The clinician attempting to evaluate the patient with failed back surgery must be well acquainted with the many physiologic and psychological disorders that can cause problems. It is also important to be familiar with available diagnostic modalities and how to interpret them in light of the clinical picture.

The history and physical examination provide the information necessary to plan further evaluation and early treatment strategies. In most instances, the clinician will reach a working diagnosis and have an idea of the severity of the condition. Frequently there will be more than one disorder present. An impression of the degree of psychological augmentation of the peripheral stimulus can usually be gained during the initial interview. Estimation of the patient's strength and knowledge of body mechanics is usually possible by use of simple bedside tests such as timed partial sit-up and wall slide, and from watching the patient move around the examining room.

Most patients can readily be evaluated as outpatients. However, severely ill or disabled patients and those with obvious extreme psychological overlays will require close observation and invasive testing early in the evaluation. It is our impression that these patients are best served by a three- or four-day evaluation in the hospital. In this way the physician, physical therapist, and psychologist can see the patient frequently in a short period and come to a rapid consensus about future treatment.
The heart of our early diagnosis and early treatment program is body mechanics training and physical reconditioning. All FBSS patients require rehabilitation except those with severe neurologic deficits and a few who are suspected to have significant medical illnesses. Most patients with FBSS have become quite weak and deconditioned. It is also surprising how few patients that we see have had adequate training in body mechanics. The exact pathogenetic mechanism of FBSS almost does not matter. Training usually will come first. In addition, the response to training helps provide information about the diagnosis. The details of training will differ according to the working diagnosis. Some patients will need training and exercises emphasizing flexion, others neutral, and still others extension, depending on the cause of the FBSS.

Many patients will improve sufficiently after they have gained knowledge and strength to return to a normal life style. However, this will take a minimum of four to eight weeks. They then will need frequent follow-up visits to ensure continued compliance with their life-long training regimens.

Other patients will fail to improve after training. Some will have their progress impeded by pain or inappropriate pain behavior. These patients require further in-depth evaluation, including detailed assessment to evaluate the contributions of the peripheral stimulus and the psychological component. Other patients will show objective improvement in physical parameters but fail to feel better. These patients also require further work-up. Even when there is suspicion of a major psychological component as a reason for the failure to improve, the peripheral stimulus must be assessed.

The evaluation is directed at localizing the anatomic and biomechanical sources of pain. In general, a CT scan with multiplanar imaging and maximum resolution is necessary to fully evaluate the failed spine surgery patient.

If areas of tenderness, the location of the pain, or neurologic changes suggest that pain is arising from the upper lumbar levels, these should be included in the CT scan, or a myelogram may be needed to detect upper lumbar disease that refers pain to the lumbosacral level. All patients who have upper motor neuron signs, sensory changes involving the trunk, bowel or bladder dysfunction, multilevel neurologic changes, or neurologic changes out of proportion to the severity of pain should be evaluated for spinal cord tumors and primary neurologic disease. Water-soluble contrast myelography of the thoracic and lumbar spine with cerebrospinal fluid studies should be performed. Magnetic resonance imaging may prove to be a complication-free substitute for myelography in the future. If there are long tract signs suggesting upper motor neuron disease, the cervical spine should be included in the myelogram as well.

Epidural and/or selective nerve root blocks are used to isolate the level or levels of the peripheral stimulus. Symptoms should be relieved in a predictable manner with local anesthetic placed in the involved area. Because of postsurgical scarring and the variability of flow of medication, it is necessary to use fluoroscopy with a contrast agent to ensure proper placement. This allows confidence in the results of these tests. Lumbar epidural injections are used to assess adjacent levels above previous surgery and the upper portion
of the surgical site. Caudal epidural injections may be used to assess the lower portion of the site of surgery and levels below. If pain relief is not obtained by either of these, postsurgical scarring may have prevented the local anesthetic from reaching the source of pain. Selective nerve root blocks may be more effective and informative in this situation. Occasionally, the affected area cannot be penetrated by medication, and for this reason diagnostic blocks must be considered only one aspect of the total clinical picture.

Lateral x-rays in flexion and extension are evaluated for signs of hypermobility. Movement in horizontal translation, an angular difference greater than 15 degrees between flexion and extension, and approximation of the posterior vertebral bodies on either side of the disc space in extension may indicate instability. This may be clinically significant in the face of neural structures that are tethered by surgical scar. Lesser degrees of instability may still cause difficulty, yet not be apparent radiographically.

Discography with emphasis on determining a specific disc that reproduces the patient's pain has been extremely helpful in identifying the level responsible for the pain. Some patients have painful or radiographically abnormal discs from L1 to L5. It is difficult to know where to stop surgery when fusion is contemplated in this setting. Often, the choice between a large multi-level surgery and continuing conservative care must be made, realizing that significant disability is expected with either option. Dynamic discograms using flexion-extension x-rays may reveal posterior protrusion of the disc in flexion or dynamic leakage, which are not visible on the CT scan or plain discograms.

Low and normal doses of metrizamide followed by CT scanning are reported to better delineate some abnormalities not discernible on plain CT scans. Intravenous contrast CT scans may differentiate scar tissue from disc material in some cases, but we have not been impressed with their diagnostic usefulness. Discograms followed by CT scanning may reveal details of internal disc architecture not available with other tests, but their clinical significance is not well understood at this time. We have found none of these techniques to be of great advantage over a fourth-generation multiplanar CT scan of high quality resolution, but more research is needed in these various scanning techniques.

Electromyographic findings are often difficult to interpret in the post-surgical patient. Surgical trauma may result in chronic abnormalities that may or may not correlate with the symptoms. Paraspinal abnormalities are created by the surgical approach and therefore may not be diagnostically useful in the FBSS patient. Findings indicative of acute radiculopathy are very helpful, especially when present beyond several months from the time of surgery or when not present preoperatively. Serial EMGs may shed light on whether changes are chronic as a result of intraneural scar tissue or ongoing active compressive neuropathy.

In summary, we would emphasize that no one test or evaluation gives a complete picture of this multifaceted problem. Furthermore, none of these diagnostic procedures is completely reliable. Therefore, we are "blind men" trying to perceive the "elephant" from every angle, using every useful technique available to obtain the most accurate view.
DIAGNOSTIC STRATEGIES—OVERVIEW

The history and physical examination may reveal whether a significant psychogenic augmentation or overlay is present. In the history, an abnormal pain drawing, psychological difficulties described by the patient, a history of symptoms not expected on the basis of structural pathologic abnormalities, and other indicators will help place the patient into a category of suspected psychological overlay. Pain behavior noted on physical examination, such as overreaction (grimacing, moaning, etc.) to maneuvers that might be painful (straight leg raising, etc.), subjective sensations and pain distributions that are not anatomic, and inconsistencies such as a great discrepancy between supine and sitting straight leg raising, also indicate significant heightening by psychological factors of the sensations induced by the peripheral stimulus.

Patients who apparently have psychological augmentation are evaluated further to judge its extent. Three tests are used in this regard. In those patients who have pain that can be evoked on physical examination by a maneuver such as straight leg raising, an examination-interview with methohexital is performed. This will give a rough impression as to what proportion of the perceived pain is from a peripheral stimulus and what proportion arises from psychological augmentation. This is a new test which has not been fully quantified, but studies are in progress to establish its validity. We consider it to be one more factor in the context of all the other pertinent tests and evaluations.

A second test is the use of an indwelling catheter placed in the epidural space at the site of the suspected abnormality. The patient receives injections of a placebo (normal saline), and then local anesthetic agents. The subjective pain level and objective components of the physical examination are carefully monitored before and after each injection. Relief from placebo means only that the patient is a placebo responder, and does not necessarily imply psychological overlay. However, response to the placebo makes it very difficult to interpret subsequent injections of the local anesthetic. No relief from placebo and relief from local anesthetic suggest a peripheral structural lesion. No relief from either placebo or local anesthetic may mean psychogenic pain or the inability of the local anesthetic to penetrate the area where the pain is produced. The block is repeated using a different approach if there is any question as to penetration of the agent into the affected area.

Last, detailed psychological interviews and testing must be performed. We have found it somewhat helpful to perform these tests on an inpatient basis. This allows observation of the patient under stressful conditions, and often reveals information about his or her psychological make-up that cannot be obtained in an outpatient setting.

If psychological factors are shown to be the principal component of the problem, the patient is placed on an appropriate pain control and rehabilitation program. If the patient has both a significant peripheral stimulus and psychological augmentation, the same rehabilitation program is recommended. In this circumstance, it is appreciated that after rehabilitation the patient may or may not improve enough to be able to avoid surgery. However, we have found that surgical outcome is far better if pain behavior is gone, social problems have been dealt with, and physical strength is max-
imized. Improvements in these reversible aspects is a prerequisite before surgery can be considered. Many patients reach a satisfactory level of function with diminished pain without surgery. Rarely, patients are not trainable because of the severity of disease, and will need surgery prior to rehabilitation.

SPECIFIC EVALUATION

Details of the evaluation will depend on the suspected diagnosis. Instability, spinal stenosis, recurrent disc herniation, missed pathology, facet syndrome, internal disc disruption, soft-tissue dysfunction, neural damage, arachnoiditis, fibrosis, pseudarthrosis, pseudomeningocele, sympathetic dystrophy, and hardware irritation must each be considered.

PATHOLOGY

Identifying an exact source of a patient's low back pain is very difficult, even in the non-operated patient. We look for the mechanism of injury, aggravating and relieving factors, physical examination findings, and as many tests as possible that confirm a single diagnosis. In the failed spine surgery patient, there are frequently many possible sources of pain. Usually, more than one site of nociception is participating in the patient's clinical syndrome.

There are usually many non-spinal factors feeding into the patient's pain presentation. General physical deconditioning with joint stiffness and postural strain can produce many areas of pain unrelated to the spine. The patient's psychological state and general pain tolerance related to endorphins can alter the subjective intensity of the pain.

In the following sections, we describe specific diagnoses that can be identified in failed back surgery syndrome patients. Rarely is any one of these entities clearly identifiable as the sole source of the patient's pain.

INSTABILITY

Frymoyer and Selby state that “The greatest challenge is to identify segmental instability as one of the many manifestations of spine degeneration.” Frymoyer has shown the 20% of women who have undergone lumbar disc excision without removal of facet joints show signs of instability, usually at L4–5. Levels adjacent to fusions are frequently seen to be unstable radiographically and appear unstable clinically. Paris has reported that complaints of “giving way, slipping out and twisting back into position” are definitely due to instability. Back pain usually predominates over leg pain. In our experience a history suggestive of instability usually involves increasing pain when static positions as sitting, standing, and especially driving are maintained. Patients are often unable to rise quickly from a sitting position because of increased back discomfort when arising which resolves after a brief period of time. Patients with marked instability are often unable to reverse their lumbar lordosis without pain. In some patients there may be
less severe symptoms during activity as multidirectional forces are applied to the affected segment. More symptoms are present with static positions and unidirectional forces.

On physical examination, repetitive movements such as repeated flexion while lying down or repeated flexion while standing will reduce the pain. Some patients may have relief with lumbar extension positions. There is often pain on return from forward flexion to a neutral position. In most cases, tenderness is present posteriorly over the facet joints and increased motion of the segment can sometimes be palpated directly.

Increased angular and/or translational motion of a vertebral segment is looked for on flexion and extension x-rays. Oblique views may be obtained if spondylolysis is suspected. When clinical and physical examination are consistent with instability but gross instability cannot be seen on dynamic x-rays, we still consider the patient to have clinical segmental instability. The amount of motion a segment can tolerate would appear to depend on the neuroforamen size, residual soft-tissue structures such as the annulus, and inflammation and irritation of the facet capsule. A small amount of mobility may be enough to overload a diseased segment in a patient who has been operated on.

If the patient has not responded to treatment for instability or has previously had a trial of this treatment, a multiplanar CT scan and EMG examination are performed. If the correct diagnosis is primarily instability, the CT scan may be relatively normal, usually showing broad-based disc bulging. The EMG may show subtle abnormalities or be normal. At this stage, an epidural block may provide the patient with temporary relief and allow him or her to increase efforts at strengthening and mastering stabilization without aggravation of pain. A lumbosacral corset or body jacket may help the patient carry out activities of daily living without causing increased pain while he or she is on a progressive strengthening program. If the patient has not become very strong and has not mastered the techniques necessary to maximize function and minimize symptoms, a full inpatient or outpatient rehabilitation program may be considered. In our center, this consists of eight hour-a-day sessions of physical therapy, strengthening, pain control, and work-hardening techniques, followed by outpatient therapy visits of decreasing frequency over a period of several months. If the patient is still unacceptably disabled, and has demonstrated improvement in strength and body mechanics, we consider further evaluation for possible surgery.

When the patient is considered for reoperation, psychological evaluation is routinely performed if it had not been done previously. An indwelling epidural block should bring a predictable and physiologic response. Discography is performed at the level of the suspected pathologic process and also at adjacent levels. We recommend fusion in most failed spine surgery patients and in all patients with instability. Fusion will result in increased stress risers at adjacent discs, and it is important to know the internal architecture of adjacent discs and whether or not they are painful. Those that are painful are considered abnormal and may be incorporated into the fusion mass and not left unprotected.
SPINAL STENOSIS

Pain, usually in the buttock and lower extremity, increased by walking and standing and relieved by sitting, is the typical complaint of the patient who has either lateral or central stenosis (present in 94% of patients with stenosis who have not been operated on). The same symptom pattern can also be seen in disc herniation, although it is not typical. In stenosis, lower extremity pain predominates. The patient may report greater ease in walking uphill (lumbar flexion) than downhill (lumbar extension). Some patients report that sleeping in a chair is more comfortable than sleeping in a bed.

Physical examination will reveal intensification of symptoms by all positions of lumbar extension. The lumbar lordosis will be involuntarily flattened in most patients. Neurologic deficit and nerve tension signs may or may not be present, depending on the severity of the stenosis.

X-ray findings will usually reveal a degenerative segment with hypertrophic facet changes. Short pedicles may suggest an element of congenital stenosis.

These patients should have a six-week trial of pelvic tilting, lumbar flexion, and abdominal strengthening. Orthoses are often somewhat helpful.

If this regimen fails to alleviate pain adequately, CT scanning and EMG are performed to confirm the diagnosis. In older patients, it is not uncommon for stenosis to involve many segments, and they may not be adjacent. For this reason CT scanning of the entire lumbar spine or myelography is necessary to screen for multilevel disease. The EMG reveals neuropathy in most cases, and is more sensitive than physical examination.

When these studies confirm the diagnosis of stenosis, further conservative care may include epidural corticosteroid injections, and if these are unsuccessful, selective nerve root injections. A prolonged period of relief with epidural steroids is more common in older patients. A body jacket may be considered as an aid to stabilize the spine and control symptoms during an intensive strengthening program. In our experience, stenosis does not generally improve with time, but may stabilize. If the patient has been shown to be free of significant psychopathology and the pain is refractory to conservative care, surgery should be performed.

RECURRENT DISC HERNIATION

The history of a patient with recurrent disc herniation includes a variable period of postoperative relief, with subsequent onset of new pain or pain similar to the preoperative pain. Symptoms are usually mechanical in nature, and are worse with loading, standing, and sitting.

Physical examination is not specific and resembles the examination of the patient with a non-operated herniated disc. Nerve tension signs are
usually present. Segmental splinting and muscle spasm are common. Neurologic deficits may or may not be present. The pain is less responsive to mobilization and postural exercises than a non-operated disc herniation.

Radiographs are likewise nonspecific, revealing limitation of motion, often most pronounced at the involved segments.

If there is no neurologic deficit and the patient has not had adequate education in strengthening and body mechanics, a six-week outpatient training trial should be instituted. If the disc herniation is at the level of previous surgery, conservative care is not often helpful.

Multiplanar CT scanning and EMG should then be performed. The CT scan will reveal a lesion or a suspect area of scar tissue versus disc, versus a combined lesion. Metrizamide CT scanning, intravenous contrast CT scanning, or discogram–CT scanning may be considered to augment resolution. The EMG is most diagnostic if acute neuropathic changes that correspond to the clinical picture are present.

Further verification and occasional prolonged symptomatic relief may be obtained by epidural and selective nerve injections. Psychological evaluation and testing should be obtained when the patient is refractory to initial conservative care.

When the patient is not greatly disabled by symptoms and is in poor physical condition, a trial of intensive rehabilitation may be considered, with or without the aid of a corset or body jacket. When the patient is in great distress and greatly disabled, reoperation should be performed. Discography assessing the integrity of adjacent levels preoperatively is useful in planning whether to fuse and which level should be fused.

**MISSED PATHOLOGY**

When an operation is followed by period of relief, it is difficult to differentiate a missed pathologic process from recurrence of the original problem. This is because the placebo effect of surgery may persist for many months. Patients who obtain no relief of symptoms postoperatively must be suspected of harboring a pathologic process which was overlooked at surgery. Old records and x-rays must be reviewed to assure both that the surgery was performed at the correct level and that coexisting disorders at other levels were not overlooked. Common causes of missed pathology have been discussed in previous chapters and include subarticular stenosis, foraminal stenosis, midline disc herniation, foraminal disc, migrated discs, “far-out” syndrome, and subtle disease at other levels, such as internal disc disruption. Burton et al. found that 56% of 225 patients with the diagnosis of herniated disc also had lateral spinal stenosis at surgery. They found similar results in their review of 500 CT scans of patients with low back pain. Lateral stenosis should always be considered.

If the patient has not had adequate experience in back school, education, and strengthening, aggressive conservative care should be instituted as the first order of treatment. Patients should have psychological evaluation. Multiplanar CT scanning, EMG, and selective local anesthetic injections will reveal most missed pathologic processes.
When a specific diagnosis has not been reached using the above-mentioned procedures, discography should be considered. It has been indispensable in our experience as well as that of others. Discograms may reveal painful segments which have not been found by all other tests. Bone scans may occasionally show discitis or neoplasms, and should be obtained any time one of these diagnoses is suspected. A myelogram or MRI scan should be performed in the presence of thoracic or thoracolumbar pain, upper motor neuron signs, multilevel neurologic involvement, trunk sensory abnormalities, neurologic deficits unexplained by other studies, and severe neurologic deficits with minimal pain.

**FACET SYNDROME**

Pain from facet arthropathy is usually limited to the back and buttock region. Localization is often vague, and this corresponds to the multisegmental innervation of the zygapophyseal joints. Increased pain in positions that load facet joints (standing, walking, and twisting) is characteristic. A lag period of one to 12 hours before the onset of pain or an increase from baseline pain after exacerbating activities is often noted. Downhill ambulation and ambulation in high-heeled shoes is associated with increased symptoms because of increased facet loading in lumbar extension. Complaints of clicking and popping are not uncommon and may also suggest segmental instability. These two entities often coexist and are considered synonymous by some. We separate them because in our experience the treatment is somewhat different when most of the symptoms arise from the facet joint.

Physical examination will reveal tenderness and reproduction of symptoms by palpation of the facet joints. Extension and rotation of the lumbar spine increases the facet joint load and results in increased pain. Conversely, flexion while lying down is usually relatively comfortable. Range of motion is more limited in extension than in flexion. Nerve tension signs and neurologic abnormalities should not be present.

Plan x-rays may reveal degenerative and sclerotic changes of the facet subarticular bone. Disc narrowing is often seen, and gross instability may or may not be present.

Initial treatment should include nonsteroidal anti-inflammatory agents and a lumbar stabilization program of education and trunk strengthening exercises within a limited range of motion. A soft orthosis may reduce symptoms by immobilizing and possibly unloading the spine. If exercise alone is unsuccessful, facet joint corticosteroid injection should both confirm the diagnosis and allow prolonged improvement in some cases.

When symptoms persist, CT scanning should be performed to rule out other causes of symptoms. Psychological evaluation and an intensive rehabilitation program should be attempted before invasive treatment is considered. Facet rhizotomies sometimes result in prolonged relief, but results have not been consistent. If pain persists, discograms should be obtained in an attempt to identify coexistent discogenic pain. For patients who have mostly posterior-element pain, it is highly unusual to have to resort to a surgical solution.
INTERNAL DISC DISRUPTION

Awareness of this entity (called IDD) is very important, as it is frequently overlooked. The history is often classic. A traumatic episode usually precedes symptoms in a young adult. Crock states that back pain is prominent, having the character of a deep-seated dull ache.\(^1\) There may be referred limb pain, but it is poorly described and widespread. It is usually unresponsive to physical therapy, mobilization and traction. Frustration is common in these patients, who may be markedly disabled by their pain, but have not been accurately diagnosed by previous physicians. Nausea, weight loss, and psychological abnormalities are common according to Crock.

Physical examination presents a picture similar to that of instability. Extension from a flexed position is painful. No neurologic abnormality is seen, but straight leg raising usually causes back pain toward the end of range of motion. Pain behavior is frequently seen. Anterior spine tenderness to abdominal palpation and sometimes back pain on abdominal palpation are present over the involved disc. Radiographs are unrevealing.

Some patients seem to improve with gradual trunk strengthening, an orthosis and time. This should be the first order of treatment. If no headway has been made after six weeks, further work-up is in order. CT scanning should be performed to assess the operated and unoperated levels. IDD by definition does not occur at a level previously operated on, but an incompletely removed disc can create the same picture. Discography should reproduce pain at the involved level(s) and cause minimal or no pain at other levels. The EMG is normal. Psychological evaluation and treatment is often helpful for further patient assessment and support. Epidural steroid injections may temporarily alleviate the pain.

If the above-mentioned tests confirm the diagnosis of internal disc disruption at one of the non-operated levels, we prefer to try an intensive rehabilitation program for a period of four to six months. If symptoms persist despite patient cooperation, surgery is offered as an option. Crock considers anterior interbody fusion to be the treatment of choice. However, whatever procedure is used, a complete discectomy with fusion is needed for optimal results.

MYOFASCIAL PAIN SYNDROMES
(SOFT-TISSUE DYSFUNCTION)

The soft tissues in and around the spine may be a source of pain in the patient with failed back surgery syndrome. The pain is often diffuse but at times may be localized to a single muscle group, especially the paraspinal musculature or the area around the posterior superior iliac crest. If trigger points are present, there may be nondermatomal, nonsclerotomal referral patterns.\(^2\) Pain is described as an ache or tightness. Tingling can occur in the area of distal referral. Pain is often increased by static positions and may de-
Diagnostic and Therapeutic Strategies

crease with rest or moving around within limits. Frequently patients report temporary relief with heat.

Some physicians feel that soft-tissue dysfunction is a primary cause of pain in FBSS. However, it may be more likely that soft-tissue pain is secondary to an underlying stimulus in the spine which activates muscle spasm and pain. Equally likely is pain from deconditioned muscles which are overworked in order to maintain postural correction.

Treatment proceeds in a stepwise fashion. Symptomatic control is accomplished by the use of ice, TNS, or massage. There is a role for injection of focal trigger points as well. However, each of these modalities will provide only short-term relief. Travell points out the need for an ongoing program to stretch muscles that are tender or in spasm or have certain trigger points. The patient should be instructed in a home stretching program.23

The second line of therapy is a combined program of body mechanics training and strengthening. This will also include work on postural correction to prevent rapid deterioration after symptomatic gains have been made.

There is very little to be gained by the addition of muscle relaxants. They have little or no direct peripheral muscle activity, act primarily on the central nervous system, and may induce dependency.

Patients who fail to improve symptomatically despite noticeable improvement in flexibility, strength, or body mechanics deserve more thorough evaluation, looking for an underlying cause of sustained soft-tissue dysfunction. Nearly any cause of FBSS can have a secondary soft-tissue dysfunction. Very often the underlying problem must be addressed before gains can be made.

NEURAL DAMAGE

Damage to a peripheral or central nerve can cause pain in the area of the body served by that nerve.17 Pain persists despite the fact that the lesion that caused the nerve injury has been removed. The pain state is no longer dependent upon the presence of a nociceptive stimulus. Nerve damage may have occurred from a prior disc herniation, or compression of the nerve in a stenotic canal or foramen or during surgery. Of course, not all patients with similar lesions develop a chronic nerve injury pain syndrome, often called deafferentation.

Pain is often severe. It is usually described as a constant dull ache with superimposed burning or electric shock sensations. Tingling, numbness and paresthesias are also described. The distribution often resembles that of the pain experienced preoperatively.

Physical examination is often not helpful. Sensory examination will often show a partial or complete loss of appreciation of pinprick to light touch. Some patients will show hyperpathia to pinprick. A small number will have no sensory changes.24 Diagnostically, the pain is relieved by local anesthetic blocks and also by sodium thiopental.24

Deafferentation pain is very difficult to treat. The patient should have
other causes of pain sought and corrected. Training is important. It is unlikely to decrease pain, but may certainly increase function.

Narcotic analgesics have no role. Tricyclic antidepressants such as amitriptyline help some patients. Carbamazepine may decrease the jolts of severe pain. TNS is helpful in a few patients. There is a role for a trial of sympathetic blocks, which also help some patients.

ARACHNOIDITIS AND FIBROSIS

Arachnoiditis is poorly understood. Arachnoiditis and fibrosis can present in a variety of ways, and there are only a few consistent symptoms. There are no consistent physical findings. As a clinical entity, there is not even any proof that these disorders are painful.

Arachnoiditis has been described many times, as long as 50 years ago. Most authors state that arachnoiditis produces leg pain, frequently with neurologic deficits, and occasionally with bowel, bladder, and sexual dysfunction. Arachnoiditis has classically been diagnosed by its typical myelographic pattern. Repeat surgery is rarely, if ever, of significant benefit. In our spine center, we make the diagnosis more by exclusion of other diagnoses than on the basis of a strong set of physical findings. Arachnoiditis can appear on myelograms and be totally asymptomatic. It is all too easy to blame a patient's pain on arachnoiditis or fibrosis and be missing the more significant underlying condition of instability or stenosis. Therefore, even though we do see the scar tissue or arachnoiditis on myelogram, CT scan or MRI, we do not make the diagnosis final until all of the previously discussed diagnoses are ruled out by appropriate diagnostic measures and all the conservative treatments that have been mentioned have been accomplished.

A diagnosis of arachnoiditis, therefore, is ultimately arrived at when a patient with mostly leg pain has failed to obtain relief from previous surgeries. The pain is more constant than the "mechanical" type of pain of instability, stenosis, and recurrent disc herniation. The patient frequently feels as if there is a band or tight restriction around some area of the lower extremity. There are often burning paresthesias of a relatively constant nature. None of the conservative measures mentioned for other entities changes the clinical pattern. Corsets, braces, manipulations, exercise, back school, etc., do not significantly alter the pain. Pain control measures such as acupuncture, biofeedback, and medications can, of course, alter the central pain response.

Physical examinations for arachnoiditis and fibrosis are also variable and inconsistent. There is frequently a neurologic deficit, often of more than one spinal nerve with arachnoiditis; with fibrosis the pattern is more consistently at the level previously subjected to surgery, and the fibrosis can be demonstrated on CT scan or MRI. There are often nerve tension signs. Dynamic evaluation of the lumbar spine with flexion and extension, active and passive, does not alter the pain significantly or give a specific pattern. The EMG will usually demonstrate chronic abnormalities, as discussed in a preceding chapter.
The treatment of arachnoiditis is mainly pain control. If one is fortunate enough to be able to identify arachnoiditis in its early stages because of admixture of a myelographic contrast agent with blood, removal of the irritating substance might prevent ongoing arachnoiditis. Otherwise, once arachnoiditis has been well established, surgery is of little value, unless one is resorting to surgical rhizotomies or spinal implants for pain control.

Epidural fibrosis is frequently associated with recurrent herniated disc, stenosis, or instability. In such cases, surgery is frequently of benefit, and release of some of the fibrosis, with removal of the recurrent disc herniation or stabilization of the unstable segment, can give many rewarding successes.

To conclude, we must reiterate that fibrosis and arachnoiditis may not really be the source of significant clinical pain syndromes. Most of the patients who come to our clinic with the diagnosis of "scar tissue" or arachnoiditis have been relegated to those diagnoses prematurely in the face of other underlying pathology that we are able to diagnose and treat successfully. We would prefer that these diagnoses not be used except by those who have the capabilities of ruling out all other possible causes of pain and providing all forms of conservative care.

**PSEUDARTHROSIS**

A history of fusion and symptoms and signs of instability suggest a symptomatic pseudarthrosis. Radiographs may show motion and poor graft consolidation. Treatment should be conservative, if possible for at least a year from the time of the surgery, and perhaps longer when interbody fusions have been done. If the patient smokes, this must be stopped. Osteoporosis should always be considered, and a dual-photon absorption scan or quantitative CT scan should be obtained. If osteoporosis is present, the metabolic cause must be corrected in order to facilitate fusion with reoperation. Education and strengthening may improve function. A soft corset or body jacket is usually useful in controlling symptoms. Corticosteroids may also provide temporary symptomatic relief, although they have a deleterious effect on osteoporosis. Multiplanar CT scanning extending to adjacent levels and EMG should be performed prior to any surgical procedure, as the pseudarthrosis is often not the sole source of symptoms. Levels adjacent to the fusion sites must be closely evaluated with selective injections and discography to assure that they are not involved in pain production. As in all cases of failed surgery, psychological evaluation should be done prior to any reoperation. Most recently, promising results have been obtained with pulsed electromagnetic field stimulators.

**PSEUDOMENINGOECELE**

The diagnosis of pseudomeningocele should be suspected when postural headache and/or back symptoms are present in the postoperative
patient. These usually occur early in the postoperative period. In most cases a dural tear was appreciated at the time of surgery. However, these tears are not always detected during the procedure. A headache is usually centered in the occipital area and frontal area and is always worse in the upright position. Later in the postoperative period, the headache often resolves. However, back and leg pain may become chronic. CT scanning is accurate in revealing an area of slightly lower density posterior to the thecal sac. Metrizamide myelograms may show entry of dye into the pseudomeningocele, or the meningocele can be injected if possible, showing entry into the thecal sac. A lesion that causes symptoms should be removed and the dura repaired. We have seen meningoceles overlie nerve roots, causing direct pressure and acute radiculopathy.

**HARDWARE IRRITATION**

In most cases of hardware-induced symptoms a mechanical pattern is present, with more severe symptoms occurring with increased activities. Back and/or limb pain may be present, but the latter is more typical. Sacral hooks may cause groin and perineal pain referral.

Physical examination may reveal almost any constellation of findings, but there is always tenderness over the area of the offending posterior hardware.

Hardware-induced symptoms are less common in the face of solid fusion. Motion on bending radiographs indicates both the likelihood of hardware irritation and pseudarthrosis. Since solid fusion may be associated with alleviation of symptoms associated with hardware motion, one should delay reoperation if possible for a year postoperatively. Stabilization and body mechanics training may help the patient control symptoms. A rigid body jacket may help reduce motion and pain.

Epidural and selective nerve blocks are useful, both diagnostically and therapeutically. Since CT scans are greatly distorted in the presence of hardware, the surgery site cannot be well visualized. Selective injections should demonstrate reproduction of symptoms during the injections and relief from local anesthetic if the pain-generating region is located. Psychological and pain evaluation testing should be performed. If the tests indicate primarily a peripheral source of pain, hardware should be removed and any pseudarthroses repaired. Exploration of the neural elements adjacent to the area of apparent hardware irritation should be performed.

**SYMPATHETIC DYSTROPHY**

Although it is not common, reflex sympathetic dystrophy (RSD) can occur in association with disorders of the lumbar spine, and can be present in the failed back surgery syndrome patient. It is very treatable and should be remembered in differential diagnosis.
Indications of vasomotor instability in the lower extremities such as complaints of ipsilateral hotness (early disease) or coldness (more advanced disease) associated with a burning hyperesthetic pain are the usual complaints. In most cases RSD symptoms occur simultaneously with a structural lumbar spine lesion, adding to the symptoms arising from the latter. Skin hypersensitivity and complaints of pain with any motion of the limb may occur in more advanced disease. A previous history of extremity coldness, increased sweating, or Raynaud's phenomenon may alert the physician to a pre-existing sympathetic lability and tendency to RSD syndromes.

Physical examination reveals a range of findings, depending on the severity and duration of involvement. Minor involvement may be subtle, but a clinically significant part of the patient's symptoms and should be looked for. Increased warmth, hyperhidrosis and visible discoloration are present in earlier stages. Tenderness may be present, especially when there has been a period of disuse. Skin hyperesthesia is characteristic though not specific. In advanced cases, coldness, swelling, stiffness, and trophic skin changes occur.

Radiographs of the involved limb will reveal minimal to severe demineralization. Bone scans show ipsilateral increased uptake. In our cases there has been a primary structural lesion in the lumbar spine associated with the RSD. Such a lesion should be evaluated and treated as completely as possible. This will facilitate control of the RSD symptoms. Complete diagnostic testing as discussed in the other sections should be performed. It may be useful to treat both the structural spine lesion and the RSD simultaneously, or the structural lesion first if the RSD is not progressing rapidly.

When much of the symptoms appears to be due to RSD, lumbar sympathetic blocks are performed. This will result in relief proportional to the amount of symptoms due to RSD. Injections are repeated when symptoms return unless the periods of relief after injections do not show any increasing trend. Alpha-blockers such as phenoxybenzamine and calcium channel blockers such as nifedipine are effective in some cases. The dosage must be titrated carefully since the therapeutic effect is often close to intolerable postural hypotension. When the drug has successfully controlled RSD symptoms for several weeks, discontinuation may be attempted.

When local sympathetic blocks and chemical alpha-adrenergic blockades have not been successful enough, then chemical sympathectomy or surgical sympathectomy may be considered. We have performed the former using phenol solution under CT scan control, with satisfactory results in a small number of patients.

DIAGNOSTIC TREATMENT ALGORITHM

Figure 1 shows diagnosis and treatment flows according to eight diagnostic and/or treatment modules. Patients are initially evaluated by history, physical examination, and bending x-rays. The examiner will be able to place all patients into one of three general diagnostic categories: A, struc-
tural disease; C, central nervous system augmentation; or B, both. Patients then progress to appropriate modules. Diagnostic categories may change according to the experience in each successive diagnostic or treatment module. For example, a B patient may drop his inappropriate pain behavior in the spine rehabilitation program (module 7) and become an A patient. Or a B patient may be reconsidered a C patient after further observation during back school physical therapy (module 2) reveals gross psychopathology that may not have been appreciated on initial evaluation.

Even patients with structural abnormalities only (A) may be able to benefit from intensive spine rehabilitation if their levels of disability allow them to perform and they have room for further improvement in insight, strength, and body mechanics.
REFERENCES