Acute spinal cord compression paralysis

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The author believes that mechanical deformation rather than anoxia causes spinal cord compression paralysis. The outlook for recovery of function is therefore better than it would be if anoxia were the cause. Recovery from immediate complete sensorimotor paralysis with spinal subarachnoid block may occur if laminectomy is prompt. Patients suffering from incomplete compressive sensorimotor paralysis may recover without laminectomy, which should be done only if there is no improvement or progressive loss of function in spite of conservutive treatment. The preservation of pain sensation in patients with motor paralysis following acute cord compression is a favorable prognostic sign.

Key Words: spinal trauma • spinal cord injury • spinal compression paralysis • spinal trauma

The immediate reduction of fracture dislocations is desirable to reduce pain and to secure a more stable and mobile spine. The primary object of such reduction in the paralyzed patient, however, is to relieve spinal cord compression. It is only after one has done whatever possible to restore neural function that one should focus on securing a stable spine. The need for treating acute spinal cord compression producing paralysis sometimes arises also in the handling of patients suffering from herniated intervertebral discs, neoplasms, or hemorrhage, either traumatic or spontaneous. Yet there is much controversy over the proper management of victims of spinal lesions producing acute paralysis.

Laboratory and clinical research has elucidated the mechanism of spinal compression paralysis and some factors governing recovery. These studies have provided evidence necessary to determine, usually, the advisability of laminectomy and the proper time for it in victims of spinal injuries or other compressive spinal lesions causing acute paralysis, and also, on the basis of certain signs, to determine the likelihood of recovery in some patients. This report summarizes these data.

Summary of Experimental Evidence on Mechanism of Spinal Compression Paralysis

Whether the paralysis caused by spinal compression is due to mechanical deformation of tissue or to anoxia has long been controversial. In evaluating their respective roles, a comparison of the electrophysiological effects produced by compressing the spinal cord with those caused by ischemia and anoxia was made.

Figure 1 shows a comparison of the action potentials taken from the dorsum of the dog's lumbosacral spinal cord after stimulating the sciatic nerve in ischemia (left tracing), and following measured spinal cord compression by means of a circular compressor lined by foam rubber (right tracing). Lumbosacral cord anoxia, whether produced by occluding the thoracic aorta, by asphyxia,
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Fig. 1. Recordings showing spinal cord ischemia from thoracic aortic occlusion and cord compression produced mechanically. Disappearance of potentials with compression is reverse of that in ischemia. Compression paralysis must therefore result from mechanical neural tissue deformation, not ischemia. IP = arrival of impulse volley at spinal cord; A = conduction in primary afferent fibers; N = response of interneurons; and P = associated with interneuron and possibly motoneuron discharge.

or by breathing pure nitrogen, modifies the various components in a specific way. The so-called "P" wave, associated with interneuron and possible motoneuron discharge, disappears first, followed by the "N" wave, a response of all interneurons. Next to disappear is the "A" wave, correlated with the conduction in the primary afferent fibers, and finally the "IP" wave, which signals the arrival of the impulse volley at the spinal cord.

If the functional disturbance caused by spinal compression were due to ischemia, one would expect the action potentials to disappear in the same general order. However, the order of disappearance of electrical
activity (Fig. 1, right tracing) is the reverse of that seen in spinal ischemia, spinal afferent impulses disappearing first. The reappearance of potentials after restoration of blood supply and after decompression is in a reverse order to that of their disappearance.

This evidence, then, as well as other data, emphasizes that mechanical deformation rather than anoxia is the main factor in acute paralysis caused by spinal compression. In spinal cord compression, the compressed segment of cord must often be ischemic. However, the interlacing vascular network of the cord, so well demonstrated for the cervical spinal cord in man, must allow for the diffusion of oxygen from the adjacent nonischemic parts through the ischemic segment.

It is fortunate that mechanical deformation rather than anoxia is the main cause of spinal compression paralysis, shown so clearly by the different patterns of electrical inactivation (Fig. 1), because the capacity for recovery of spinal cord function following removal of the compressive force is much greater in the former case. About an hour of complete ischemia kills all nerve cells of the spinal cord in dogs. On the other hand, dogs whose spinal cords had been compressed for 2 hours with forces producing instantaneous sensorimotor paralysis showed full functional recovery. In man, too, recovery from acute total sensorimotor paralysis has occurred when the compressive force was removed within 24 hours. This could not have happened if the paralysis had been the result of ischemia or any type of anoxia. Such clinical experience shows that there may be longer safety periods between trauma and reversibility of function in man than experiments in dogs would seem to indicate.

Favorable Prognostic Sign in Spinal Cord Compression

In a series of experiments, spinal cord compression was arrested at a stage when the dogs showed total motor paralysis and yet retained responses to pain. Full recovery of function in these animals could occur after acute spinal cord decompression even though the compression had been maintained for up to 1 week. Nerve fibers conducting pain sensation are less vulnerable to spinal compression than fibers supplying motor power or position sense. This differential sensitivity parallels fiber size, thick fibers being more sensitive to compression than thin fibers. In my experience with clinical cases of spinal cord compression, position sense and motor power are usually impaired sooner than pain perception. Pain sensation in animals and in man, too, or the presence of some position sense or motor power, however slight, makes the outlook for recovery after removing the compression much more favorable than when there is no pain sensation or no motor power. This is important in clinical prognosis, as shown by the following case.

Case 1

A 37-year-old lawyer was hospitalized with a 2-year history of pain in the left shoulder radiating to the thumb and forefinger. The abnormal neurological findings were markedly weakened left elbow flexion and a decrease in the left biceps reflex. A cervical myelogram showed an extradural filling defect between C5-C6 on the left. At laminectomy an extradural dumbbell-shaped tumor arising from the sixth cervical nerve root, extending outward through the intervertebral foramen, was removed with the involved root. The stumps of the sixth cervical nerve roots and the peripheral nerve appeared normal on inspection and palpation. The thecal sac seemed normal.

The day after operation the patient could move his legs normally but his left hand grip was weak. Thirty-six hours after operation he had complete limb paralysis except for barely perceptible knee and finger flexion. All deep limb reflexes were absent. Pinprick could be felt everywhere though it was slightly blunted over the trunk, abdomen, and extremities. Position and vibratory sense were totally absent below the shoulders. The wound was immediately reopened. There was no hematoma. The dura mater was opened. The spinal cord was severely compressed by a left anterolateral encapsulated tumor which was easily removed. It measured 0.5 cm in diameter and was attached to the C-6 anterior root. The tumor was a neurofibroma. It was not clear whether there was microscopic continuity of the two tumors removed.
The patient made a good recovery and was playing golf 3 months later.

That preserved sensation in the presence of complete motor paralysis in spinal cord injuries also makes the prognosis more favorable than it would otherwise be has been well documented.14

**Timing of Decompression**

Acute complete paralysis in dogs may be totally reversed if spinal cord compression is removed within 2 hours provided the compressive force is not too great. The gradual development of the paralysis over 36 hours due to spinal cord compression in Case 1 made the prognosis more favorable than acutely developing paralysis. However, even acutely developing motor paralysis in man is recoverable if the mass is promptly removed.

Early operation may have favorable results in some cases of acute complete paralysis due to spinal injuries. This is best shown by Wannamaker's results in a large series of spinal cord injuries (208 patients with complete sensorimotor paralysis) sustained during the Korean conflict. Recovery, in all cases referable to the long tracts of the spinal cord, was incomplete in 52 and complete in seven patients. In only 28% of the patients was the laminectomy done within 24 hours. More recoveries would probably have resulted if it had been possible to operate sooner on more of these patients. The patients with complete sensorimotor paralysis due to spinal cord injuries recently reported are difficult to assess because of inadequate documentation.

Even patients who have sustained acute complete sensorimotor paralysis due to spinal tumor and edema may show dramatic recovery after decompressive laminectomy and tumor removal, as illustrated in Case 2.

**Case 2**

A 62-year-old man complained of pain between the shoulder blades and a sensation of coldness in the hands for 3 months with increasing weakness of his hands for 5 weeks. He had bilateral interosseous atrophy, moderate weakness of the hands, and decreased pinprick appreciation over the thumbs and radial sides of both forearms. Myelography showed an hourglass constriction of the thecal sac at the level of the C7-T1 vertebra. A laminectomy of C-7, T-1, and T-2 revealed a metastatic carcinoma surrounding the dural sac and infiltrating the body of T-1. The spinal cord was widely decompressed but the tumor incompletely removed.

When the patient awoke from the anesthesia, he showed a complete sensorimotor paralysis of the lower limbs with a sensory level at the outer parts of the upper arms (C-5 dermatome). The knee and ankle jerks, abdominal reflexes, and plantar responses were absent. Queckenstedt testing showed evidence of a spinal subarachnoid block, and a myelogram showed a complete arrest of Pantopaque at the lower border of the laminectomy at T-2. The wound was re-opened and the laminectomy was extended to include T3-T4. There was a 3 mm layer of tumor underlying the T-3 vertebra and surrounding the cord. The tumor was excised. The dural sac did not pulsate. The outer layer of the dura mater was opened from C7-T4; there was visible expansion of the inner dural sac which then began to pulsate. The operation was completed approximately 2 hours after the first one ended.

The next morning the man was able to move his legs normally and sensation had fully returned, the reflexes returning the following day.

**Comment**

The acute spinal compression in Case 2 was probably attributable to the combined effects of compression by the residual tumor augmented by postoperative edema. This case is a striking example of the efficacy of decompressive laminectomy done within a few hours after the onset of acute compressive paralysis in restoring spinal cord function. In such circumstances external dural decompression may be effective without incurring the risk of exuding spinal cord when the entire thickness of the dura mater is opened. If external dural decompression fails to restore cord pulsation, then the entire thickness of the dura may be opened provided measures have been taken to reduce edema by using urea, mannitol, or intravenous steroids such as dexamethasone. The danger of cord extrusion upon opening the dura is greatest within the first 2 or 3 days.
after injury when edema is at its height. If there is certain evidence of an intradural hematoma, or possibly a midline herniated intervertebral disc, one that could not more safely be approached by an anterior cervical or lateral thoracic route, the dura must be opened. Midline posterior pial incision to increase the decompressive effect should not be used because the diffluent tissue that escapes from the incision in the acutely compressed cord carries with it some nerve tissue which might be expected to recover if not disturbed.15

Treatment of Acute Complete Sensorimotor Paralysis

Animal experiments16,18 and clinical experience24 as outlined above show that it is possible to reverse the immediate complete sensorimotor paralysis due to spinal compression provided the compression is relieved within a short time and provided the compressive force is not too great.

In any case of immediate complete sensorimotor paralysis caused by vertebral fracture or fracture dislocation, precious time should not be lost in manipulating the spine or in other nonsurgical methods of treatment. Laminectomy should be done immediately to decompress the spinal cord, and, if there is dislocation, attempts to reduce it should be made at that time. Patients suffering from immediate complete paralysis following spontaneous hemorrhage or trauma, even if unaccompanied by plain x-ray abnormality, should have immediate laminectomy. In these patients, however, a test for spinal subarachnoid patency should precede the operation to exclude those rare cases of immediate paralysis resulting from spinal cord concussion. These patients show no spinal subarachnoid block and improve rapidly.

This concept of treating patients with immediate sensorimotor paralysis associated with a spinal subarachnoid block is illustrated in Fig. 2. The irreversible changes are indicated by the black areas representing hemorrhages and severed or contused nerve fibers (Fig. 2 A) resulting from the direct mechanical effect of the blow. The destruction secondary to these rather immediate changes is accentuated by the secondary edema compressing the fibers, particularly when the swollen cord is compressed against the dura and the unyielding bone. Nothing can be done to reverse the paralysis resulting from the severed nerve fibers. However, if the secondary edema could be prevented or its effect minimized by decompression, it is possible that those fibers physiologically blocked by the edema and compression might be salvaged. Complete destruction of the spinal cord might thus be averted, and recovery of function, at least partial, achieved in some cases. If this pressure is allowed to go unchecked, irreversible damage will result (Fig. 2 B). Probably the beneficial effects1 of cooling the experimentally injured spinal cord are due to reducing associated edema. The administration of steroids and also urea4 may act similarly.

The experiments of Ducker and Hamit4 using 375 gm-cm forces delivered to the spinal cord in laminectomized dogs showed that intramuscular dexamethasone, as well as local hypothermia to the cord, favored "statistically significant improvement and recovery of neurological function." Their dogs had received the benefit of decompressive laminectomy before the treatment. They conclude quite plausibly: "With the exception of certain medical centers set up to properly administer and to follow local hypothermia treatment12 of spinal cord patients, dexamethasone medication is recommended for acute spinal cord injury when there is no persistent anatomical compression of the cord, and especially when an incomplete or threshold traumatic lesion is found or suspected."

Patients suffering from penetrating wounds of the spine should have operations for debridement, removal of foreign bodies, if they are accessible, and the closure of dural defects. Late laminectomy in patients with complete sensorimotor paralysis following trauma may benefit patients by the recovery of muscles innervated by decompressed nerve roots. Significant recovery of function referable to the long tracts of the spinal cord does not occur, in my experience, after late laminectomy.

The following case illustrates an unusual mechanism of spinal cord injury, one suggested by Taylor's39 cadaver experiments and supported by Waltz35 study of patients suffering from quadriplegia without radio-
Fig. 2. Diagrams illustrating the effect of acute compression causing immediate complete paralysis associated with subarachnoid block (A). Irreversible neural damage may result partly from trauma at the moment of impact and partly from subsequent edema and its accompaniments. If these changes persist beyond a certain critical period they may lead to irreversible destruction of neural elements (C). If compression and edema are relieved by early laminectomy (B) the pathological trend may be reversed (D) and function recovered in some patients. The open circles and starshaped symbols represent undamaged nerve fibers and nerve cells; the blackened symbols represent hemorrhages and nerve cells and fibers irreversibly damaged.
graphic evidence of vertebral fracture or dislocation. Such patients should be regarded as suffering from injuries possibly due to ligamentum flavum buckling caused by forcible hyperextension.  

Case 3

An elderly man was hospitalized after having been found lying helpless. He was quadriplegic; all reflexes of the trunk and extremities were absent. There was loss of pinprick and position sensation over the trunk and extremities. A tracheostomy was done and there was sudden cardiac arrest. External cardiac massage followed by open cardiac massage restored rhythmic cardiac contractions. Cervical spine films revealed no abnormality. A Queckenstedt test was normal. The cerebrospinal fluid was clear. The attending neurosurgeon, suspecting a cervical fracture dislocation that did not show on x-ray, applied skeletal traction. Two days later the patient began moving his fingers. His blood pressure remained under 80 systolic unless levophed was given intravenously. On the fifth day he died.

Autopsy revealed a left anterior coronary artery thrombosis. The tip of the C-3 spine was fractured. There was no other bone injury. There was no herniated intervertebral disc, and there was no encroachment of bone upon the spinal canal. The anterior and posterior longitudinal ligaments were intact. The spinal cord at C-3 was contused. There was gross destruction of the gray matter at C-3 (Fig. 3) and of the left posterolateral funiculus. The central hemorrhage extended from C-1 to C-4. There were a few small hemorrhages elsewhere in this area, and edema was observed microscopically. There was no thrombosis of the anterior spinal artery or of any of the other spinal blood vessels.

Comment

Barnes,  and also Brain, et al.,  have described patients in whom severe paralysis followed traumatic hyperextension of the cervical spine, the site of spondylosis. Their pathological observations have demonstrated severe damage to the spinal cord without any evidence of fracture dislocation, herniated intervertebral disc, or ruptured posterior longitudinal ligament. Probably inward-bulging of the ligamentum flavum contributes to the spinal cord damage in these patients. The ligamentous infolding was probably the main factor producing the spinal cord damage in my patient because there was no appreciable evidence of spondylosis. If there had been a spinal subarachnoid block, immediate decompressive laminectomy would have been indicated in view of his immediate complete sensorimotor paralysis. In fact, if the patient had not begun moving his fingers 2 days after injury and, provided his general condition was satisfactory, myelography would have been undertaken to determine whether the spinal cord was compressed. I have seen normal Queckenstedt tests in patients with blocks on Pantopaque myelography. The viscosity of the

![Fig. 3. Case 3. Autopsy specimens of the spinal cord showing central hemorrhage and marked destruction of the posterior columns mainly on one side, which are probably the result of the ligamentum flavum infolding due to hyperextension of the spine.](image-url)
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Pantopaque accounts for such occurrences. The difficulty in performing a myelogram by the usual lumbar technique in patients with cervical injuries, particularly after applying skeletal traction, may be circumvented by using a lateral cervical puncture as used by Mullan, et al. for percutaneous cordotomy.

Lesions such as that in Fig. 3 occurring either spontaneously or after trauma may produce a central spinal cord syndrome. The inner spinal pathways suffer the greatest damage so that the motor loss is greater in the upper than in the lower extremities. The disturbed sensation is of the syringomyelic type in which pinprick appreciation is more impaired than tactile or discriminative sensation. Decompressive laminectomy is indicated if the sensorimotor paralysis is complete and associated with a spinal subarachnoid block, or if the paralysis, although not quite complete, is associated with a spinal subarachnoid block.

Queckenstedt testing or myelography is important to avoid an unnecessary operation in a patient whose paralysis may be due to destruction of spinal cord tissue rather than compression primarily. I have not encountered untoward effects of carefully-performed Queckenstedt testing. However, care is necessary to avoid the sudden escape of cerebrospinal fluid, which might convert a partial into a complete block with further compression.

Treatment of Acute Incomplete Sensorimotor Paralysis

Patients suffering from traumatic partial interruption of cord function often retain considerable function, or recover completely without a laminectomy. The following case is illustrative.

Case 4

A 28-year-old policeman was hospitalized 1 hour after having been hit by an automobile. Examination revealed complete lower limb paralysis. He was capable of weak flexion and extension of his elbows. Shoulder movements were normal. Pain and touch modalities were lost from the second rib downward, although sensation over the upper limbs was normal. Deep and superficial tendon reflexes of all extremities were absent. Position sense was absent in the lower extremities. There was tenderness over the C-5 and C-6 vertebrae, at which site x-rays showed a marked forward displacement of C-5 on C-6. A Queckenstedt test revealed no evidence of a spinal subarachnoid block. Skeletal traction was applied and the fracture reduced (Fig. 4).

About 2 hours after skeletal traction was applied, slight hand movements returned, and perception of touch and pinprick was regained below the second rib. The next day the legs could be voluntarily moved and power in the hands and elbows increased. Power and sensation improved steadily, and 6 weeks after the injury traction was discontinued. A Forrester (four-poster) brace was applied, and the patient allowed out of bed. Nine weeks after his injury, power in the lower limbs was normal although hand grips were moderately weakened and pinprick sensation below the second rib was somewhat diminished. Clonus at the knees and ankles persisted, but the right plantar response had become flexor. Examination of the patient 7 months after his injury showed normal lower limb function. The knee and ankle clonus had disappeared and the abdominal and cremasteric reflexes had returned. There was slight weakness of flexion and extension of the left elbow and the left hand grip. Sensation was normal. The plantar responses were normal.

Fig. 4. Case 4. Left: X-ray film showing fracture dislocation of C5-C6 before treatment. Right: X-ray film after reduction by skeletal (tong) traction; there is a small “tear-drop” fracture of C-7.
Immediate laminectomy in this patient was not done because of the absence of a cerebrospinal fluid block. Moreover, the paralysis of sensorimotor function below the level of the injury was incomplete. Therefore, conservative treatment by skeletal traction was indicated. If immediate complete sensorimotor paralysis associated with a spinal fluid block had been present, prompt laminectomy would have been done. The anterior approach to the cervical spine is useful for removing intervertebral disc extrusions, a rare cause of immediate complete sensorimotor paralysis in spinal injuries. It also permits anterior interbody fusion, which produces excellent spinal stability and allows early mobilization of the patient. However, the prime consideration in treating these patients who show spinal subarachnoid block is decompression of the spinal cord, which cannot as effectively be achieved by the anterior as by the posterior approach. Moreover, laminectomy, by permitting a more extensive exposure of the cord, affords a better opportunity to remove broken laminar fragments and foreign bodies. The decision on spinal fusion may have to be delayed a week or two, or longer, after prime consideration has been given to the advisability of decompression as the initial procedure in attempting to relieve the paralysis.

In the following patient immediate operation was not done, even though a cerebrospinal fluid block was present, because the paralysis was incomplete.

Case 5

A 40-year-old physician was hospitalized and examined 3½ hours after having fallen from a second-story porch. He was unable to move his legs and had pain along the inner aspect of his arms. Examination revealed nearly complete lower limb paralysis except for slight toe movements. Pinprick sensation was lost from the third rib downward and also along the inner aspect of the arms and forearms. Position sense of the thumbs and great toes was intact. The ankle jerks were absent but the knee jerks were present.

There was tenderness over the C-6 spinous process at which site x-rays showed a marked forward displacement of C-6 on C-7. There was a complete cerebrospinal fluid block on Queckenstedt testing. Skeletal traction was applied. Traction was gradually increased to 40 lbs over the course of 3 days without reducing the dislocation. The spinal subarachnoid block persisted on Queckenstedt testing. The patient had lost all voluntary power and sensation in his lower limbs, and therefore on the fourth day after his injury he was operated upon. The C-6 and C-7 facets were locked. The superior margins of both the laminae of C-7 and the articular facets were removed after which the introduction of a periosteal elevator between the facets allowed the sixth lamina to move posteriorly and resume its normal position. Inspection between the laminae revealed cord pulsation and a normal epidural space. Therefore, complete laminectomy was unnecessary.

X-rays revealed normal spinal alignment. The traction was removed 7 days after operation. Pinprick appreciation began returning 10 days after operation. Movement of the toes began 6 days later. Recovery increased steadily, and the patient was able to walk well with the aid of a cane 5 months after his injury, when he was last seen.

Comment

Because the paralysis advanced in this patient, and furthermore since it was impossible to reduce the severe dislocation by skeletal traction, partial laminectomy and open reduction of the dislocation were imperative. It is possible that earlier laminectomy might have hastened the patient's recovery.

A policy of watchful waiting is reasonably safe provided the loss of spinal function is not complete. If during this period paralysis advances or if the paralysis although stationary is severe, laminectomy should be done provided there is a spinal subarachnoid block or a configuration on x-ray or myelography suggesting extrinsic pressure on the cord. Many patients with partial spinal lesions of traumatic origin may be spared the ordeal of an operation without jeopardizing their recovery by following this policy.

When the partial paralysis results from compression by a neoplasm known, with a fair degree of certainty, to be malignant, radiation therapy or chemotherapy or both may be used even though a spinal subarachnoid block exists. Recovery of function fre-
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frequently follows such medical management and a myelographic block may disappear. If, however, the paralysis is not improved by medical management alone, then laminectomy and removal of the tumor should be undertaken. Our studies show that total removal of spinal cord tumors, even malignant ones, is much more apt to be followed by satisfactory recovery than is partial removal. If the paralysis is complete in malignant spinal neoplasms and accompanied by a spinal subarachnoid block, then immediate laminectomy for removal of tumor is preferable to medical management. I am not aware of recovery of function with medical management alone in such patients.

Outlook for Acute Sensorimotor Paralysis Without Operation

In human cases of fracture dislocation with immediate total sensorimotor paralysis, the outlook without laminectomy is hopeless, in my experience, if a spinal subarachnoid block is present. In nine surgically unexplored cases of spine injuries in which complete sensorimotor paralysis immediately followed the injury, not one made a useful functional recovery. I know of no well-documented case with complete spinal subarachnoid block in which the patient recovered useful motor function without laminectomy, if there was immediate and complete loss of sensation and motor power below the level of the spinal cord lesion.

Guttmann's opposition to laminectomy as an immediate measure in managing traumatic paralysis is based on the assumption that significant additional trauma will be inflicted during the operation. But experienced neurosurgeons can perform laminectomy in these patients without significant trauma. Moreover, one questions the validity of his objection since patients who develop immediate total paralysis below a spinal lesion do not recover spinal function referable to the long tracts without laminectomy. On the other hand, considerable return of function may follow the operation. Guttmann's statement that "the presence of a subarachnoid block in the early stage following spinal cord injury is of no diagnostic value in differentiating between oedema, intramedullary hemorrhage or pressure from dislocated bone ..." is true but irrelevant. The point is that compression of any sort, if relieved early, may be reversible, as we have shown, provided its magnitude is not too great. This one cannot know in advance, nor can one foretell the extent of the irreversible damage done by laceration and contusion. For practical purposes one must assume that recovery of function may occur and treat these patients in the light of rational principles evolved from laboratory and clinical researches. Accordingly, laminectomy should be done at the earliest possible time in patients with immediate complete sensorimotor paralysis accompanied by a spinal subarachnoid block following spinal injuries or other compressive lesions unless there is clear-cut evidence of a severed spinal cord. Recovery, at times complete, may follow decompressive laminectomy done within 24 hours or longer in some of these patients. Probably in a majority of these patients little or no functional improvement will occur in the paralyzed limbs or bladder after operation. It is not surprising that even very early laminectomy may not result in recovery from paralysis in spinal cords that are transected or severely contused. The significant fact is that expectant treatment offers no hope in these patients but that, on the other hand, recovery may be brought about by early decompressive laminectomy in some cases in which continued compression irreversibly damages the spinal cord.

Conclusions

Mechanical deformation rather than any type of anoxia is the main cause of spinal cord compression paralysis. This is fortunate because the period of mechanical compression compatible with recovery is much greater than the comparable period of anoxia. Recovery from immediate complete sensorimotor paralysis accompanied by spinal subarachnoid block caused by spinal compression does not occur without laminectomy. Recovery may sometimes occur, however, if laminectomy is prompt. Laminectomy is therefore urgently indicated within the shortest possible time in such patients.

Patients suffering from incomplete sensorimotor paralysis due to spinal compression may recover with conservative treatment. For fracture dislocations, conservative treatment includes skeletal traction or postural...
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reduction; for malignant tumors (metastatic or not), compressing the spinal cord, conservative treatment includes radiation or possibly chemotherapy. Laminectomy should be done in these patients only if there is progressive loss of function in spite of conservative treatment, or if such management fails to relieve a severely paralyzed patient in whom myelography has demonstrated cord compression.

The presence of pain sensation in patients with motor paralysis following acute spinal cord compression is a favorable prognostic sign.

References

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