stressors, which is especially problematic with hypervigilant patients who have poor pain tolerance because they may simply refuse to normalize their activities, thereby creating a vicious cycle of pain and deconditioning. Thus, although an ounce of prevention may be worth a ton of cure, nihilism with respect to surgical decision making is not reasonable.

PREOPERATIVE RISKS

- Prior surgery
  - Spinal instability has been noted to occur in 12% of cases after a first surgery and increases to greater than 50% after 4 or more revisions.\(^12\)
- Surgery based on imagining abnormalities without good clinical correlation\(^1\)
- Nonsurgical causes of radiculopathy and neuropathy, including toxic-metabolic neuropathies (eg, diabetes), viral and inflammatory radiculitis, vascular disease, and plexopathies due to a pelvic mass or trauma

INTRAOPERATIVE RISKS

- Difficult radiographic localization intraoperatively during microsurgical cases or when there are segmentation defects\(^3\) causing operation at the wrong level, thus leaving the true pain generator without intervention\(^1,3\)
Inadequate decompression may leave a pain generator intact.

Aggressive decompression may lead to spinal instability and pain. \(^1\)

Loss of disk height after diskectomy may lead to vertical stenosis, compression, and pain.

Unrecognized pathology, such as disk fragments in the neural foramen, kinking of the nerve root by the adjacent pedicle, root compression by the articular process, spinal stenosis, extraforaminal disk herniation, and conjoined nerve roots may frustrate a successful outcome. \(^1,3\)

Excessive retraction, bleeding, or use of cottonoid patties may lead to battered root syndrome. \(^1,3\) Battered root syndrome is thought to occur in as many as 13% of cases with diskectomy and should be suspected if there is incomplete resolution of sciatic symptoms with or without progressive neurologic deficit that progresses over 3 to 6 months. \(^1,3\) It may be more common if there are conjoined nerve roots. \(^1\)

Pars interarticularis fracture may occur during decompression.

Use of methyl methacrylate as a primary means of stabilization or as a salvage technique for stripped screws may damage neural elements through compression or heat. \(^3\)

Roots may be damaged by graft extrusion after posterior lumbar interbody fusion (PLIF) or from excessive retraction.

Finally, the aim of surgery may be difficult to achieve. Proper decompression for foraminal stenosis due to ligamentous hypertrophy or far lateral disk herniation may cause destabilization of the segment and postoperative pain. \(^1\)

**POSTOPERATIVE FACTORS**

- Postsurgical complications
  - Hematoma or infection
  - Symptomatic pseudoarthrosis after fusion surgery
  - Epidural fibrosis may tether spinal nerve roots and interfere with cerebrospinal fluid-mediated nutrition of the nerve roots or interfere with the vascular supply of the nerve roots.
  - Pseudomeningocele, which can result from inadequate closure or inadvertent meningeal tear. This is a pseudocyst with no true meningeal lining that is secondary to postoperative dehiscence and can affect up to 2% of postlaminectomy patients. Patients with pseudomeningocele complain of wound swelling, headache, and focal neurologic symptoms, including radicular pain and cauda equina symptoms. \(^1,3,13\)
  - Persistent irritation of the nerve roots may also result from postsurgical arachnoiditis and can result in both axial and lower limb pain. \(^1\) MRI T2-weighted fast spin-echo sequences provide the best evaluation for arachnoiditis. \(^13\) There are 3 patterns of presentation on MRI:
    - Type 1 is a conglomerate of nerve roots seen as nerve root clumping, and this pattern is associated with the mildest involvement.
    - Type 2 is due to peripheral adhesions of the nerve roots to the thecal sac producing the so-called empty sac appearance and is associated with moderate involvement.
    - Type 3 refers to an intermediate attenuation mass obliterating the subarachnoid space below the conus medullaris and is thought to produce the most severe presentation. \(^13\)
  - Anatomic or biomechanical alterations
Spinal instability has been noted to occur in 12% of cases after a first surgery and increases to greater than 50% after 4 or more revisions.\textsuperscript{14} Loss of disk height after diskectomy may lead to vertical stenosis, compression, and pain. Transition syndrome,\textsuperscript{1,15–17} or adjacent level disease, is due to altered biomechanics that imposes increased load across adjacent spinal segments after diskectomy, thereby accelerating preexisting disk degeneration. Similar mechanisms are thought to predispose to sacroiliac (SI) dysfunction, especially after fusion,\textsuperscript{16,17} and this is thought to occur in up to 36% of patients after lumbar fusion.\textsuperscript{1,16} Recurrent disk herniations are known to occur in approximately 15% of patients at the site of operation or in adjacent segments because of altered load distributions.\textsuperscript{1,18} Altered load distributions may exacerbate adjacent level spondylolisthesis or further stenosis as part of a transition syndrome.\textsuperscript{1,13} Myofascial pain

Myofascial pain may result from dissection or prolonged retraction of the paraspinals during surgery, causing denervation and atrophy. Altered postural changes postoperatively may also create permanent chronic strain.\textsuperscript{1} There are no large studies to demonstrate that development of minimally invasive fusion surgeries, such as PLIF, axial lumbar interbody fusion, and transforaminal lumbar interbody fusion, have an impact on the incidence of FBSS or this component of FBSS, despite that these surgeries do not disrupt the dorsal musculature extensively. The author’s anecdotal experience over 10 years in a university-affiliated spine center is that there is little if any difference.

Fusion disease is a form of myofascial pain that has been attributed to compensatory hyperextension of the lumbar spine exacerbating poor posture. It has been attributed to paraspinal and hamstring muscle spasm or atrophy.\textsuperscript{1} In the author’s experience, however, flexed posture and difficulty arising from sitting is even more common after low lumbar fusion. Short-term relief can be afforded with manipulation and focused physical therapy but recurrent and persistent pain, spasm, and limited of range of motion is common. In the author’s view, the recurrence of these symptoms is most likely due to a combination of permanently altered postural biomechanics from fusion hardware constructs and self-perpetuating muscle imbalances with the hip flexors overpowering the multifidi, short lumbar rotators, paraspinals, and transversus abdominis.

The difficulty demonstrated by long-term treatment of this problem emphasizes that myofascial pain is a complex problem demanding an individualized approach that targets specific deficits. Complicating factors include

- Differentiating referred versus true radicular pain
- Differentiating the role of osteoligamentous structures versus the role of neuromuscular coordination (motor control) for pain-free motion\textsuperscript{19}
- Understanding how predominantly tonic muscles and phasic muscles respond to injury\textsuperscript{20}

Myofascial referred pain can be easily confused with radicular pain. The description of chronic ligamentous strain or musculotendinous pain as deep burning ache is similar to dysesthesia and the patterns of pain can also confound diagnosis. Travell and Simons\textsuperscript{21} provide several examples of pain patterns responsive to trigger point therapy that seem indistinguishable from dermatomal radicular pain, for example, trigger points in the gluteus minimus and gluteus medius that mimic L5 radiculopathy.
The clinical presentation of myofascial pain is important for 2 reasons: (1) it is not responsive to many of the treatments commonly offered to chronic FBSS patients, such as opioids and interventional spine techniques; and (2) if left untreated, it becomes worse, making it more difficult to resolve. This is important because it feeds patient perceptions of incapacity and hopelessness and contributes to the vicious cycle of pain and deconditioning commonly seen with FBSS patients. There are 2 dominant theories of myofascial pain propagation. One focuses on peripheral mechanisms where damaged motor endplates locally propagate more trigger points, and the other posits that central sensitization in the dorsal horn causes expanded receptive fields in the spinal cord and amplifies perception. The issues of posture and balance most likely also play a significant role.

Another major consideration, relative to lumbar myofascial pain, is the issue of motor control if overloading of muscles, tendons, ligaments, disks, and joints are to be minimized. Motor control is the regulation of coordinated muscular activity allowing efficient transfer of loads to joint services. Motor control requires (1) accurate instantaneous feedback from mechanoreceptors at the joint/soft tissue interface, (2) appropriate interpretation of this input, and most importantly, (3) modulation and timing of the responses to accomplish specific tasks. The loss or perturbation of these neuromuscular adaptations can result in refractory pain, inefficient movement, and inappropriate force closure of joints during motion. These, in turn, can lead to articular microtrauma and inflammation, ligamentous laxity, and disuse atrophy, which can mimic neurogenic weakness.

The role of the inner ring muscles, consisting of the multifidus, thoracolumbar fascia, and transverses abdominis, and their interaction with the diaphragm and pelvic floor muscles to create a cylindrical supporting system for the lumbar spine through the regulation of intra-abdominal pressure is especially important. This is not an issue of strength but rather of timing and coordination, which are fundamentally important because the posterior portion of this ring, the multifidus, receives its innervation from the posterior rami and medial branches of the lumbar roots. Consequently, if there is injury to the lumbar roots, coordination of the multifidus can be compromised. Richardson and colleagues presented persuasive data that these timing issues play an important role and are common denominators in many cases of chronic low back pain. Motor control is generally regulated by reflex mechanisms at the level of the spinal cord but it can be affected by supraspinal influences, such as mood and arousal, and this too is consistent with chronic pain variability.

The propagation of myofascial pain is also affected by how tonic and phasic muscles respond to injury. Physicians learn that muscle fibers can be categorized histologically and by their speed of contraction:

- Type I, or slow oxidative muscle fiber, has high mitochondrial content.
- Type IIa, or fast glycolytic muscle fiber, has low mitochondrial content.
- Type IIb, or fast oxidative muscle fiber, has a high mitochondrial content like type I fiber, but, it has a lower glycogen content than type IIa fiber and an intermediate range of fatigue between type I and type IIa fiber.

Most human skeletal muscle is not truly represented by any of these types, however. A more functional classification characterizes muscles as either predominantly tonic or phasic. Tonic muscles are postural and not susceptible to early fatigue. Phasic muscles provide ballistic function (movement) and are less suited to endurance activities. Some muscles serve both functions depending on situational context, such as the vastus medialis, which stabilizes the knee during loading and standing (tonic function) but also extends the leg ballistically during activities, such
as drop kicking a football (phasic activity). The point of this classification is how these muscle types respond to overloading and injury. Tonic muscles tend to shorten with injury and overloading. Phasic muscles tend to weaken with injury and overloading. Examples of tonic muscles include the erector muscles of the lumbar spine, piriformis, and hip flexors. Examples of phasic muscles include the abdominal muscles, knee extensors, and gluteus muscles. The important point is that shortened tonic muscles can inhibit phasic antagonists and synergists, thereby preventing maximal activation and optimal trainability. Consequently, a vicious cycle is created and compounded by faulty substitution patterns that develop after injury and surgery. This situation is especially problematic after denervation or if fusion constructs prevent range of motion, because normal muscle balance may not be recoverable. Consequently, any successful rehabilitation strategy has to account not only for strength and flexibility but also for posture and motor control issues. Treatment of myofascial pain is also thought dependent on improving a patient’s aerobic capacity.

EVALUATION OF THE POSTOPERATIVE SPINE: HISTORY AND PHYSICAL

History and physical examination are the most important parts of evaluating FBSS. There is considerable overlap, however, of the types of pain associated with this syndrome. The potential anatomic and pathologic processes responsible for postoperative pain complaints are difficult to differentiate. The types of pain include myofascial pain, arthropathic joint pain, and radicular pain that follows true dermatomal patterns associated with dysesthesia, loss of sensation, or loss of power. Chronic neural pain of any kind, whether visceral, sympathetically mediated, or phantom pain, is diffuse, poorly localized, and not well described anatomically. It is sometimes described with combinations of cramping, aching, and tight or burning sensations and is, therefore, similar to myofascial pain; it is ill described. It may also be associated with hyperalgesia or allodynia, which is important because, like myofascial pain, chronic neural pain is less responsive to opioids and better treated with adjuvant medications and therapies.

Physical examination typically focuses on neurologic findings, such as deep tendon reflexes and motor and sensory examination. Musculoskeletal examination, however, should include flexibility and tests of hip mobility, seated and standing trunk rotation, lumbar flexion and extension, lumbar side-bending, and palpation of the SI joints and trochanters. The entire spine should be examined and concomitant disease causing myelopathy with increased tone, clonus, and Babinski and Hoffmann signs should be ruled out. Spondylosis is a generalized condition and does not affect the lumbar spine in isolation.

Side-to-side asymmetry of motion is more important than measured range of motion. Straight leg raise and femoral nerve stretch test are also important with respect to the postoperative spine. True radicular pain with numbness or dysesthesia in dermatomal patterns is important to differentiate from decreased flexibility and discomfort due to lost range of motion from chronic injury or postsurgical guarding. A good representative neurologic examination is available from the American Spinal Injury Association, and the American College of Rheumatology has excellent materials on trigger/tender point examination. Patients should be assessed for leg length discrepancies and focal weakness, especially footdrop, because orthotics and bracing may be necessary to minimize repetitive strain over the kinetic chain. Finally, screening tests for motor control and stabilization are also recommended. Two easy tests include
- Assessing balance from the hands and knee position with opposite arm and leg extension (the pointer dog test)
- Maintaining a stable prone plank position from a modified push-up position where the patient starts prone and elevates to the elbows and balls of the feet

The entire physical examination should not take more than 8 to 10 minutes of office time. Evaluation of functional potential is more important than immediate relief of pain. The initial cause of surgery, prior symptoms and neurologic deficits, type of surgery, current symptoms, and time since surgery are critical to know to evaluate the advantages and limitations of particular imaging studies, further work-up, and treatment modalities. It is also imperative to assess compliance with previous treatments and rehabilitation efforts to appreciate any psychosocial overlay that has an impact on care.

EVALUATION OF THE POSTOPERATIVE SPINE: WORK-UP

Radiographic evaluation is an important tool because it may reveal anatomic aberrations, allowing for definitive treatment and better prognostication, such as residual lateral recess stenosis. Diagnostic studies may also provide reassurance that serious pathology is not present and that neurologic deterioration is not likely to occur with increased activity.

Radiologic findings specific to FBSS should not be confused with postoperative normal variants. Small seromas and edema of the subcutaneous tissue after surgery should be expected and herniation of the thecal sac through a new laminectomy defect may produce a mass effect that is normal if it is seen to decrease on serial films over the first 30 to 60 days.

MRI demonstrating epidural fibrosis has to be interpreted with caution because most patients with epidural fibrosis are asymptomatic. Nerve root enhancement is often seen in asymptomatic patients for 6 months after surgery, but if nerve root thickening and displacement are seen, the positive predictive value of imaging improves. The main differential in this setting is recurrent disk herniation, and recurrent disk herniation needs to be ruled out in symptomatic patients.

Similarly, postoperative inflammation and fibrosis due to disruption of the annulus fibrosis and epidural edema may simulate recurrent disk herniation with noncontrast MRI studies. Vertebral endplates can demonstrate edema and enhancement in 19% of patients between 6 and 18 months after surgery and nerve root enhancement may linger for 6 weeks or longer in 20% to 62% of patients after surgery. Consequently, careful correlation with progressive clinical signs and symptoms is mandatory during any investigation for postoperative or persistent pain.

MRI is generally the modality of choice in the postoperative setting because it allows for evaluation of soft tissues, bone marrow, and intraspinal content. Metallic hardware may lead to magnetic artifact but this can be minimized with fast spin-echo sequences, short echo time, and longer repetition time. Titanium and vitallium hardware produce less artifact than stainless steel on MRI. T2-weighted sequences should also afford better visualization with less artifact and short time inversion recovery sequences and should be used for fat suppression to improve homogeneity of the image.

Contrast administration with T1 imaging is useful to distinguish inflammatory tissue from recurrent disk herniation. Contrast is also useful when infection is suspected because bacterial diskitis is associated with particularly intense contrast enhancement compared with normal inflammatory change that can be seen postoperatively. A heightened index of suspicion for infection should arise if fluid collections are seen in paraspinal areas or the anterior epidural space; if they are located adjacent to the
disk involved; or if there is psoas enhancement. Contrast-enhanced MRI with fat saturation is the modality of choice when infection is suspected because it allows for evaluation of bone edema and diskitis earlier than other modalities. Contrast-enhanced CT allows for assessment of associated bone involvement, phlegmons, and abscesses and both CT and ultrasound are useful for guiding biopsies.

CT is the modality of choice for bone and abnormal calcification assessment. Intravenous iodine contrast is required to investigate suspected infection. Metal artifact can be reduced with attenuation techniques, and software manipulation and orthopedic hardware with lower attenuation coefficients are known to produce less distortion. Titanium produces less distortion than stainless steel, which, in turn, produces less distortion than cobalt-chrome.

CT is particularly useful for investigating suspected misplaced or loosened hardware. Root irritation can be seen with misplaced pedicle screws encroaching on the lateral recess or foramen. In general, root irritation is associated with low or medial screw placement. Loosening of hardware can be seen with infection or stress fatigue and is associated with a halo or hypoattenuation greater than 2 mm around the hardware.

Conventional radiographs are particularly useful when there is hardware because there is no metallic artifact. Anteroposterior, lateral, oblique views and flexion/extension views are all useful when planning diagnostic or therapeutic interventional procedures and to diagnose structural problems, such as postoperative loss of disk height or spondylolisthesis with instability.

Electrodiagnostic evaluation of the postoperative spine can be useful for both localization and prognosis, especially if there is a preoperative study to compare with. A change in compound motor action potential and increase in peripheral membrane irritability may reflect worsening postoperative axonopathy. Similarly, membrane irritability may localize to the involved nerve root when there is multilevel disease. The presence of membrane irritability that correlates with at least one grade of motor loss on physical examination may assist treatment considerations if differentiating motor loss from involuntary guarding is difficult. For example, assistive devices and lifestyle management may be more appropriate in the presence of extensive neurogenic atrophy whereas aggressive strengthening exacerbates joint pain and instability.

Interpreting electrodiagnostic evaluations requires some caution. Sampling size always has an impact on recognition of membrane irritability and both positive and negative findings have to be measured against the clinical picture and radiology. As a rule, MRI is more sensitive than electromyogram (EMG) for diagnosing the cause of radiculopathy but EMG is more specific for the presence of radiculopathy. If both sensory and motor nerve conduction abnormalities are seen, a peripheral nerve problem is more likely than a preganglionic root problem. It is, however, possible for vertical stenosis or a large lateral disk herniation to affect the dorsal root ganglion and cause sensory nerve conduction abnormalities too, most commonly at L5.

Paraspinal denervation does not always equal lumbar disease. It can be seen in diabetics and, if associated with extensive denervation in more than the expected peripheral myotomes, the differentials for myopathic, neuropathic, or motor neuron diseases should come to mind. Finally, irritability in the paraspinals may persist because of incisional muscle damage and therefore, muscle sampling should be further than 2 cm from an incision line and interpretation must still be made with caution.

TREATMENT
The goals of treatment are to maximize neuromuscular and musculoskeletal efficiency with activity, control pain, and interrupt and reverse the progression of debility.
Medical therapy should be advanced with the goal of increasing physical activity and community involvement. Pharmacologic management includes

1. Nonsteroidal antiinflammatory drugs or acetaminophen
2. Muscle relaxants, such as cyclobenzaprine, methacarbamol, and metaxalone
3. True antispastic medications, such as baclofen or tizanidine
4. Antidepressants, such as tricyclics, selective serotonin reuptake inhibitors, and combined serotonin and norepinephrine reuptake inhibitors
5. Gabapentinoids
6. Tramadol
7. Opioids

Medication prescription should take into account mechanisms of action, presumed modification of specific symptom complexes, and side-effect profiles. In actuality, off-label indications, trial and error, and empiric decision making are common in community medicine settings; for example, low-dose amitriptyline or tizanidine may prescribed at hour of sleep to promote better sleep hygiene and improve myofascial pain.

The American Pain Society has published clinical practice guidelines for chronic low back pain but not specifically for FBSS. Cochrane reviews are also available for symptomatic relief of low back pain but again these do not specifically address FBSS. There is at least 1 case report of decreased pain and improved function with gabapentin as monotherapy for FBSS, but there are no formal studies of gabapentinoids and the effect on FBSS. Side effects and drug interactions often limit these therapies, and polypharmacy increases the risk of serious complications, such as serotonin syndrome. Consequently, opioids are commonly prescribed.

All of these medications have been well reviewed in Braddom’s textbook, Physical Medicine and Rehabilitation, 4th ed, with respect to mechanisms of action and dosage ranges. Another focused review is available from the American Pain Society/American College of Physicians Clinical Practice Guideline.

Nonopioid medications are considered adjunctive pain medications and are usually insufficient in the setting of FBSS. Opioids are generally considered safe and effective for moderate to severe pain but the use of opioids for chronic noncancer pain is becoming more controversial. This is especially true in the setting of FBSS, especially in the setting of instrumented lumbar fusion. Chan and Peng comment on a recent publication investigating mortality after lumbar fusion:

In this study, the leading cause of mortality (accounting for 31% of all deaths) was analgesic related. The overwhelming majority of deaths were related to opioids (20/22 patients with analgesic related death). While the majority were accidental, three deaths were the result of suicide. Of those patients who suffered from analgesic related mortality, all had undergone either instrumented fusion or intervertebral cage procedure. No patient with receiving lumbar fusion from autograft or allograft suffered from analgesic related death. While more investigation is required to determine why patients with instrumentation may be more prone to serious complications of opioid analgesia, this finding should caution the physician to be careful when prescribing analgesics for FBSS and to undertake close monitoring of patients on chronic opioids for pain.

FBSS patients are often pushed aside and told that their surgeons have little to offer them after their surgeries were unsuccessful. These patients are generally managed by community-based physicians, including primary care physicians, anesthesiologists, physiatrists, and neurologists. Scope of practice and the limits of a patient’s third party coverage influence the pharmacologic management, interventional
therapies, and physical therapy treatments that are offered. Once treatment options are expended, FBSS patients are often told to seek out a physician who will prescribe pain medications chronically.

Treatment of FBSS demands recognition that it is a chronic pain syndrome. One reason that FBSS is a difficult-to-treat syndrome and a public health problem is that the antecedent back pain did not respond to intervention and was often augmented with additional pain complaints caused by the surgery. The psychosocial burden for individuals is huge, especially if there is job loss or loss of function physically. Many of the immediate postoperative pharmacologic strategies complicate the situation further by altering the patient sensorium, mood, affect, and even libido. Opioid-induced hyperalgesia is probably under-recognized and under-reported.

Prescription opioid abuse is now considered a major public health problem in itself. Ten years after pain was recognized as the fifth vital sign, primary care physicians and specialists alike are being placed under scrutiny for excessive opioid prescribing. As of the time of this writing, states, including Washington, Utah, Ohio, Indiana, Kentucky, and New York, all regulate and monitor opioid prescribing carefully and this trend can be expected to continue.

Patients often become chronically habituated to opioids and underinsured after job loss, leading to further marginalization by institutions and practitioners, who of necessity are becoming more focused on competitive cost containment and the potential for clinical censure. These patients are often viewed as doctor shopping when they are unable to obtain adequate relief and most do not have good insight into the nature of their problem. Despite high doses of opioids, many patients still report 10/10 pain during clinical interviews. This clinical observation has been corroborated by a study from 2007 where opioids did not give patients a significant reduction in pain from baseline.\(^1,33\)

Therapeutic encounters need to emphasize that complete pain relief may not be reasonable, but pain control allowing increased activity and enjoyment may be attainable. In general, long-acting opioids should be preferred to short-acting opioids to decrease peaks and troughs that stimulate craving, and patients need to be monitored for aberrant drug behavior, including secondary financial gains from diversion. Patient education addressing the deleterious effects of prolonged immobility and deconditioning is essential if the vicious cycle of pain and deactivation is to be interrupted.

INTERVENTIONAL PAIN TREATMENTS AND SURGERY FOR TREATMENT OF FBSS

Interventional pain management techniques are frequently used to treat FBSS because pharmacologic interventions have significant morbidities of their own. More importantly, a ceiling effect is often reached before a patient has satisfactory relief. Some of these modalities, such as medial branch blocks, selective foraminal epidurals, and SI joint injections, offer further diagnostic insight. Others are clearly salvage techniques to minimize the deleterious effects of oral pain medicines, improve quality of life, or substitute for revision surgery. The justification for these procedures is derived from the extensive morbidity associated with FBSS and these patients experiencing permanent loss with respect to their earning capability, activity level, and life enjoyment. Treatment algorithms are designed to address either predominantly axial or radicular pain\(^1\) and are aimed at arthroidal or neural structures.

**Facet Interventions**

Zygapophysial joint injection, medial branch block, and radiofrequency neurotomy/ablation (RFA) are used to address axial back pain. When strict criteria are followed,
these procedures can provide important diagnostic information even if they are not therapeutically successful. Repeated successful diagnostic medial branch block followed by radiofrequency ablation of the facet joint(s) is generally preferred to intra-articular facet injection because of difficulty entering the joints, potential epidural spread, or venous uptake, which has been documented as occurring in as many as 6.1% of cases. Similarly, chemical medial branch neurotomy with alcohol or phenol is now discouraged because of potential epidural spread. A positive diagnostic response for medial branch block is 80% pain reduction after 2 blinded blocks with concordant responses. RFA should then be expected to provide sustained analgesia. Efficacy has been reported to offer 60% of patients 90% relief at 12-month follow-up, and 87% of patients have greater than 60% relief over a similar time span. Other investigators cite similar statistics, and previous surgery has not been found to have an effect on the efficacy of radiofrequency neurotomy.

Facet-mediated pain has been attributed to 16% of FBSS cases and RFA is now used widely throughout the United States. It does not offer a permanent fix to facet-mediated pain, which should raise concern with regard to overutilization. More importantly, there are other potential confounding variables that raise questions as to whether it is counterproductive. One of these is the physiologic effect of RFA on the multifidus muscle. This is important because the multifidus forms the posterior portion of the inner muscular ring responsible for spine stabilization. RFA lesioning is targeted proximal to the branch point for facet and multifidus innervations and, therefore, the muscle is denervated along with the facet. One method to determine whether RFA lesioning is effectively accomplished is to obtain a baseline EMG of the multifidus and then perform a repeat study 4 weeks after RFA lesioning. Denervation can be considered successful if there is a decrease of more than 90% muscle activity registered with the second study.

Retraining this muscle with motor control techniques is at the core of most presurgical or postsurgical spine rehabilitation strategies and therefore, premature RFA may be more damaging than helpful. RFA usually offers only temporary relief for approximately 10.5 to 12 months before the effect diminishes. Consequently, RFA should be reserved for the most refractory cases as a quality of life salvage procedure. This author has never seen a patient successfully perform either of the stabilization tests (described previously) after RFA. However, 100% of these patients were seen for refractory or recurrent pain that was predominantly axial.

**Epidural Steroid Injection**

Epidural steroid injection (ESI) is indicated for radicular pain. It is considered effective for epidural fibrosis, disk disruption, and spinal stenosis and, therefore, addresses several of the causes attributed to FBSS. Proposed mechanisms of action include an antiinflammatory effect by addressing phospholipase A2 elicited from disk material, sodium channel blockage, and an effect on vascular permeability. In a study comparing caudal ESI with just local anesthetic versus local anesthetic plus steroid, however, both groups had greater than 50% pain relief in 60% of the patients from either arm of the study. Functional improvement was noted in 55% to 70% of the patients, with no significant differences at 1-year follow-up. Understanding regarding the mechanisms responsible for the therapeutic effect of epidural injections is further questioned in recent literature. Bicket and colleagues evaluated randomized, double-blind studies comparing high doses of steroid with lower doses in which the steroid was replaced by saline or local anesthetic. This review is remarkable for a consistent failure to demonstrate any significant differences between treatment groups. They noted another systematic
review by Rabinovitch and colleagues where larger injectate volumes provided a statistically significant benefit over smaller injectate volumes irrespective of the injectate contents, suggesting several other mechanisms of action for the therapeutic effect of ESI, including suppression of ectopic discharges from inflamed nerves, enhanced blood flow to ischemic nerve roots, lysis of iatrogenic and inflammatory adhesions, washout of proinflammatory cytokines, and reversing peripheral and central sensitization.

Steroid delivery to the lumbar epidural space can be accomplished with reasonable certainty when the target pain generator is L4/5 or below with one of several approaches. These include translaminar, transforaminal, and caudal approaches. Fluoroscopic guidance is recommended to assist placement. The presence of epidural fibrosis, instrumentation, and other anatomic alteration after surgery makes needle placement more difficult and increases the risk of dural puncture, which has been reported as high as 20%. Even if the needle is placed properly, medication may not reach the pain generator due to scarring, fibrosis, and altered anatomy. Because the posterior longitudinal ligament is taken during laminectomy, loss of resistance technique is compromised with translaminar approaches at the operative level. In general, if this approach must be used, the next level caudal is used.

Transforaminal approaches are often cited as more effective than translaminar approaches because they access the anterior epidural space and are thought more selective relative to the disk and root exit zone in the foramen. They have also been assessed as more efficacious with respect to pain relief based on the Numeric Rating Scale. Dye flow to adjacent levels, however, is often seen with this approach and, therefore, specificity is questionable.

Caudal ESI offers a more global medication delivery and the roots forming the entire lumbosacral plexus up to the L4/5 level can be bathed at once. Large-volume caudal ESI has also been compared with epiduroscopy to treat epidural fibrosis. Epiduroscopy is a technique where a specialized RK needle is inserted percutaneously, allowing a Racz catheter to be threaded into the epidural space. The Racz catheter is then advanced to the area of fibrosis and injectate is then delivered focally to lyse adhesions. Other wire-bound catheters can also be used to mechanically lyse adhesions and there are several protocols and injectates that have been reviewed in the literature. These are specialized techniques not commonly performed in community settings. In contrast, caudal ESI is a commonly used technique throughout the United States.

Manchikanti and colleagues proposed a protocol in 2010 to evaluate the comparative effectiveness of large-volume caudal ESI versus percutaneous epiduroscopy and reported results in 2012 with a cohort of 120 randomized subjects who received multiple procedures over a 2-year period. There was a significant and dramatic difference between the 2 groups with respect to duration of relief after each procedure and a statistically significant change in pain and functional status was noted between the 2 groups. The caudal ESI cohort fared worse but benefit between 3 and 6 months was noted in both groups. Opioid decreases, measured in morphine equivalents, dropped from baseline in both groups but never extinguished, and no significant differences were noted between the 2 groups.

Evaluation of epiduroscopy by Takeshima in 2009 noted similar trends. Twelve weeks of improvement in the Roland-Morris questionnaire were noted whether the nerve roots were mechanically separated or not by the procedure, but better long-term improvement was noted if the nerve roots were mechanically separated by the procedure.

The evidence supporting the use of ESIs and epiduroscopy is controversial. There have been several summaries of the literature since 2005 with evaluations of the
strength of the evidence, and the consistent trends are that short-term relief is better than long-term relief for all modalities; acute pain and subacute pain are better relieved than chronic pain (ie, lasting greater than 3 months); and, overall, 60% to 70% have a good response of some duration and 30% have little or no benefit at all. These procedures are, therefore, at best, an adjunct in the treatment of FBSS and should not be expected to return patients to employment, decrease oral medication usage significantly over the long term, or affect disability in the setting of FBSS. The use of ESI is, therefore, at best limited as an adjunct to control exacerbations of radiculopathy in FBSS patients, allowing them to return to baseline functionality and medication levels.

Like medial branch blocks, ESI may also provide diagnostic insight. Consider a patient with postoperative persistent pain and radiographic evidence of foraminal compromise that correlates well with the clinical presentation. A foraminal block with alleviation of symptoms of any duration argues for a potentially reversible process, such as reherniation or incomplete decompression of the lateral recess. Therefore, reoperation with decompression may be a reasonable and definitive alternative as opposed to placement of a spinal cord stimulator for refractory postsurgical neuropathic pain.

**Sacroiliac Injections**

Si injections are the gold standard for diagnosing SI pain. SI pain is thought to occur between 16% and 43% postoperatively in the setting of lumbosacral fusion. It has been estimated that approximately 14.5% of patients presenting to spine surgeons before operation have SI joint pain. SI joint pain is important in the preoperative and postoperative differential because it is often associated with referred pain down the leg and can mimic radiculopathy. The general dictum that SI referred pain does not go below the knee is disputed. Schwarzer and colleagues contend that pain below the knee and into the foot are as common in SI joint pain as other sources. A potential confounding variable in this study is that when diagnostic SI joint blocks are performed, up to 24% of the time dye can be seen in sacral foramina. Physical examination testing and imaging are not considered reliable for diagnosing SI joint pain. In asymptomatic patients greater than 50 years old, 24.5% of were found to have abnormal SI joints on plain radiographs. Abnormal CT findings, such as sclerosis, erosions, and narrowing have a sensitivity of 58% and a specificity of 69% compared with pain relief from diagnostic local anesthetic injection.

SI joint pain has been attributed to ligamentous or capsular tension, shear forces, extraneous compression, hypomobility or hypermobility, aberrant joint mechanics, and myofascial or kinetic chain imbalances, resulting in inflammation and pain. Ivanov and colleagues postulated a model where fusion increased angular motion and stress across the SI joint and maintains that because the ligaments around the SI joint articulation are richly innervated, even small increases in motion trigger pain.

Innervation of the SI joint is derived from the lumbar plexus and sacral nerve roots. Radiofrequency lesioning can be performed at the L4/5 and L5/S1 dorsal rami along with the sacral foramina, at the joint itself, or with a combination of neural and ligamentous approaches. A 50% reduction in pain is considered therapeutic. Pain relief of 50% or greater has been reported to last from 6 weeks to as long as 12 months. SI joint injections with local anesthetic and steroid can be therapeutic but the duration of efficacy has been noted shorter in patients with a history of lumbar fusion. Like epidurals, SI joint injections show an approximately 60% success rate at decreasing preprocedure pain by 50% and, therefore, can be a useful adjunct in the
setting of exacerbated pain associated with FBSS. Diagnostic SI joint injections should be concordant and replicate or increase a patient’s typical pain with pressure loading of the joint, and at least 50% pain relief should be noted to correlate with the expected duration of the local anesthetic. Other strategies to manage SI joint pain include the use of pelvic belts and physical therapy and manipulation.52 The goal of physical therapy for SI joint pain, and all lumbosacral spine problems, is to restore motor control and postural and dynamic muscle balance, stabilize the pelvis, equalize flexibility, centralize referred and/or radicular pain, and correct gait abnormalities.52

**Intrathecal Pumps and Spinal Cord Stimulators**

With the exception of caudal ESI and epiduroscopy, most interventional procedures are aimed at specific structures. After these have failed, systemic pain control for quality of life can be augmented with implantable pain pumps or spinal cord stimulation (SCS). Revision surgery is usually not recommended unless a surgically correctable lesion can be identified because success rates are unusually low and continue to decline with each additional surgery. Successful outcomes have been reported in only 22% to 40% of patients who undergo revision surgery,1 leading to an expanded use of intrathecal pumps and spinal cord stimulators, which are less invasive and less costly and have an added advantage of being potentially reversible procedures.

Until recently, intrathecal delivery systems were reserved for cancer pain. The popularity of these systems, however, has increased for chronic nonmalignant pain.1 Analgesia has been found effective in FBSS and 88% to 92% of patients have reported satisfaction with these devices globally in several observational studies.1 Winkelmuller and Winkelmuller61 evaluated the therapeutic response relative to nociceptive somatic pain versus neuropathic pain. Short-term follow-up revealed better pain reduction in the nociceptive group at 77%. Some decrease in efficacy at long-term follow-up was noted, with only 68% achieving long-term results, whereas only 62% of the cohort with deafferentation pain achieved significant pain relief.1,61 In another study, a long-term reduction in total morphine dose of 23% was noted when comparing intrathecal opioid plus local anesthetic administration to intrathecal opioid alone.1,62 Two other reviews concluded that intrathecal delivery systems resulted in improvements for 30% to 56% of patients with better than 50% pain relief and function.1,63

None of these studies is a randomized controlled trial (RCT) and side effects can be formidable, including urinary retention, constipation, equipment malfunction, and catheter tip granulomas.1 Therefore, intrathecal infusion can be recommended only when all other viable options have failed and side effects from oral medications have become intolerable.1 Psychological evaluation and a trial with a temporary catheter are recommended before permanent implantation to assure effectiveness. Some patients may be unresponsive to intrathecal opioids if they have had to escalate their oral opioids because of tachyphylaxis.1

SCS has become a regularly used treatment of refractory FBSS. The proposed mechanism of action is via the gate control theory and modulation of neurotransmitter release in the dorsal horn.63 A subcutaneously buried pulse generator is connected to electrodes placed in the epidural space over the dorsal columns and the location, frequency, and intensity of the electrical stimulation is adjusted to provide coverage to the painful area(s).1,8,63 Results are considered better for radicular/neuropathic pain than axial/somatic/nociceptive pain.1

Several randomized controlled studies have influenced the upsurge in popularity for this modality. North and colleagues64 have published data based on self-reported pain relief along with patient satisfaction comparing SCS with reoperation for FBSS.2,65 The overall conclusion is that SCS is more successful than reoperation.64 The total number
of patients was 60 with cohorts of 30 in each arm of this RCT. What makes these data compelling is that the follow-up data extend to approximately 3 years and there was significant crossover to the SCS group from the revision surgery group.

Prospective Randomized Controlled Multicenter Trial of the Effectiveness of Spinal Cord Stimulation (PROCESS) compared SCS in conjunction with conventional medical management (CMM) to CMM alone. The population size was 100 patients and an outcome measure of greater than 50% pain relief was followed at 6, 12, and 24 months. Both groups showed improvement but the difference is noteworthy: 48% for the SCS group versus 18% for the CMM group. A 32% complication rate was also noted, however, in the SCS group at 6 months. Relative low morbidity complications can occur after implantation. These may necessitate explantation and reimplantation and include local wound infection, pain at the generator site or subcutaneously burrowed lead tract, and paddle or lead migration.

Higher morbidity complications that may be associated with the initial implantation itself include wet tap with spinal headache, total spinal block due subarachnoid injection, local anesthetic toxicity, epidural hematoma, midline or generator pocket seromas, and subsequent epidural abscess. Relative contraindications to SCS trial and placement include inability to pass the leads past scar tissue or scoliosis; untreated infection; presence of a cardioverter, defibrillator, or cardiac pacemaker; anticoagulation; and antiplatelet therapy. Absolute contraindications to implantation include pregnancy, previous dorsal root entry zone lesions, critical central stenosis along the lead pathway, serious neurologic deficit with surgically correctable pathology, anatomic spine instability at risk for progression, coagulopathy or immunosuppression, requirement for therapeutic diathermy, substance abuse, and severe cognitive impairment or failed psychological screening. The need for future MRI is also listed as an absolute contraindication and should be considered carefully in the decision-making process leading up to SCS implantation, especially in younger patients, given the likelihood of developing adjacent level disease over the course of a normal lifetime.

One of the factors leading to the increase in popularity of SCS in community settings is that it is considered cost effective, based on prospective modeling and assuming that the benefit is maintained over the lifetime. Kumar and colleagues noted in 2002 that 9 patients (15%) from the treatment group who received SCS plus CMM returned to some form of gainful employment compared with none of the patients in the control group, who received CMM alone. The average cost of drug therapy for pain (in Canadian dollars) was also noted to decrease from $78.00/month to $25.00/month after SCS implantation, whereas the average cost remained higher at $72.00/month over 5 years for the control group. Projected long-term modeling assigning economic quality-of-life cost values has also been performed in both Canada and the United Kingdom, with cost savings purported as an alternative to CMM or repeat surgery.

Two studies from the University of Washington, however, with a cohort of 51 workers’ compensation patients, question both the effectiveness and cost savings of SCS versus usual care (UC) or pain clinic care (PC). The investigators concluded that few patients in any group achieved success at follow-up with respect to less daily opioid use or improvement in leg pain and function. At 6 months, the SCS group showed modest improvement in leg pain and function but higher rates of opioid use, and by 12 months, these differences disappeared. In addition, 19% of the SCS group had their stimulators removed by 18 months and complications were noted with both trial and permanent implantations. The follow-up cost-effectiveness study maintained that the high cost of SCS was not counterbalanced by lower costs of subsequent care. The median cost for the SCS group in US dollars was $52,091,
which was $17,291 higher than the PC group and $28,128 higher than the UC group.70,71

Reconciling these disparities is not easy. The Kumar and North studies are randomized controlled studies with mixed population bases and were managed in university clinics. The University of Washington studies are population-based cohort studies and consisted of only workers’ compensation patients from community referrals with community-based management and telephone questionnaire follow-up. Ostensibly, these were higher-risk patients without as tight monitoring. Finally, all the patients in the Kumar and North studies underwent preimplantation psychological screening and only 25% of the University of Washington patients did so. Consequently, Kumar and North conceivably screened out patients who would have failed prior to implantation, thereby contributing to better outcomes. All these studies evidenced significant complication rates and the cohort groups are remarkably small, suggesting that the issues surrounding cost benefit are far from settled and that the increasing use of this modality for FBSS needs to be reassessed over time.

Currently, approximately 20,000 spinal cord stimulators per year are placed in the United States.72 Since introduced in the 1967 as a salvage procedure for refractory debilitating pain,8 its use is becoming widespread in community settings. At present, it is not clear whether best practice guidelines for implantation are adhered to and what the ultimate impact of this procedure on FBSS outcomes will be. Reliance on preimplantation psychological testing prior to stimulator implantation has not changed the significant rate of stimulator removal or failure rates seen in these studies.

A final disconcerting issue regarding the literature for SCS, and probably all interventional procedures for FBSS, is that many of the data are dependent on patient self-reporting of pain scales. The modest incremental changes in opioid use, the minimal return to work rate, and the statistically significant but not dramatic changes in disability scales and depression scales pose the question of whether the right questions are asked clinically both before and after initial spine surgeries.

THE RATIONALE FOR A GLOBAL APPROACH

Kelly and colleagues73 noted that “while subjective self-report assessment is vital in providing patient-centered opinion on treatment success, objective outcome measures are necessary to supplement these findings and quantify change.” Their publication reviewed multiple studies with respect to patient self-reports and measurable functional scales and concluded that most were level C evidence studies and, consequently, generalizations to large diagnostic groups must be interpreted with reservation. The lack of consistency between clinical observation and patient self-reporting has to be reconciled if the pain literature is meaningful. Supporting addenda to this article offer a summary of validated instruments for self-reporting as well as objective outcome measures of physical function that are available today.73

The problem with these instruments is that time constraints preclude their use in a nonresearch setting. Another problem is that there is less control of patient follow-through, and, in general, communication between clinicians is not well integrated in community settings. In addition, each clinician has a different vantage point: surgeons see a structural problem to be corrected; physical therapists see motor function and flexibility issues to address; manual medicine practitioners see motion barriers to remove; interventionalists see inflammatory cycles or pain patterns that need to be interrupted; and psychotherapists see anxiety and perceptual issues that undermine care. All have legitimate concerns.
Interventional and pharmacologic treatments are based on narrow disease/impairment models where patients are recipients of passive care. FBSS, like most presurgical low back pain, is a biopsychosocial phenomenon. Cost mitigation and clinical success have to be predicated on functional outcomes and specific goal setting, not just reported pain alleviation. Complete pain relief is not often possible either before or after surgery. Rehabilitation strategies must be individualized to improve physical function and reduce disability in the context of daily activities because improved physical function is linked to improved psychosocial function and mood. The overall goal is to interrupt the cycle of pain, guarding, anxiety, disuse atrophy, and deactivation commonly seen with these patients.

FBSS is a challenge to treat because there are multiple physical and psychosocial problems that have to be addressed. It is the prototypical chronic pain syndrome. It is poorly defined anatomically and there is significant overlap of the types of pain presentations. There is a disproportionate emphasis in the medical literature regarding drug, interventional, and surgical treatments and these options clearly have the most corporate support for research.

Interdisciplinary approaches have been shown to achieve clinical and cost-effective improvements in patient function, but they are perceived as costly. The Commission on Accreditation of Rehabilitation Facilities (CARF) recognizes and accredits outpatient interdisciplinary programs where different disciplines collaborate. Evaluation, goal setting, and treatment are all monitored as part of these programs and are much less costly than the traditional inpatient/outpatient centers of the 1980s. But they are few and far between.

The author has an affiliation with the only CARF-accredited interdisciplinary pain center within an approximately 100-mile radius of Cincinnati. The cost for this individually tailored 20-day outpatient program, including physical therapy, occupational therapy, and psychological support, is approximately $15,000 (Pain Solutions Network, personal communication, 2013). This is similar to the cost for a single-level laminectomy not requiring a hospital stay and is substantially less costly than the direct costs for lumbar fusion when hospital, hardware, surgeon, and anesthesia costs are added up.

As a chronic pain syndrome, FBSS demands a global set of clinical skills that is different from that needed for the evaluation and management of acute pain. Most therapies and interventions described in the medical literature are discussed with respect to back pain treatment and do not distinguish between presurgical or postoperative care. It is important that the evaluating physician evaluate the physiology and physical limitations of the back problem and not just the imaging. Treatment becomes dangerous when it is overly influenced by symptoms that are magnified by the prospect of job loss, the expectation of a perfect cure, or other psychosocial issues. A global evaluation of a patient’s life situation and a discussion of the morbidities associated with potential interventions are required, along with specific goal setting during every stage of treatment. A more appropriate question for patients is not, How is your pain on a 1 to 10 scale? but rather, What is your pain preventing you from doing?

Many clinicians and patients have a perception that if there is an abnormal imaging study associated with persistent back pain and radiculopathy, it requires a surgical evaluation. The rate of spine surgery in the United States is double that of Australia, Canada, and Finland and 5 times the rate of spine surgery performed in the United Kingdom. Population-based outcomes are not very different. One study found that in the state of Maine, the best results from spine surgery (pain and function) occurred in the areas with the lowest surgical rates and the worst outcomes occurred in areas with high surgical rates. Most back pain patients do not require surgery. Similarly, cost constraints should not preclude an interdisciplinary pain program trial,
if elective fusion surgery is being planned without clear-cut indications—especially for predominantly axial low back pain, given that the occurrence of FBSS in this population is between 30% and 46%.1

Prevention of FBSS requires setting clear expectations of what a proposed surgery offers:

- The kind of pain relief to expect—whether axial or radicular
- The expected healing time and reconditioning needs
- Activity limitations or work restrictions, along with their expected duration

In addition, some form of psychological assessment and treatment or vocational counseling should be offered both prior to and after surgery, if the expected outcome is not full return of function.

Multiple reviews of the literature have demonstrated that FBSS is ill defined and difficult to manage. In 1997, 317,000 lumbar surgeries were performed in the United States with a cost of $4.8 billion for the surgeries alone.1 In 2002, there were more than 1 million spine procedures performed in the United States and 400,000 were instrumented.1 Between 1990 and 2010, there was a 220% increase in the number of spinal fusions performed with no demonstrated increase in efficacy.4 The increasingly large influence of the cost of fusion surgery on total cost of care is reflected by the $16 billion for hospital charges alone that occurred with spinal fusions in the United States by 2004.1

Similarly, between 1994 and 2001, Medicare data revealed a 271% increase in the number of epidural injections and a 231% increase in the number of facet interventions performed in the United States without a concordant increase in the health status for Medicare low back patients demonstrated.50 Virtually every review of the literature regarding spine surgery and interventional procedures demonstrates significant treatment failures of approximately 30%. It should be clear that thinking about back pain and FBSS, in particular, is too parsimonious and that a more global approach is necessary.

In summary, there are several generalizations that can be made regarding pitfalls to avoid in the treatment of FBSS:

- First, interventional procedures should only be used to control flares of pain, returning patients to preinjection baselines; to allow them to partake in restorative physical therapy; or as diagnostic measures that may guide other treatments.

- Second, the role of pharmacotherapy can be expected to change because of increased recognition of opioid-induced hyperalgesia and the regulatory climate surrounding opioids.

- Third, although no specific physical therapy recommendations can be universal, it is clear that the complications of immobility and deactivation make restoration of function imperative.

- Finally, medical education is going to have to be broadened so that physicians learn to focus on functional performance and avoid assigning importance to terms that are diagnostically ambiguous, such as degenerative disk disease, or assigning significance to MRI findings without confirming correlation to pain complaints, loss of motor function, or bowel and bladder instability.

REFERENCES


