TRAUMA OF THE SPINE AND SPINAL CORD. PAIN IN THE BACK, NECK AND EXTREMITIES
TRAUMA OF THE SPINE AND SPINAL CORD.

Throughout recorded medical history, signal advances in the understanding of spinal cord disease have coincided with periods of warfare. The first thoroughly documented study of the effects of sudden total cord transection was that of Theodor Kocher in 1896, based on his observations of 15 patients. During World War I, Riddoch, and later Head and Riddoch gave the classic descriptions of spinal transection in humans; in France, Lhermitte and Guillain and Barre made additional observations. World War II marked a turning point in the understanding and management of spinal cord injuries. The discovery of antibiotics, and rapid and efficient means of transportation permitted the survival of unprecedented numbers of soldiers with spinal cord injuries and, incidentally, provided the opportunity for long-term observation. In special centers, such as the Long Beach, Hines, and West Roxbury Veterans Administration Hospitals in the United States and the Stoke Mandeville National Spinal Injuries Centre in England, the care and rehabilitation of the paraplegic were perfected. Studies conducted in these centers have greatly enhanced our knowledge of the functional capacity of the chronically isolated spinal cord.

Mechanisms of Spine and Spinal Cord Injury

canal and damages the spinal cord directly. In some the missile strikes the vertebral column without entering the spinal canal but virtually shatters the contents of the dural tube or produces lesser degrees of impairment of spinal cord function. Rarely, the transmitted shock wave will cause a paralysis of spinal cord function that is completely reversible in a day or two (spi*nal cord concussion).* This latter condition may also be produced by forceful falls flat on the back, as occurs not infrequently in athletes engaged in contact sports or in falls from a ladder. Little known of the underlying pathologic changes. The term *concussion* as applied to spinal cord injury, has led to much confusion because it has been applied indiscriminately to a variety of minor or partial spinal cord injuries in addition to the completely and rapidly reversible form of spinal cord paralysis.

Pathology of Spinal Cord Injury

As a result of squeezing or shearing of the spinal cord, there is destruction of gray and white matter and a variable amount of haemorrhage, chiefly in the more vascular central parts. These changes are maximal at the level of injury and one or two segments above and below it. Rarely is the cord cut in two, and seldom is the piarachnoid lacerated. The condition is best designated as *traumatic necrosis of the spinal cord tissue.* Separation of pathologic entities such as hematomyelia, concussion, contusion, and hematorrhachis (bleeding into the spinal canal) that are concomitant is of little value either clinically or pathologically. As a lesion heals, it results in a gliotic focus or cavitation with variable amounts of hemosiderin and iron pigment. Progressive meningeal fibrosis and a tension syringomyelia sometimes develop and lead to a delayed central cord syndrome.

In most traumatic lesions, the central part of the spinal cord with its vascular gray matter suffers greater injury than the peripheral parts. And in some instances, the lesion is virtually restricted to the interior and posterior gray matter, giving rise to segmental weakness and sensory loss in the arms with few long tract signs. This has K called the *central cervical cord syndrome* (or Schneider syndrome, see further on). Fragments of the syndrome are not uncommon as transient phenomena that reverse over several days.

In most lesion, the total clinical effect consists of an irreversible structural component and a reversible disorder of function, each of which may vary in degree.

Clinical Effects of Spinal Cord Injury

When the spinal cord is suddenly and completely or almost combed, three disorders of function are at once evident: (1) all voluntary movement in parts of the body below the lesj immediately and permanently lost; (2) all sensation from the lover (aboral) parts is abolished; and (3) reflex functions in all segments of the isolated spinal cord are suspended. The last effect called *spinal shock,*

involves tendon as well as autonomic reflexes it lasts for weeks to months and is so dramatic that Riddoch u I it as a basis for dividing the clinical effects of spinal cord transe tion into two stages: (1) spinal shock or areflexia and (2) height" ened reflex activity. The separation of these two stages is not as this statement might imply but is nevertheless fundarnental. Less complete lesions of the spinal cord may result in little or no spinal shock, and the same concerns any type of lesion that develops slowly.

Stage of Spinal Shock or Areflexia

The loss of the motor function at the time of injury quadriplegia (better termed tetraplegia) with lesions of the fourth to fifth cervical segments, paraplegia with lesions of the thoracic cordis accompanied by atonic paralysis of bladder and bowel, gastric atony, loss of sensation below the level corresponding to the spinal cord lesion, muscular flaccidity, and complete or almost complete suppression of all spinal segmental reflex activity below the lesion. The neural elements below the lesion fail to perform their normal function because of their sudden separation from those of higher levels. Impaired also in the segments below the lesion is the control of the autonomic function. Vasomotor tone, sweating, and piloerection in the lower parts of the body are temporarily lost. Systemic hypotension may be severe and contribute to the spinal cord damage. The lower extremities lose heat if left uncovered, and they swell if dependent. The skin is dry and pale, and ulcerations may develop over bony prominences. The sphincters of the bladder and the rectum remain contracted to some degree due to the loss of inhibitory influence of higher central nervous system centers but the detrusor and smooth muscle of the rectum are atonic. Urine accumulates unti the intravesicular pressure is sufficient to overcome the sphincters, then driblets escape (overflow incontinence). There is also passive distention of the bowel, retention of feces, and absence of peristalsis (paralytic ileus). Genital reflexes (penile erection, bulbocavernosus reflex, (penile erection, bulbocavernosus reflex, cjntraction of dartos muscle) are abolished or profoundly depressed.

The duration of the stage of complete areflexia varies greatly. In a small number it is permanent, or only fragmentary reflex activity is regained many months or years after the injury. In such patients the spinal segments below the level of transection may have themselves been injured perhaps by a vascular mechanism, although explanation is unproven. More likely there is a loss of the brainstem-spinal facilitatory mechanisms and an, increase in inhibitorv activity in the isolated segments as indicated below. In some patients, minimal genital and flexor reflex activity can be detected within a few days of the injury. In the majority of patients, this *minimal reflex activity* appears within a period of 1 to 6 weeks. Usually the bulbocavemosus reflex is the first to return. Noxious stimulation of the plantar surfaces evokes a tremulous twitching and brief flexion or extension movements of the great toes. Contraction of the anal sphincter can be elicited by plantar or perianal stimulation, and other genital reflexes reappear at about the same time.

The explanation of spinal shock, which is brief in submammalian forms and more lasting in higher mammals, especially in primates, is believed to be the sudden interruption of suprasegmental descending fiber systems that normally keep the spinal motor neurons in a continuous state of subliminal depolarization (ready to respond). In the cat and monkey, Fulton found the facilitatory tracts in question to be the reticulospinal and vestibulospinal. Subsequent studies showed that in monkeys, some degree of spinal shock can result from interruption of the corticospinal tracts alone. This cannot be the significant factor, however, at least in humans, realise spinal shock may be very mild or inevident as a result of acute cerebral and brainstem lesions that interrupt the cortieospinal tracts. F waves of the isolated cord are suppressed. Interest in recent years has focused on a possible role for neurotransmitters (catecholamines, endorphins, substance P, and 5-hydroxytryptamine). The claim that naloxone and the endogenous opiate antagonist thyrotropin releasing factor might reduce the extent of an acute spinal cord lesion has not been corroborated. Clonidine, a noradrenergic receptor activator is reported to reduce flexor spasms and restore the balance between excitatory and inhibitory activity, allowing the spinal reflex generator for locomotion to function.

Stage of Heightened Reflex Activity

Usually, after a few weeks, the reflex responses to stimulation, which are initially minimal and unsustained, become stronger and more easily elicitable and come to include additional and more proximal muscles. Gradually the typical pattern of heighten flexion reflexes emerges: dorsiflexion of the big toe (Babinski sing), fanning of the other toes, and later, flexion or slow withdrawal movements of the foot, leg, and thigh with contraction of the tensor fascialata (triple flexion).

Achilles reflexes and then the patellar reflexes return. Retention of urine becomes less complete, and at irregular intervals urine is expelled by active contractions of the detrusor muscle. Reflex defecation also begins.

After several months the withdrawal reflexes become greatlv exaggerated, to the point of flexor spasms, and may be accompanied by profuse sweating, piloerection, and automatic emptying of the bladder (occasionally of the rectum). This is the "mass reflex" which is evoked by stimulation of the skin of the legs or by some interoceptive stimulus, such as a full bladder. Varying degrees of heightened flexos refle[activity may last for years. Heat-induced sweating is defective, but reflex-evoked ("spinal") may be profuse. Above the level of the lesion, thermoregulatory sweating may be exaggerated and is accompanied by cutaneous flushing, pounding headache, hypertension, and reflex bradycardia.

Extensor reflexes eventually develop in most cases, but they do *not lead to the abolition of the flexor reflexes.* The overactivity of extensor muscles may appear as early as 6 months after the injury, developed. Extensor responses are at first manifest in certain muscres of the his and thigh and later of the leg. In a few patient extensor reflexes are organized info support reactions sufficient to permid spinal standing.

From these observations one would suspect that the ultimate posture of the legs flexion or extension does not depend solely on the completeness or incompleteness of the spinal cord lesion. The development of *paraplegia in flexion* relates also to the level of the lesion, being seen most often with cervical lesions and progressively less often with more caudal ones. Repeated flexor spasms, which are more frequent with higher lesions, and the ensuing contractures ultimately determine a fixed flexor posture. Conversely, reduction of flexor spasms by elimination of nociceptive stimuli (infected bladder, decubiti, etc.) favors an extensor posture of the legs *(paraplegia in extension).* The positioning of the limbs during the early stages of paraplegia greatly influences their ultimate posture. Thus, prolonged fixation of the paralyzed limbs in adduction and semiflexion favors subsequent paraplegia in flexion. Placing the patient prone or placing the limbs in abduction and extension facilitates the development of predominantly extensor postures. Nevertheless, strong and persistent extensor postures are observed only with partial lesions of the spinal cord.

Of some interest is the fact that many patients report sensory symptoms in segments of the body below the level of their transection. Thus, a tactile stimulus above the level of the lesion may be felt below the transection (synesthesia). Patients describe a variety of paresthesias, the most common being a dull, burning pain in the lower back and abdomen, buttocks, and perineum. We have encountered patients in whom aching testicular or rectal pain were the main problem. The pain may be intense and last for a year or longer, after which it gradually subsides. It may persist after rhizotomy but can be abolished by anesthetizing the stump of the proxtfnal (upper) segment of the spinal cord, according to Pollock and his collaborators. Transmission of sensation over splanchnic afferents to levels of the spinal cord above the lesion, the conventional explanation, is therefore not the most plausible one.

The overactivity of neurons in the isolated segments of the spinal cord has several explanations. One assumes that suprasegmental afferent sensory impulses evoke exaggerated nocifensive and phases and tonic myotatic reflexes. But isolated neurons also become hypersensitive to neurotransmitters.

High cervical lesions may result in extreme and prolonged tonic spasms of the legs due to release of tonic myotatic reflexes. Under these circumstances, attempted voluntary movement may excite intense contraction of all flexor and extensor muscles lasting for

several minutes. Segmental damage in the low cervical or lumbar gray matter, destroying inhibitory Renshaw neurons, may release activity of remaining anterior horn cells, leading to spinal segmental spasticity. Any residual symptoms persisting after 6 months are likely to be permanent, although in a small proportion of patients some return of function (particularly sensation) is possible after this time. Loss of the motor and the sensory function above the lesion, coming on years after the trauma, occurs occasionally and is due to an enlarging cavity in the proximal segment of the cord.

Central Cord Syndrome of Schneider and "Cruciate Paralysis"

In the acute central cord lesion, the loss of the motor function is characteristically more severe in the upper limbs than in the lower ones and particularly severe in the hands. Bladder dysfunction with urinary retention occurs in some of the cases and sensory loss is often slight (hyperpathia over the shoulders and arms may be the only sensory abnormality). The destruction of gray matter (motor and sensory neurons) may leave an atrophic, are flexive pa- ralysis and a segmental loss of pain and thermal sensation. Retro-flexion injuries of the head and neck are the ones most often associated with this central cord syndrome, but hematomyelia, nccro-tizing myelitis, fibrocartilagenous embolism, and possibly infarction due to compression of the vertebral artery in the medullary cervical region are other causes.

4 % of patients who survive high cervical cord injuries demon- strate what these authors refer to as "cruciate paralysis." The latter state is similar to the central cord syndrome except that the weakness is even more selective, being practically limited to the arms, a feature that is attributable to the segregation of corticospinal fibers to the arms (rostral) and to the legs (caudal) within the decussation. The patients described in the literature have had . iuries, basically contusions, of the C1-C2 region. The arm weakness may be asymmetrical or even unilateral; sensory loss is inconsistent.

Examination of the Spine-Injured Patient

The level of the spinal cord and vertebral lesions can be determined from the clinical findings. A complete paralysis of the arms and legs usually indicates a fracture or dislocation at the fourth to fifth cervical vertebrae. If the legs are paralyzed and the arms can still be abducted and flexed, the lesion is likely to be at the fifth to sixth cervical vertebrae. Paralysis of the legs and only the hands indicates a lesion at the sixth to seventh cervical level. Below the cervical region, the spinal cord segments and roots are not opposite their similarly numbered vertebrae. The spinal cord ends opposite the first lumbar interspace. Vertebral lesions below this point give rise predominantly to cauda equina syndromes; these carry a better prognosis than injuries to the lower thoracic vertebrae, which involve both cord and multiple roots. The level of sensory loss on the trunk, determined by perception of pinprick, is also an accurate guide to the level of the lesion, with a few qualifications. With lesions of the lower cervical cord, even if complete, sensation may be preserved down to the nipple line, because of the contribution of the C3 and C4 cutaneous branches of the cervical plexus, which innervate skin below the clavicle. Rarely, a lesion will involve only the outermost fibers of the spinothalamic pathways, sparing the innermost ones, in which case the sensory level (to pain and temperature) will be below the level of the lesion. In all cases of spinal cord and cauda equina injury, the prognosis for recovery is more favorable if any movement or sensation is elicitable during the first 48 to 72 h.

If the spine can be examined safely, it should be inspected for angulations or irregularities and gently percussed to elicit signs of bony injury. Collateral injury of the thorax, abdomen, and long bones must always be sought, to direct early radiologic studies and detect serious complications such as pneumothorax, splenic rupture, or fat embolism.

Management of Spinal Injury

In all cases of suspected spinal injury, the immediate concern is that there be no movement (especially flexion) of the cervical spine from the moment of the accident. The patient should be placed supine on a firm, flat surface (with one person assigned to keeping the

head immobile) and should be transported by a vehicle that ca accept the litter. Preferably, the patient should be transported by an ambulance equipped with spine boards, to which the head is rigidly fixed by straps. The latter provide a more effective means of immobilization than sandbags or similar objects placed on each side of the head and neck. On arrival at the hospital, it is useful to have the patient remain on the backboard until a lateral film and MRI of the cervical spine have been obtained.

A careful neurological examination with detailed recording of motor, sensory, and sphincter function is necessary to follow the clinical progress of spinal cord injury. A common practice is to define the injury according to the standards of the American Spinal Injury Association and to assign the injury to a point on the Frankel Scale.

Complete: motor and sensory loss below the lesion

A. Incomplete: some sensory preservation below the zone of injury.
B. Incomplete: motor and sensory sparing, but the patient is nonfunctional.
C. Incomplete: motor and sensory sparing and the patient is functional (stands and walks).
D. Complete functional recovery: reflexes may be abnormal.

Obviously, groups B, C, and D have a more favorable prognosis for recovery of ambulation than does group A.

Once the degrees of injury to spine and cord have been assessed, corticosteroids are given in high dosage. In patients receiving methylprednisolone (bolus of 30 mg/kg followed by 5.4 mg/kg every hour), beginning within 8 h of the injury and continuing for 23 h, Bracken and colleagues reported a slight but significant improvement in both motor and sensory function compared to controls. Naloxone was found to be of no value. Also, in a small series of patients, the administration of G_{MI} ganglioside (100 mg intravenously each day from the time of the accident) was found to enhance ultimate recovery to a modest degree, but this finding needs to be corroborated.

Next, radiologic examinations are undertaken to determine the alignment of vertebral bodies and pedicles, compression of the spinal cord or cauda equina due to malalignment or bone debris in the spinal canal, and the presence of tissue damage within the cord. The MRI is ideally suited to display these processes but if it is not available, or if there is uncertainty regarding cord compression, it is useful to perform myelography with CT scanning to obtain more detailed views of the spinal subarachnoid space. Instability of the spinal elements can often be inferred from dislocations or from certain fractures of the pedicles, pars articularis, or transverse processes, but gentle flexion and extension of the injured areas must sometimes be undertaken and films obtained in each position (Fig.1).

The results of the conservative and aggressive surgical plans of management have been difficult to compare and have not been evaluated with modern neurological techniques. Collins, a participant in the National Institutes of Health study of acute management of spinal cord injury, concluded that the survival rate was increased due to early surgical stabilization of fractures and fixation of the spine (Fig.2).

Radiography. Spondylodesis of a lumbar part of the spine

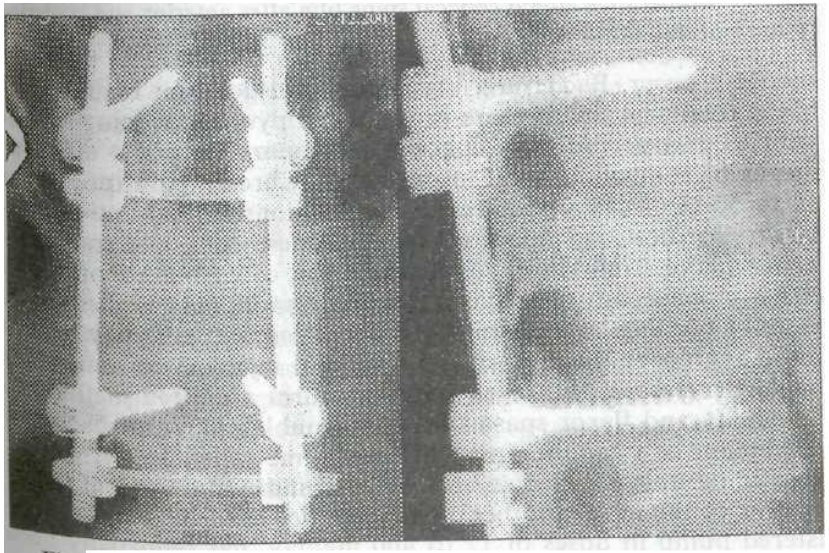

Fig.

The greatest risk to the patient with spinal cord injury is in the first week or 10 days when gastric dilatation, ileus, shock, and infetion are the main threats to life. The mortality rate falls rapidly after the first 3 months; beyond this time, 86 % of paraplegics and 80 % of quadriplegics will survive within 10 years or longer. In children, the survival rate is even higher. The cumulative 7-year survival rate in spinal cord-injured patients (who had survived at least within 24 h after injury) are 87 %. Old age at the time of injury and complete quadriplegia are the worst prognostic factors.

The aftercare of patients with paraplegia is concerned with management of bladder and bowel disturbances, care of the skin, prevention of pulmonary embolism, and maintenance of nutrition. Decubitus ulcers can be prevented by frequent turning to avoid pressure necrosis, use of special mattresses, and meticulous skin care. Deep lesions require debridement and full-thickness grafting. At first continual catheterization is necessary; then, after several weeks, the bladder can be managed by intermittent catheterization once or twice daily, using a scrupulous aseptic technique. Close watch is kept for bladder infection, which is treated promptly

The Main Ligamentous Structures of the Spine

A. Buckling of the yellow ligament may compress the nerve root or the spinal nerve at its origin in the intervertebral foramen, particularly if the foramen is narrowed by osteophytic overgrowth.B. Posterior aspect of the vertebra/bodies. Fibers of the posterior longitudinal ligament merge with the posteromedial portion of the annulus fibrosus, leaving the posterolateral of annulus relatively unsupported.

General Clinical Features of Low-Back Pain

Types of Low-Back Pain. Of the several symptoms of spinal disease (pain, stiffness, limitation of movement, and deformity), pain is of foremost importance. Four types of pain may be distinguished: local, referred, radicular, and that arising from secondary

(protective) muscular spasm. These several types of pain can often be discerned from the patient's description; reliance is placed mainly on the character of the pain, its location, and conditions that modify it. *Local pain* is caused by any pathologic process that impinges upon structures containing sensory endings. Involvement of the periosteum, capsule of apophyseal joints, lumbodorsal fascia, muscles, annulus fibrosus, and ligaments is often exquisitely painful, whereas destruction of the vertebral body or nucleus pulposus alone produces little or no pain. Inflammatory or traumatic swelling of the affected tissues is not apparent if it is located deep in the back. Local pain is most often described as steady and aching, but it may be intermittent and sharp and, though not sharply circumscribed, is always felt in or near the affected part of the spine.

Referred pain is of two types, one that is projected from the spine to viscera and other structures lying within the territory of the lumbar and upper sacral dermatomes and another that is projected from pelvic and abdominal viscera to the spine. Pain due to disease of the upper part of the lumbar spine is often referred, the flank, lateral hip, groin, and anterior thigh. This has been attributed to irritation of the superior cluneal nerves, which are ^ rived from the posterior divisions of the first three lumbar spinal nerves and innervate the superior portions of the buttocks. Pain from the lower part of the lumbar spine is usually referred to the lower buttocks and posterior thighs and is due to irritation of lower spinal nerves, which activate the same pool of intraspinal neurons as the nerves that innervate the posterior thighs. Pain of this type is usually rather diffuse and has a deep, aching quality, but it tends at times to be more superficially projected. In general, die intensity of the referred pain parallels that of the local pain. In other words, maneuvers that alter local pain have a similar effect on referred pain, though lacking the precision and immediacy of socalled root pain. Usually pain from visceral diseases is felt within the abdomen, flanks, or lumbar region and may be modified by the state of activity of the viscera. Its character and temporal relationships are those of the particular visceral structure involved, I and posture and movement of the back have relatively little effect I on either the local pain or that referred to the back.

Radicular, or "root" pain has some of the characteristics of referred pain but differs in its greater intensity, distal radiation, circumscription to the territory of a root, and factors that excite it. The mechanism is stretching, irritation, or compression of a spinal root, within or central to the intervertebral foramen. The pain is sharp, often intense, and usually superimposed on the dull ache of referred pain; it nearly always radiates from a central position near the spine to some part of the lower limb. Coughing, sneezing, and straining characteristically evoke this sharp radiating pain, although each of these actions may also jar or move the spine and enhance local pain. In fact, any maneuver that stretches the nerve root — e.g., "straight-leg raising" in cases of L4, L5, or SI root involvement or thigh extension with L3 root involvement - evokes radicular pain; jugular vein compression, which raises intraspinal pressure and may cause a shift in position of the root, may have a similar effect. Involvement of L4, L5, and SI roots, which form the ^{5}'atic nerve, causes pain that extends down the posterior aspects f the thigh and the postero- and anterolateral aspects of the leg, ° to the foot — so-called sciatica. The region of the leg and foot in which the pain is experienced depends upon the nerve root that is effected: with L5 radiculopathy, pain radiates to the medial aspect $_{o}$f the foot into the great toe, and with SI involvement, into the lateral part of leg and foot. Involvement of the L3 and sometimes L4 root causes pain in the groin and anterior thigh. Paresthesias or superficial sensory loss, soreness of the skin, and tenderness in certain circumscribed regions along the nerve usually accompany radicular pain. If the anterior roots are involved as well, reflex loss, weakness, atrophy, and fascicular twitching may also occur.

In patients with severe circumferential constriction of the cau-da equina due to spondylosis, sensory-motor impairment, and sometimes referred pain is elicited by standing and walking. The neurological symptoms involve the calves .and the back of the thighs, imulating the exercise-induced symptoms due to vascular insufficiency - hence the term spinal claudication.

Examination of the Lower Back

Some information may be gained by inspection of the back, buttocks, and lower limbs in various positions. The normal spine shows a dorsal kyphosis and lumbar lordosis in the sagittal plane, which in some individuals may be somewhat exaggerated (swayback). In coronal plane, the spine is normally straight or shows a slight curvature, particularly in women. One should observe the spine losely for excessive curvature, a list, flattening of the normal lumbar lordosis, presence of a gibbus (a sharp, kyphotic angulation fsually indicative of a fracture), pelvic tilt or obliquity, and asymmetryof the paravertebral or gluteal musculature. A sagging gluteal fold suggests involvement of the SI root. In sciatica one may observe a flexed posture of the affected leg, presumably to reduce tension on the irritated nerve. Patients in whom a free fragment able to lie down and extend the spine.

Passive straight-leg raising (possible up to 90 in normal individuals except in those who have unusually tight hamsorigs), like forward bending in the standing posture with the legs straight, places the sciatic nerve and its roots under tension, thereby producing radicular radiating pain. It may also cause an anterior rot the pelvis around a transverse axis, increasing stress on the lum- hosacral joint and causing pain if this joint is arthritic or wise diseased. Consequently, in diseases of the lumbosacra and roots, passive straight-leg raising evokes pain and is on the affected side and, to a lesser extent, on the (Lasegue sign).

ft is important to remember that the evoked is always referred to the diseased side, no matter which leg; vated. While in the supine position, leg length (anterior-sun iliac spine to medial mallcolus) and the circumference of the ti and calf should be measured. Abdominal, rectal, and pelvic examinations, in addition to sessment of the integrity of the peripheral vascular system, are sential elements in the study of the patients with low-back sym toms which fail to be clarified by these simple spinal maneuvj Neoplastic inflammatory, or degenerative disorders may prodj symptoms referred to the lower part of the spine.

Ancillary Diagnostic Procedures

Depending on the circumstances, these may include a blow count and erythrocyte sedimentation rate (especially helpful in screening for infection or myeloma); measurement of the serum proteins, calcium, phosphorus, uric acid, alkaline phosphatase, acid phosphatase and prostate specific antigen (if one suspects metastatic carcinoma prostate): a serum protein eleciropnoresis (myeloma proteins): a tuberculin test or an agglutination test for Brucellti, and a test for rheumatoid factor. Radiographs of the lull bar spine (preferably with the patient standing) in the anteroposterior, lateral, and oblique planes are still useful in the routine evaluation of low-back pain and sciatica. Readily demonstrable in plain films are narrowing of the intervertebral disc spaces, displacernew of vertebral bodies (spondylolisthesis), and an unsuspected infiltration of bone by cancer. In cases of suspected disc herniation or tumor infiltration of the spinal canal, one proceeds directly to *CT*° MRI. Although these imaging procedures have largely replaced conventional myelography, the latter examination, when combined with CT, provides detailed information about the dura sleeves that surround the spinal roots, at times disclosing sub truncations caused by laterally situated disc herniations and revealing surface abnormalities of the spinal cord, such as arteriovenous malformations.

Principal Conditions Giving Rise to Pain in the Lower Back

Congenital Anomalies of the Lumbar Spine. Anatomic variations of the spine are frequent, and though rarely *of* themselves the source of pain and functional derangement, they may predispose an individual to discogenic and spondylotic complications by virtue of altering the mechanics and alignment of the vertebrae or size of the spinal canal.

The commonest anomaly is a lack of fusion of the laminae of one or several of the lumbar vertebrae or of the sacrum (spina bifida). Occasionally, a subcutaneous mass, hypertrichosis, or hy- perpigmentation in the sacral area betrays the condition, but in most patients it remains occult until disclosed radiologically. The anomaly may be accompanied by malformation of vertebral joints and usually induces pain only when aggravated by injury.

Many other congenital anomalies affect the lower lumbar vertebrae: asymmetrical facetal joints, abnormalities of the transverse processes, "sacralization" of the fifth lumbar vertebra (in which L5 appears to be fixed to the sacrum), or "lumbarization" of the first sacral vertebra (in which SI looks like a sixth lumbar vertebra) are seen occasionally in patients with low-back symptoms hut apparently with no greater frequency than in asymptomatic individuals. The role of these anomalies in the genesis of low-bade derangement is unclear, they are rarely the cause of specific symptomt.

Spondylolisthesis consists of a bony defect in the pars interar-ticularis (the segment at the junction of pedicle and lamina) of the lower lumbar vertebrae. The defect is remarkably common: it affects approximately 5 % of the North American population, is probably genetic, and predisposes to fracture at this location. Radio-graphically, the pars interarticularis defect is best visualized on oblique projections. In some persons it is unilateral and may cause unilateral back pain that is accentuated by hyperextension and twisting. It is not uncommon in athletes. In the usual bilateral form, the vertebral body, pedicles, and superior articular facets move anteriorly, leaving the posterior elements behind. This latter disorder, known as spondylolisthesis, is mainly a disease of young per-sons (peak incidence is between ages of 5 to 7 years). It may cause little difficulty at first but eventually becomes symptomatic. The patient complains of pain in the low back radiating into the thighs and of limitation of motion. Examination discloses tenderness near the segment that has "slipped" (most often L5, occasionally L4), a palpable “step" ol the spinous process forward from the segment below, of spondylolisthesis L5-S1 hamstring spasm, and, in severe cases (spondylolisthesis), shortening of the trunk and protrusion of the lower abdomen (both of which result from the abnormal forward shift of L5 on SI), and signs of involvement of spinal roots — paresthesias and sensory loss, muscle weakness, and reflex impairment. These neurological symptoms and signs tend not to be severe.

Herniation of Lumbar Intervertebral Discs. This condition is a major cause of severe and chronic or recurrent low-back and leg pain. It occurs mainly during the third and fourth decades of life, when the nucleus pulposus is still gelatinous. The disc between the fifth lumbar and first sacral vertebrae is the one most often involved, and, with lessening frequency, that between the fourth and fifth, third and fourth, second and third, and first and second lumbar vertebrae. Rare in the thoracic portion of the spine, disc disuse is again frequent at the fifth and sixth and sixth and seventh cervical vertebrae.

The cause of a herniated lumbar disc is usually a flexion injury, but a considerable proportion of patients do not recall any traumatic episode. Degeneration of the nucleus pulposus, the posterior longitudinal ligaments, and the annulus fibrosus may have taken place silently or have been manifested by mild, recurrent ache. A sneeze, lurch, or other trivial movement may then cause the nucleus pulposus to prolapse, pushing the frayed and weakened annulus posteriorly. Fragments of the nucleus pulposus through rents in the annulus, usually to one side or the other (sometimes centrally), where they impinge upon a root or roots. In more severe cases of disc disease, the nucleus may protrude through the annulus L1 be extruded epidurally as *a* free fragment. A large protrusion may compress the root(s) against the articular apophysis or lamina. The protruded material may be resorbed to some J tent, but often it does not cause chronic irritation of the root or a discarthrosis with posterior ostcophyte formation.

The Clinical Syndrome

The fully developed syndrome of prolapsed intervertebral lum- bar disc consists of: (1) pain irradiating into the buttock, thigh, catf and foot, (2) a stiff or unnatural spinal posture, and (3) some combination of paresthesias, weakness, and reflex impairment.

The pain of herniated intervertebral disc is of several types. First there is "spontaneous" pain that ranges from a mild discomfort to the most severe knife-like stabs irradiating lengthways into the leg and superimposed on a constant intense ache. With the most acute and

severe pain, the patient must stay *in* bed, avoiding the slightest movement: a cough, sneezing, or strain is intolerable. The patient is usually most comfortable lying on his back with legs flexed at the knees and hips (dorsal decubitus position) and with the shoulders raised on pillows to obliterate the lumbar lordosis. For some patients, a particular lateral decubitus position is more comfortable. As mentioned earlier, free fragments on disc that find their way to a lateral and posterior position in the spinal canal may produce the opposite situation, of being unable to extend the spine and the supine. When the condition is less severe, walking is possible, though fatigue sets in quickly, with a feeling of heaviness and draw- ing pain. Sitting and standing up from a sitting position are partic- ularly painful. The pain is usually located deep in the buttock, just lateral to and below the sacroiliac joint, and in the posterolatef region on the thigh, with irradiation to the calf and infrequently to the heel and other parts of the foot. Irradiation of pain into the foot should at least raise the possibility of an alternative cause of nerve damage. Pain is also characteristically provoked by pressure over the course of the sciatic nerve at the classic points of Valleix (sciatic notch, retrotrochanteric gutter, posterior surface of thigh, head of fibula). Pressure at one point may cause irradiation of pain and tingling down the leg.

The signs of severe spinal root compression are hypotonia, impairment of sensation, loss or diminution of tendon reflexes, and muscle weakness. The hypotonia is evident on inspection and palpa- tion of the buttock and calf, and the Achilles tendon tends to be less salient. Paresthesias (rarely hyperesthesia or hypoesthesia) are reported by one-third of patients; usually they are felt in the foot, sometimes in the leg. Often there is a diminution of pain perception over the appropriate dermatome. Muscle weakness occurs, but less frequently. The ankle or knee jerk is usually diminished or lost on the side of the lesion. The reflex changes have little relationship to the severity of the pain or sensory loss. Furthermore, compression of the fourth or fifth lumbar roots may occur without any change in the tendon reflexes. Bilaterality of symptoms and signs is rare, like sphincteric paralysis, but they may occur with large central protrusions.

The CSF protein is then predictably elevated, usually in the range of 55 to 85 mg/dL, sometimes higher.

With lesions of the first sacral root the pain is felt in the midgluteal region, posterior part of the thigh, posterior region of the calf to the heel, outer plantar surface of the foot, and fourth and fifth toes. Tenderness is most pronounced over the midgluteal region (in the region of the sacroiliac joint), posterior thigh areas, and calf. Paresthesias and sensory loss are mainly in the lower part of the leg and outer toes, and weakness, if present, involves the flexor muscles of the foot and toes, abductors of the toes, and hamstring muscle. The Achilles reflex is diminished or absent in the first and the majority of cases. Walking on the toes is more difficult and uncomfortable than walking on the heels because of weakness of the plantar flexors.

The rarer lesions of the third and fourth lumbar roots give rise to pain in the anterior part of the thigh and knee and anteromedial part of the leg (fourth lumbar), with corresponding sensory loss. The knee jerk is diminished or abolished. L3 motor root lesions may weaken the quadriceps, thigh adductor, and iliopsoas; L4, the anterior tibial innervated muscles, LI root pain is projected to the groin, and L2 — to the lateral hip. Motion of the spine and certain positions are most evocative of root pain; if the pain is constant in all positions, root irritation is seldom the cause.

Rarer still are protrusions of thoracic intervertebral discs (0.5 % of all surgically verified disc protrusions). The four lowermost thoracic interspaces are the most frequently involved. Trauma, particularly hard falls on the heels or buttocks, is an important causative factor. Deep boring spine pain, root pain circling the body or projected to the abdomen or thorax (sometimes simulating visceral disease), paresthesias below the level of the lesion, loss of sensation, both deep and superficial, and paraparesis or paraplegia are I the usual clinical manifestations. Careful CT-myelography and I MRI are the most important diagnostic maneuvers.

When all components of the syndrome are present, diagnosis is easy; but most neurologists prefer to corroborate their clinical im- pression by CT, with or without contrast myelography, or prefe- rably by MRI of L3-S1. Usually this demonstrates the extruded disc at the suspected site and also excludes herniations at other sites or an unsuspected tumour.

Spondyiotic Spurs and Lateral Recess Stenosis

Symptoms due to these disorders need to be distinguished from those of hemiated disc. Disabling pain in one or both legs on standing and walking, relieved by squatting and lying down, with variable motor, reflex, and sensory changes referable to a roof or roots are present. Radiologically there was stenosis at one vertebral level. Most often the superior facet of L5 narrowed the lateral recess at the upper border of the pedicle, compressing the L5 root and sometimes the SI as well. This is one of the anatomic configurations that is consistently shown to advantage by CT-myelography and may not be appreciated by conventional CT or MRI. This is particularly true, as already mentioned, if there is an associated far lateral disc hemiation. This condition is more properly designated as a unilateral lumbar spondy/osis, or as a spondylolysis. The adjacent articular capsules may suffer injury and add direct and referred pain to a radicular syndrome. Intraarticular lidocaine relieves the pain, according to some orthopedists.

Spondyiotic Caudal Radiculopathy or Lumbar Stenosis Syndrome

In the lumbar region, osteoarthritic or spondyiotic changes, su-perimposed on a smaller than normal spinal canal, may lead to compression of the caudal roots. The roots are actually caught between the posterior surface of the vertebral body and the ligamentum fla-vum posterolaterally. Upon standing or walking (downhill walking is especially difficult), there is in many cases a gradual onset of numbness and weakness of the legs, which forces the patient to sit down. When this condition is more severe, the patient gains relief by lying down with the legs flexed at the hips and knees. Usually the numbness begins in one leg, spreads to the other one, and increases on standing or walking. Tendon reflexes may disappear, only to return on flexing the spine. Pain in the low back is variable. Disturbances of micturition and impotence are rare. In some patients with lumbar stenosis, neurological symptoms persist without relation to body position. The clinical picture, with its inter-mittency, corresponds to the so-called intermittent claudication of the cauda equina. Soon thereafter to be due not to ischemia but to encroachment on the cauda by hypertrophied apophyseal joints, thickened ligaments, and small protrusions of disc material engrafted upon a canal that is develop mentally shallow in the anteropos-terior diameter. Sometimes a slight subluxation at L3-L4 or at L4-L5 is also present. Later it became evident that the canal in these cases is also narrow from side to side (reduced interpedicular distance radiographically). Decompression of the spinal canal relieves the symptoms in a considerable proportion of the cases.

The Facet Syndrome

This syndrome has been clarified in recent years, but its definition is still somewhat imprecise. Some authors use the term to de-signate a painful state that responds to the injection of analgesics into the facet; others use the term in a more general sense, to describe pain that emanates from the facet joint and lateral recess, in either case, the condition is closely related to the one described above. At operation, the spinal root is compressed against the floor of the intervertebral canal by overgrowth of an inferior or superior facet. Foraminotomy and facetectomy, after exploration of the root from the dural sac to the pedicle, relieve the pain in operated cases. Denervation of lumbar facets by radio-frequency electroj introduced percutaneousiy onto nerves supplying the zygoapophyseal joints has been proposed as therapy.

PAIN IN THE NECK, SHOULDER, AND ARM

General Considerations

In this connection, it is useful to distinguish three major catego-ries of painful disease — that originating in the spine, in the bra-chial plexus, and in the shoulder. Although the distribution of pain from each of these sources may overlap, the patient can usually

indicate its site of origin.

Pain arising from the cervical part of the spine is felt in the neck and back of the head (although it may be projected to the shoulder and upper arm), is evoked or enhanced by certain movements or positions of the neck, and is accompanied by limitation of motion of the neck and by tenderness to palpation over the cervical spine.

Pain of brachial plexus origin is experienced in and around the shoulder and in the supraclavicular region, or in the axilla, is induced by certain maneuvers and positions of the arm, and is sometimes associated with tenderness of structures above the clavicle. There may be a palpable abnormality above the clavicle (aneurysm of the subclavian artery, tumor, cervical rib). The combination of circulatory abnormalities and signs referable to the medial cord of the brachial plexus are characteristic of the thoracic outlet syndrome, which is described further on.

Pain localized in the shoulder region, worsened by motion, and associated with tenderness and limitation of movement (especially internal and external rotation and abduction) points to a tendonitis, subacromial bursitis, or tear of the rotator cuff, which is made up of the tendons of the muscles surrounding the shoulder joint. The term bursitis is often used loosely to designate these disorders. Shoulder pain, like spine and plexus pain, may irradiate into the arm and rarely into the hand, but sensory, motor, and reflex changes — which always indicate disease of nerve roots, plexus, or nerves — are absent. Shoulder pain of this type is very common in the middle and late adult age. It may arise spontaneously or after unusual or vigorous use of the arm. Local tenderness over the greater tuberosity of the humerus is characteristic. Radiographs of the shoulder may be negative or show a calcium deposit in the supraspinatus tendon or subacromial bursa. In most patients the pain subsides with immobilization and analgesics. If it does not, the injection of small amounts of corticosteriods into the bursa, or the site of major pain with shoulder movement in the case of rotator cuff injuries, is often effective and allows the patient to mobilize the shoulder.

Cervical Disc Protrusion

A common cause of neck, shoulder, and arm pain is disc herni-ation in the lower cervical region. It may develop after trauma, which may be major or minor (from sudden hyperextension of the neck, diving, forceful chiropractic manipulations, etc.). The roots most commonly involved in cervical disc protrusion are the seventh (in 70 % of cases) and the sixth (20 %); fifth- and eighth-root involvement make up the remaining 10 %.

With a laterally situated disc protrusion between the fifth and sixth cervical vertebrae, the symptoms and signs are referred to the sixth cervical segment. The full syndrome is characterized by pain at the trapezius ridge and tip of the shoulder, with radiation into the anterior-upper part of the arm, radial forearm, and often the thumb; parcsthesias and sensory impairment in the same regions; tenderness in the area above the spine of the scapula and in the supraclavicular and biceps regions; weakness in flexion of the forearm: and diminished to absent biceps and supinator reflexes (triceps retained or exaggerated).

When the protruded disc lies between the sixth and seventh vertebrae, there is involvement of the seventh cervical root. The pain is in the region of the shoulder blade, pectoral region and medial axilla, posterolateral upper arm, elbow and dorsal forearm, index and middle fingers, or all the fingers. Tenderness is most pronounced over the medial aspect of the shoulder blade opposite the third to fourth thoracic spinous processes and in the supraclavicular area and triceps region. Paresthesias and sensory loss are most evident the second and third fingers or may be experienced in the tips all the fingers. Weakness involves the extensors of the forearm and sometimes of the wrist; occasionally the hand grip is weak as well; the triceps reflex is diminished or absent; and the biceps a supinator reflexes are preserved.

The fifth cervical root syndrome, produced by disc herniationi between the fourth and fifth vertebral bodies, is characterized pain in the shoulder and trapezius region and by supra- and infra. I spinatus weakness, manifested by an inability to abduct the and rotate it externally with the shoulder adducted.

Compression of the eighth cervical root at C7-TI may mimic an ulnar palsy. The pain is along the medial side of the forearm and the sensory loss is in the distribution of the medial cutaneous nerve of the forearm and of the ulnar nerve in the hand. The weakness involves the muscles supplied by the ulnar nerve.

These syndromes are usually incomplete since only one or sev- eral of the typical findings are present. Particularly impressive is the case of isolated weakness without pain in laterally placed cervical disc rupture, especially with discs at the fifth and sixth levels. Virtually every patient, irrespective of the particular root(s) involved shows a limitation in the range of the neck motion and aggravation of pain with movement (particularly hyperextension). Coughing, sneezing, and downward pressure on the head in the hyperextended position usually exacerbate the pain, and traction (even manual) tends to relieve it. Nerve conduction studies, F responses, and EMG are extremely helpful in determining the level of root compression and distinguishing pain of a radicular origin from that originating in the brachial plexus.

In many of the cervical radicular syndromes, the onset is acute and there has been no traumatic incident. It is difficult to relate the syndrome to osseous changes that have been present for years. Even more difficult to explain is the tendency to recovery within *a* few weeks when movement of the neck is limited by traction or a collar. Conservative measures should always be tried before turning to laminectomy and foraminotomy unless there are additional signs of a myelopathy, such as weakness, gait ataxia, or sphincteric dysfunction.

Unlike herniated lumbar discs, cervical ones, if large and centrally situated, may result in compression of the spinal cord (central disc, all of the cord; paracentral disc, part of the cord). The centrally situated disc may be painless, and the cord syndrome may simulate a degenerative disease (amyotrophic lateral sclerosis, combined system disease).

Management of Back and Limb Pain

The pain of muscular and ligamentous strains and minor disc Protrusions is usually self-limited, responding to simple measures in a relatively short period of time. The basic principle of therapy in both disorders is rest, in a recumbent position, tor several days. Lying on the side with knees and hips flexed is usually a favored Position. With strains of the sacrospinalis muscles and sacroihac laments, the optimal position is hyperextenswn. This position is best maintained by having the patient lie with a small pillow or folded blanket under the lumbar portion of the spine or lie face down. Physical measures - such as application of ice in the acute