REVIEW ARTICLE

Current concepts in the immediate management of acute spinal cord injuries

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The management of acute spinal cord injuries has changed considerably during the past 10 years owing to new information about the pathophysiology of cord trauma and new diagnostic and treatment methods. It is now known that the cord suffers not only from the immediate physical effects of trauma, but also from secondary pathologic processes, such as ischemia and edema, which are treatable in the first few hours after injury. New neuroradiologic and neurophysiological techniques, such as the recording of the somatosensory evoked potential, increase the accuracy of diagnosis and prognosis in the acute phase. Current immediate treatment includes the administration of steroids and mannitol, with careful attention to respiratory and cardiovascular homeostasis, to overcome post-traumatic ischemia and edema, and immobilization of the spine with devices such as the halo. New surgical procedures are used in selected cases to improve neurologic recovery, to provide rigid immobilization of the spine or to allow earlier mobilization of the patient. The care of spinal cord injuries in the acute phase is facilitated by multidisciplinary units.

Le traitement des blessures aiguës de la moelle épinière a changé considérablement au cours des 10 dernières années grâce aux nouvelles connaissances sur la physiopathologie des traumatismes de la moelle et aux nouvelles méthodes de diagnostic et de traitement. Il est maintenant reconnu que la moelle souffre non seulement des effets physiques immédiats du traumatisme, mais aussi des processus pathologiques secondaires tels que l'ischémie et l'oedème qui peuvent être traités dans les quelques premières heures qui suivent la blessure. Les nouvelles techniques neuroradiologiques et neurophysiologiques, telles que l'enregistrement du potentiel somatosensitif évoqué, augmentent la précision du diagnostic et du pronostic dans la phase aiguë. Le traitement immédiat actuel comprend l'administration de stéroïdes et de mannitol, tout en portant une soigneuse attention à la respiration et à l'homéostase cardiovasculaire afin de combattre l'ischémie et l'oedème post-traumatiques, et l'immobilisation de la colonne vertébrale avec des appareils tels que le halo. De nouvelles interventions chirurgicales sont utilisées dans des cas choisis afin d'améliorer la guérison neurologique, d'assurer une immobilisation rigide de la colonne ou de permettre la mobilisation plus rapide du patient. Les soins aux blessures de la moelle épinière dans la phase aiguë sont facilités par les unités multidisciplinaires.

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Acute injury to the spinal cord is a very serious injury for a number of reasons. In upper cervical injuries and in older people the mortality is high, owing mainly to respiratory problems. The morbidity of severe spinal cord injury is great because of the profound effect on most body functions and the social and psychologic effects on the patient, the family and society. Indeed, the cost to society of each new case of traumatic quadriplegia amounts to hundreds of thousands of dollars in terms of lifelong medical care and lost earnings.

During the past 10 years there has been intensive research into the pathophysiology of acute spinal cord injury,[1-3] and this has resulted in a greater understanding of the acute changes occurring in the cord after trauma. In turn, there have been many innovations in the management of patients with acute spinal cord injuries that promise to improve the prognosis for neurologic recovery. These innovations include new diagnostic methods, new methods of immediate medical and surgical treatment, and the development of acute spinal cord injury units for the management of the acute phase of the condition (Table I). These units are extensions of the paraplegic units that were developed several decades ago for the management of the rehabilitation phase of spinal cord injury.

This paper will discuss the results of recent research into acute spinal cord injury and describe the current immediate management of patients in the acute spinal cord injury unit at Sunnybrook Medical Centre, which, since its inception in 1974, has treated more than 100 patients with such injuries. It is hoped that this paper will be of assistance to the many general practitioners and specialists who are in-

volved in the initial management of acute spinal cord injuries.

Pathophysiology of acute spinal cord injury

Severe trauma to the spinal cord rarely causes complete anatomic transection of the cord initially,[3,4] although it often causes immediate and complete disruption of cord function, or spinal shock. The initial pathological picture often appears to be relatively benign, with scattered hemorrhages in the grey matter and some edema of the white matter (Fig. 1A).[1] However, several weeks after such an injury the cord appears as if it had been anatomically transected because much of the cord tissue at the injury site has become necrotic and subsequently replaced by large cavities with surrounding gliosis and fibrosis (Fig. 1B).[1] The initial physical forces of trauma, such as cord compression, laceration or stretching, initiate a series of pathologic changes that result in almost complete anatomic transection of the cord at the injury site. A great deal of research has been done in the past few years to determine the mechanisms responsible for these destructive post-traumatic changes. Histologic studies of cord tissue days to weeks after injury reveal extensive necrosis of grey and white matter, the picture of infarction (Fig. 1B).[1] Our hypothesis, which is shared by others,[2] is that the initial trauma produces a secondary chain of events that results in infarction of the cord due to ischemia (Fig. 2). Mechanical forces acting on the cord in the acute stage produce vascular injury and resulting ischemia. This hypothesis has important therapeutic implications in that all may not be lost with the initial trauma and, therefore, treatment directed toward reversal of the ischemia may prevent infarction and improve recovery.

Studies have indicated that there are at least two types of vascular injury in the spinal cord after trauma. The first is an immediate injury to small blood vessels in the cord, especially the venules of the grey matter,[5] which results in numerous hemorrhages, particularly into the grey matter, during the first few minutes after trauma (Fig. 1A). Early hemorrhage into the grey matter appears to be a basic response, being found in virtually all forms of acute cord injuries, including experimental. In severe trauma, bleeding into the grey matter may be so extensive that it results in central hematomyelia, for which myelotomy has been advocated by some.[6,7]

The second type of vascular injury in the spinal cord is much more destructive than central hematomyelia because it results in severe post-traumatic ischemia, which, if not relieved, leads to infarction of both the grey and the white matter. Regeneration of the axons in the long tracts of the spinal cord sufficient to restore function has not been achieved; therefore, once post-traumatic infarction has occurred, the accompanying neurologic deficit is irreversible. Fortunately, post-traumatic ischemia appears to be reversible, which probably explains why several forms of experimental therapy have resulted in recovery of cord function. These successful forms of therapy include local hypothermic or normothermic irrigation and administration of steroids, diuretics and hyperbaric oxygen;[1] they will be described later.

In our laboratory we have shown

Table I—Innovations in the acute management of spinal cord injuries

Speed to interrupt autodestructive pathophysiologic processes to prevent post-traumatic ischemia and infarction of the spinal cord.
Diagnostic aids: neuroradiologic and neurophysiological.
The acute spinal cord injury unit, developed to provide multidisciplinary acute care.
New methods of medical and nursing management: administration of steroids and diuretics, intermittent catheterization and maintenance of blood pressure.
New methods of surgical management: halo devices, internal fixation, anterior approaches to the spinal cord and closed reduction under general anesthesia.

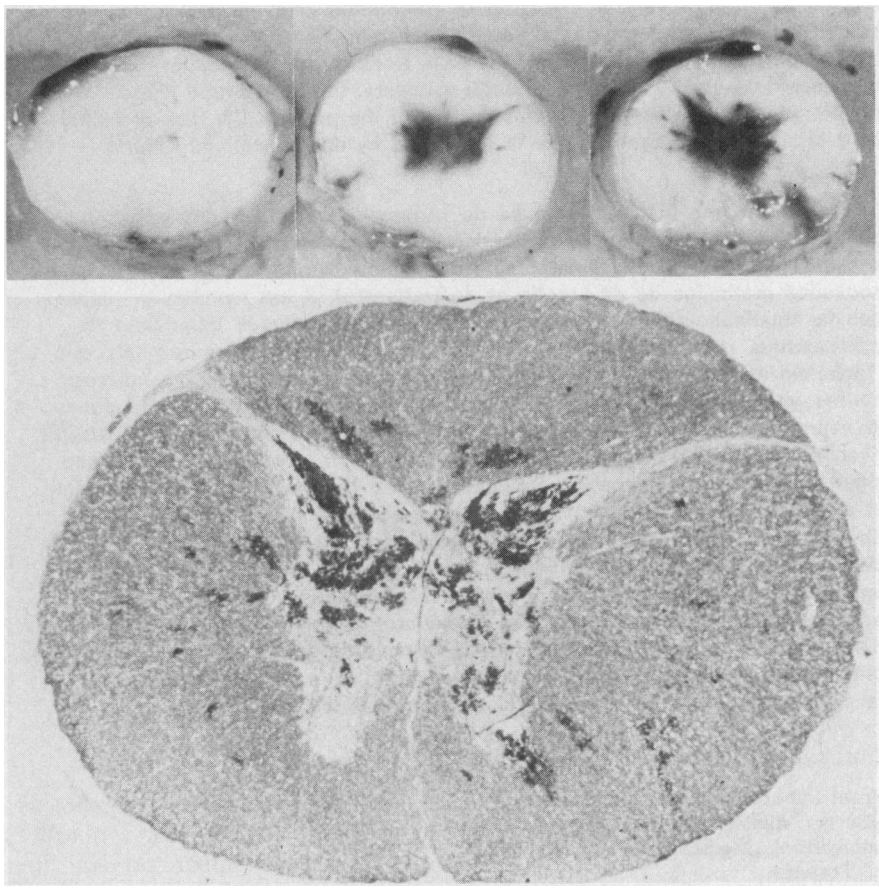

FIG. 1A—Sections of thoracic spinal cord of monkey 1 minute after injury by acute extradural spinal cord compression. Top: left, 3 cm from injury site; middle, 5 mm from injury site; right, at injury site. Bottom: microscopic section from injury site, showing hemorrhages, mainly in grey matter (hematoxylin–eosin–Luxol fast blue).

that ischemia of the spinal cord begins immediately after trauma and persists for at least 24 hours after severe injury.[8,9] The ischemia involves both grey and white matter and spreads for a considerable distance in the cord proximal and distal to the injury site. By 24 hours after trauma, ischemic zones can be identified histologically as infarcts. Other investigators have also documented a marked reduction of spinal cord blood flow after trauma.[10-12] The concept of post-traumatic ischemia is also supported by other pathophysiologic and biochemical features of acute spinal cord injury, such as the severe reduction in oxygen tension of the cord[13] and the marked accumulation of lactic acid at the cord injury site.[14] The edema that accompanies spinal cord injury may also be due to the effect of ischemia on the blood vessel walls.

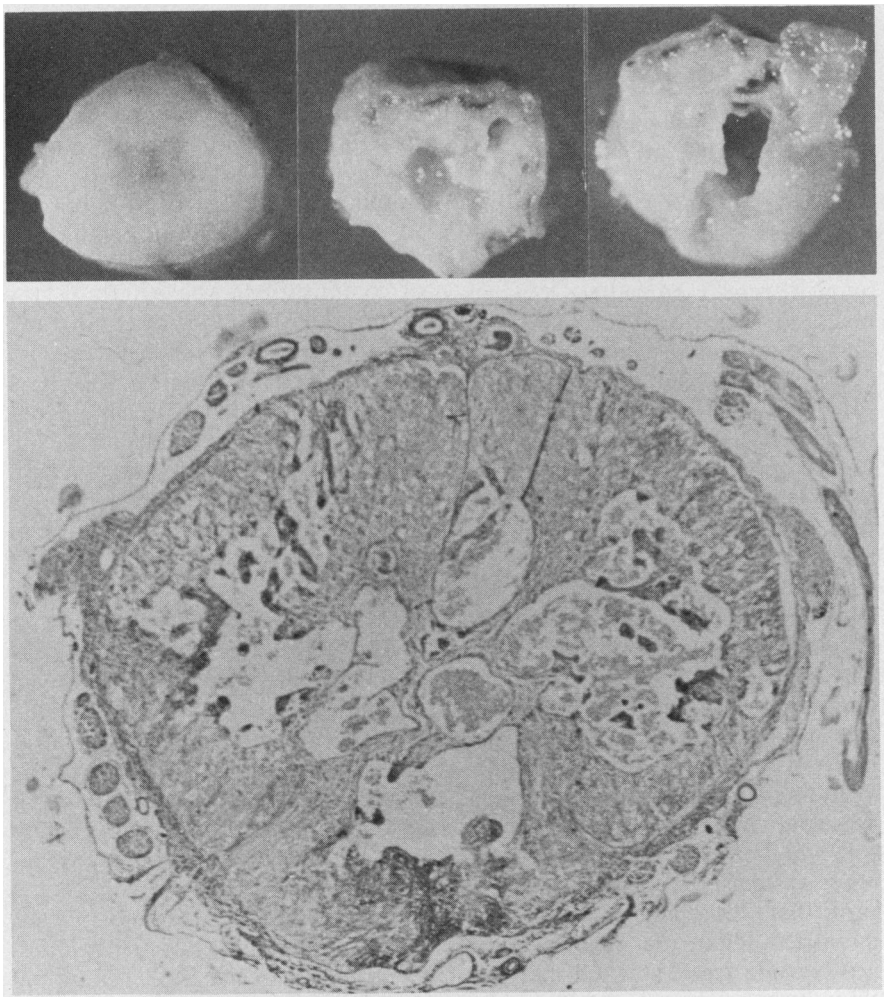

FIG. 1B—Sections of thoracic spinal cord of monkey 3 months after similar injury. Top: left, 3 cm from injury site; middle, 5 mm from injury site; right, at injury site. Bottom: microscopic section from injury site, showing marked disruption and cavitation of grey and white matter (hematoxylin–eosin–Luxol fast blue).

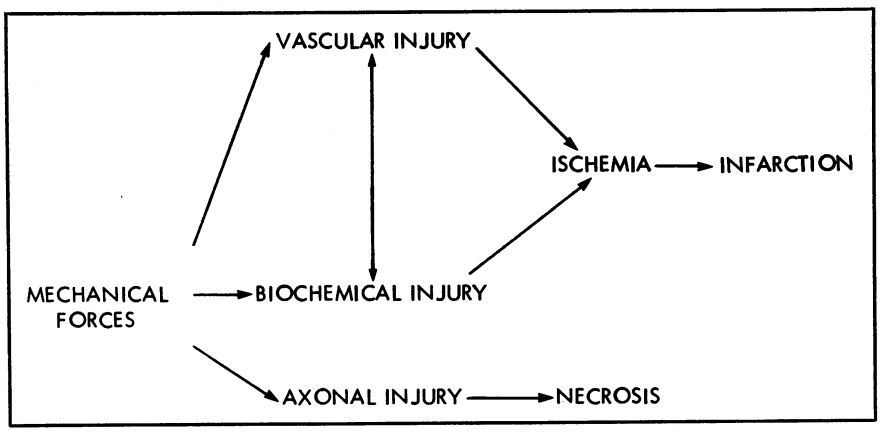

FIG. 2—Hypothesis for pathophysiology of acute spinal cord injury, indicating role of post-traumatic ischemia and infarction of spinal cord.

The mechanism of this secondary vascular injury leading to post-traumatic ischemia is still unknown. Osterholm[2] postulated that the biochemical injury also involves an accumulation of norepinephrine at the injury site, and that this amine causes vasospasm, which in turn causes ischemia. However, this theory remains unproven. Other vasoactive amines may play a role, or the ischemia may be due to other factors, such as increased blood viscosity, thrombosis and precapillary shunting.[15]

In addition to the vascular and biochemical injuries, direct mechanical forces on the cord cause other physical injuries. In terms of function the most important is axonal interruption (Fig. 2). This can occur from laceration, compression or stretching of the cord. The result of axonal interruption is permanent loss of function because, as was noted previously, central axons do not regenerate sufficiently to restore function. Although some central axonal regeneration is now known to occur,[16,17] clinically significant functional restoration does not occur, and there is no known way of promoting functional recovery on the basis of central axonal regeneration.

Besides the pathological changes, the initial physical forces on the cord cause physiologic transection of the cord, which produces immediate loss of motor, sensory, autonomic and reflex function below the level of the lesion. This condition, termed spinal shock, lasts for a variable period — days to weeks in severe injuries, but only minutes to hours in slight injuries. In most instances if spinal shock is still present by the time the patient reaches hospital the injury has been a severe one and the manifest neurologic deficit is due to structural cord damage rather than spinal shock. The mechanisms underlying spinal shock remain unknown, although Eidelberg,

Sullivan and Brigham[18] have presented convincing evidence that spinal shock is due to the increased extracellular concentration at the cord injury site of potassium that has leaked from cells.

The time factor

It is not known how long a period of post-traumatic ischemia is compatible with recovery. There is evidence that many factors affect the "time of no return", the most important of which is the severity of the initial trauma. In our experiments on acute cord compression in rats, recovery was directly related to the duration of compression.[19] Similarly, we recently found that with extradural compression of the spinal cord for 3 hours only 4 of 20 monkeys recovered, whereas when the compression was relieved at 1 hour 11 of 20 monkeys recovered — a significant difference ($P < 0.05$) (unpublished data, 1978). Tarlov[20] had also found a direct relation between duration of cord compression and recovery in experiments with more slowly developing and longer lasting compression. Our studies of post-traumatic spinal cord blood flow in monkeys showed that the ischemia was greater and involved a longer segment of the spinal cord when the duration of cord compression was longer.[9] Edema has also been noted to increase progressively with time after spinal cord trauma.[21]

The cord cooling experiments of Albin and colleagues[22,23] have also provided evidence concerning the critical nature of the interval between trauma and treatment. Animals treated with hypothermic irrigation 4 hours after acute spinal cord trauma produced by weight dropping showed remarkable recovery,[22] but if the cooling was delayed until 8 hours after trauma the therapeutic benefit was lost.[23]

Definite clinical confirmation of the importance of the time factor is still lacking. However, there is some recent clinical evidence that early treatment, such as the reduction of fracture–dislocation within 2 hours of trauma, may bring substantial reward, even improvement of the neurologic deficit in complete spinal cord injuries.[24,25]

Thus, recent studies have shown that important changes occur in the cord during the first few hours after trauma, and that it is possible to improve recovery by early treatment. The mechanisms by which these new remedies, such as hypothermic irrigation and steroid administration, exert their beneficial effects are unknown. The possible mechanisms include relief of ischemia and reduction of edema.

Current clinical management of acute spinal cord injuries

Responsibility for the care of patients with acute spinal cord injury rests jointly with all those called upon to render immediate management to such patients. The ambulance attendants must carefully extract the patient, immobilize the spine and provide first aid during transportation, with special attention to the respiratory assistance often required by patients with cervical or thoracic cord injuries. The emergency department physician must take a careful history of the mechanism of the injury and determine whether there have been any changes in the patient's function from the time of the injury. The primary physician must also perform a complete examination, including assessment of neurologic function, palpation of the entire spine and a search for injuries to other systems. He or she must ensure that the spine remains immobilized, that the bladder does not become overdistended and that skin pressure areas, such as the sacrum and the heels, are padded. The ambulance attendants and primary physician are no less responsible for care than the specialist team rendering specific treatment.

Table II outlines the principles of the acute management of spinal cord injuries in our unit. Early hemodynamic and respiratory support and the administration of steroids are important, as are early immobilization of the spine and reduction of dislocations. Persisting cord compression in incomplete spinal cord injuries must be detected and treated. Restoration and maintenance of spinal alignment and stability are considered important for early mobilization of the patient, for reduction in the chance of complications, the length of stay in hospital and the costs of care, and for improvement in the recovery of function. The following sections describe many recent innovations in diagnosis, medical and nursing management, and surgical treatment, several of which have become essential components of management in our unit.

Diagnostic techniques

The diagnosis of spinal cord injury has been markedly improved during the past few years by the introduction of several new neurophysiological and neuroradiologic techniques, and by the refinement and adaptation of several older techniques to the immediate management of patients with acute spinal cord injuries.

Neurophysiological techniques: Neurophysiological tests of spinal cord function augment the information obtained from clinical neurologic examination. For example,

Table II—Principles of acute management of the injured spinal cord and spinal column

Hemodynamic and respiratory support: ensure normal blood pressure and respiration to counteract post-traumatic ischemia and infarction of the spinal cord.
Administer steroids (and mannitol if possible) to treat spinal cord edema.
Spinal immobilization is essential for spinal instability to provide the best milieu for spinal cord and nerve root recovery, to decrease pain and to allow rapid mobilization of the patient.
Spinal immobilization can be achieved with halo devices or operative fusion anteriorly or posteriorly, with or without internal fixation devices.
Laminectomy is useless for most immediately complete lesions of the cervical or thoracic cord, but may be indicated in selected cases of complete conus medullaris or cauda equina lesions with persisting compression.
Persisting cord compression in selected cases of incomplete lesions should be relieved immediately.
Persisting compression may require surgical decompression from either an anterior or a posterior approach or by skeletal traction (sometimes by closed reduction under general anesthesia).
Open wounds of the spinal cord generally require débridement and closure.
Anterior decompression and fusion for complete lesions of the cervical cord is indicated in some cases for nerve root decompression, stability and early mobilization.
Spinal cord irrigation or myelotomy may be used in selected cases of complete lesions.

they help to confirm the completeness of an injury, especially in unconscious or uncooperative patients, and give information of prognostic value at an early stage. In patients with incomplete lesions of the spinal cord, neurophysiological tests help to determine the response to treatment. In the first few hours after injury the clinical examination cannot differentiate between a totally transected cord and one that is severely damaged yet might recover some function if treatment were instituted early. This is because spinal shock in the early hours or days after trauma limits the diagnostic and prognostic accuracy of the clinical examination. Many clinicians have been influenced by the late Sir Frank Holdsworth of Sheffield, England. His definitions of stability and instability of the traumatized spine[26] remain useful, but his definitions of neurologic completeness after spinal cord injury[27] require amplification. He defined a complete lesion with irreparable cord damage as one in which there was absence of motor and sensory function for 24 hours after the injury. As we have outlined, severe autodestructive changes occur within the first few hours after injury, so that if treatment is delayed, recovery is compromised. Thus, one cannot wait for 24 hours before being able to determine the completeness of a lesion. Holdsworth also placed much reliance on the clinical detection of the return of the bulbocavernosus reflex in determining the completeness of a cord lesion. However, the return of this reflex may take 24 hours or longer, which is now considered an unacceptably long time to wait before instituting treatment.

The most widely used neurophysiological test for assessing spinal cord function after injury has been the recording of the somatosensory evoked potential.[28] The test is done by stimulating a peripheral nerve in the arm or leg and recording the evoked potential from the cerebral cortex through scalp electrodes similar to those used in electroencephalography. A small computer is required for summating the responses to magnify them with respect to the electroencephalogram tracing, which is of larger amplitude. The equipment used in our unit is portable and can be taken to the bedside, emergency department or operating room (Fig. 3). The test takes only a few minutes and is noninvasive, repeatable and well accepted by patients. Perot[28,29] has had the most experience with the use of this test, and our results parallel his in that we have found complete loss of the normal evoked potential when stimulation is performed distal to the site of the lesion in a clinically complete spinal cord injury (Figs. 4A and 4B) and marked changes in the configuration of the response in cases of incomplete spinal cord injury (Fig. 4C). This test has also been very valuable in the assessment of prognosis and response to treatment. We have found that early persistence and progressive normalization of the somatosensory evoked potential antedates the appearance of clinical evidence of improvement and is therefore a favourable prognostic sign (Fig. 5).[30,31]

Other new neurophysiological tests for assessment of cord function after trauma have had less clinical application to date. For example, Cracco[32] has shown that a spinal evoked response can be obtained in humans from the skin of the back after peripheral nerve stimulation. Evoked potentials can also be recorded from the surface of the spinal cord. We have studied this technique in the laboratory in monkeys with spinal cord injuries but have not yet applied it to human patients.[33] The smallness of the surface-recorded spinal evoked potential under certain circumstances may limit its clinical usefulness. It has recently been shown that the electrospinogram, the record of

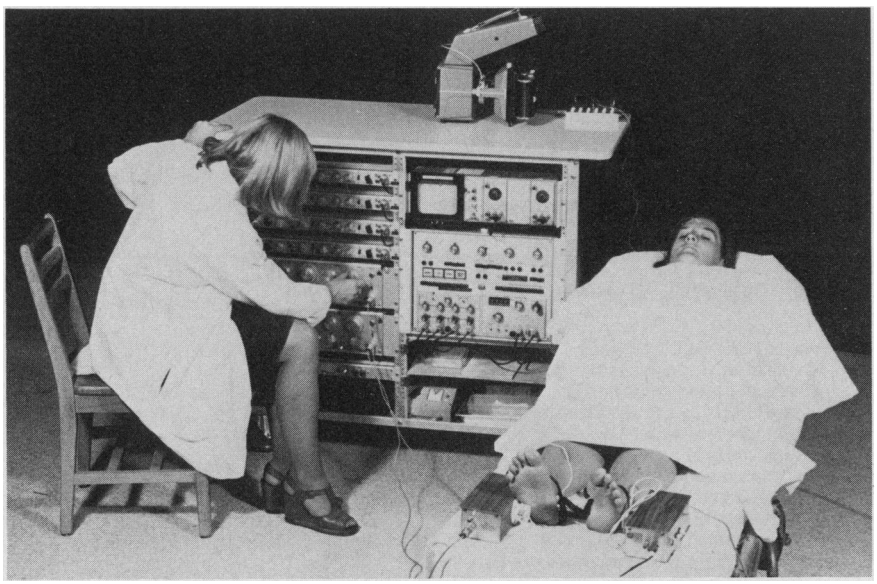

FIG. 3—Recording of somatosensory evoked potentials from tibial nerves. Equipment mounted in trolley for portability; small computer on right side under oscilloscope.

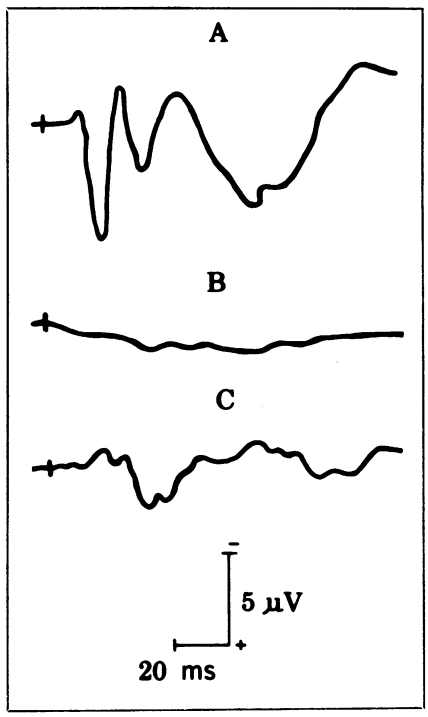

FIG. 4—Somatosensory evoked potentials obtained by recording computer-averaged evoked potential following stimulation of posterior tibial nerve. Stimulus artefact at far left. A = normal response; B = complete absence of response in patient with complete cord injury at T7; C = marked alteration in response in patient with incomplete cord injury at C7.

spontaneously occurring electrical activity measured directly from the cord, is increased in amplitude and frequency distal to the site of cord trauma.[34] The electrospinogram has not been recorded noninvasively.

Several other electrophysiological techniques are available for the assessment of spinal cord function in the injured patient. These include eliciting the tonic vibration reflex and the H reflex[35] and recording the spinal action potential.[36] None of these techniques has been extensively employed clinically. The H reflex depends on conduction through the central grey matter of the spinal cord and can be recorded during the early phase of spinal shock. The spinal action potential depends on efferent conduction in the spinal cord and is therefore a potentially very useful technique; unfortunately, particularly for patients with cervical injuries, this method is highly invasive.

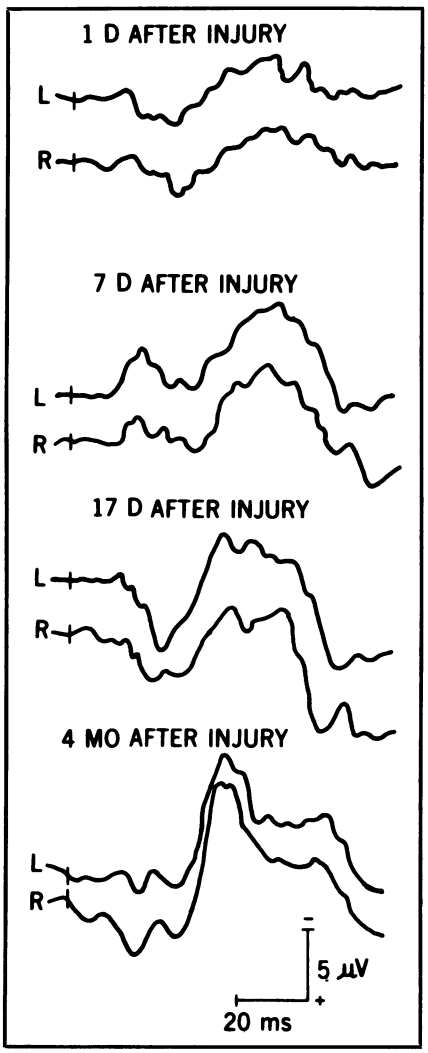

FIG. 5—Somatosensory evoked potentials in patient with incomplete motor and sensory loss distal to site of injury at C6–C7: progressive normalization of wave form between 1 day and 4 months after injury.

Radiologic techniques: Radiologic examination of patients with acute spinal cord injuries and those suspected of having such an injury is often difficult because the examination must be performed quickly and without movement of the spine. Clear visualization of the spine in at least two planes must be obtained. Movement is avoided because it must be assumed that the patient has an unstable spinal injury and that any spinal movement may produce or worsen the cord or nerve root injury. In the typical patient, a young, muscular man, it is often impossible to obtain adequate lateral views of the lower cervical and upper thoracic spine. Definitive lateral views of the suspected area must be obtained because often the lesion is seen only in the lateral view. The clinician and the radiology technician faced with inability to obtain good lateral views should request the immediate assistance of a radiologist. In some instances caudally directed traction on the arms, or "swimmer's" pillar (obtained by exerting rostral traction on one arm and caudally directed traction on the other), or oblique (orthogonal) views of the cervicothoracic junction will improve visualization. However, often even these procedures fail to provide the necessary information, cause pain or waste time at critical stages, and in these instances there should be no hesitation in immediately obtaining spinal tomograms. Machines that can take anteroposterior and lateral tomograms with the patient supine are especially useful in patients with spinal cord injuries. It has been well documented that tomography can identify fractures not seen on plain roentgenograms.[37]

Myelography plays an important role in the diagnosis of persisting compression of the cord, conus medullaris or cauda equina, especially in those with incomplete lesions. Table III shows the indications for myelography in our unit; the procedure, when indicated, is usually performed as soon as possible after admission. We have continued to use conventional myelographic techniques, with a positive contrast medium injected via the lumbar route, although there are now two other ways in which myelography can safely be performed in patients with spinal cord injuries. First, in cervical cord injuries the positive contrast medium can be injected from a lateral C1–C2 approach.[38,39] A portable C-arm image intensifier may be used with the patient in skull traction lying prone on a Stryker frame.[38] The second method, air myelography–tomography, is done with the patient in skull traction, if there is a cervical lesion, and in a lateral decubitus position.[40] Air myelography provides visualization of the entire subarachnoid space and spinal cord. These new myelographic techniques are safe and reliable, and offer the advantage of less movement of the patient during the procedure. Water-soluble contrast agents have not been used extensively in cases of spinal cord trauma but may prove to be very useful because of the clear delineation they give of the anterior and posterior aspects of the subarachnoid space.[41] Radioisotope myelography has been used experimentally for detecting complete block after trauma,[3] but its clinical use has not yet been re-

Table III—Indications for immediate myelography to establish that compression is persisting

With complete neurologic deficit
Cervical or thoracic lesion: no proven indications.
Thoracolumbar or lumbar lesion: indicated when persisting compression of the conus medullaris or cauda equina is suspected but not obvious on plain roentgenograms; unnecessary when persisting compression is obvious (e.g., with a serious dislocation).
With incomplete neurologic deficit
When the procedure is necessary at any level of the spine it is because persisting compression is not obvious on plain roentgenograms. The procedure is unnecessary when persisting compression is obvious. It is usually indicated in patients with normal plain roentgenograms to detect disc herniation.
— Usually necessary in those with a progressive deficit.
— Optional in those with a stable deficit.
— Usually unnecessary in those with an improving deficit.

ported. Spinal cord angiography has beautifully demonstrated certain traumatic lesions in humans, such as ruptured discs and posteriorly protruding vertebral fragments that displace the anterior spinal artery.[42,43] Unfortunately, the procedure is time consuming and requires considerable expertise for its performance and interpretation; thus, it will only be useful in selected cases. At present it is not being used in our unit. Computerized tomography has recently provided accurate neuroanatomic definition of the spinal column and cord in patients with spinal trauma,[44] and it promises to be an excellent technique for detecting persisting compression or displacement of the cord by bone or disc fragments in the spinal canal.

Medical and nursing management

Our acute spinal cord injury unit is a multidisciplinary unit with members from neurosurgery, orthopedics, physical medicine, urology, plastic surgery, neurology, neuroradiology, physiotherapy, occupational therapy, social work and psychology. Most members see each patient shortly after admission, and each patient is discussed at the weekly unit rounds. A skilled nursing staff has been assembled and given added responsibility for day-to-day management decisions. They and the medical staff are assisted by a special studies coordinator, a neurophysiology technician and a spinal cord injuries fellow. The overall pattern of patient management, as outlined in Table III, has been determined by this multidisciplinary team.

Medical management: Recent research has shown that the administration of steroids[45-47] and diuretics, such as mannitol,[48-50] improves the recovery of animals subjected to controlled injuries of the spinal cord. However, there is no definite confirmation of the effectiveness of such therapy in humans with spinal cord injuries. Steroids — usually dexamethasone, 20 mg, followed by 4 mg every 4 hours — are given to all patients admitted to the unit with complete or incomplete cord injuries. If the cardiovascular status of the patient is not severely compromised by other injuries or by a lesion high in the cord, 500 ml of 20% mannitol is administered in an attempt to reduce cord edema. Hypotension is present in almost all patients with a spinal cord injury and is usually more severe in lesions high in the cervical cord owing to loss of sympathetic tone. Hypotension is treated vigorously in our unit, mainly with blood transfusions and intravenous infusion of fluids. In this regard it is relevant to recall the experiment of Hardy and colleagues,[51] who placed weights in increments on the exposed spinal cord of dogs until the somatosensory evoked potential was abolished. The blood pressure was then raised and within a short time the somatosensory evoked potential returned. We have not personally observed such a dramatic response in a patient, but one priority in the immediate management of patients in our unit is the restoration and maintenance of normal respiratory and cardiovascular function to achieve normal oxygenation and blood pressure. Although there is no direct proof at present, it is highly likely that these measures help to overcome post-traumatic ischemia and to improve recovery.

Other experimental therapeutic measures that have improved the recovery of animals subjected to experimental cord injury, such as the administration of hyperbaric oxygen,[52,53] catecholamine antagonists[3,54,55] and dimethyl sulfoxide,[56,57] are not being used in our unit.

Nursing management: In our unit nursing management has changed considerably during the past 4 years. At present almost all patients are nursed in regular hospital beds. Stryker frames, circle-electric beds and other turning frames are avoided because they are often distressing to the patient, do not always maintain spinal alignment and, in patients with lesions high in the spinal cord, may lead to further respiratory insufficiency when the patient is placed prone. We have found that these frames are unnecessary when skilled nursing care is available. Pressure sores are eliminated by turning the patients in log-rolling fashion every 2 hours. Antiembolic stockings or elastic bandages on the lower legs are used as prophylaxis against venous thrombosis. Consideration is being given to prophylactic anticoagulant therapy, which has been shown to reduce the frequency of deep venous thrombosis and pulmonary embolism in patients with acute spinal cord injury.[58] Transport of patients with a spinal cord injury from one place to another within the hospital is facilitated by the use of the Mobilizer® (Medical Products, Summit, New Jersey), a stretcher with a conveyer belt top that is mechanically driven.

Rehabilitation: Rehabilitation and physical medicine efforts directed by the physiatrist begin on the day of admission. Physiotherapists and occupational therapists play a much greater role in our unit than has traditionally been given them. For example, they can accurately assess and record day-to-day neurologic progress. The physiatrist is an important link between our unit and Lyndhurst Hospital, which provides rehabilitation services for patients with spinal cord injuries who are not well enough to return home. Very large sums of money are being saved by this arrangement, which allows much earlier discharge of patients from active treatment beds.

Urologic management: Intermittent catheterization is used for all patients with bladder paralysis admitted to our unit. The urologists direct the intermittent catheterization program and supervise the urology technicians, who perform the catheterization for the male patients, and the nursing staff, who do so for the female patients. Usually the program does not begin until the second or third day after trauma because of the difficulties of balancing intake and output in the first 2 to 3 days. Thereafter catheterization is done every 4 to 6 hours until spontaneous bladder emptying occurs or until the bladder can be satisfactorily emptied by the Credé or another maneuver. A special chart is used to record the progress from intermittent catheterization to voluntary voiding or induced emptying. The results in our unit are similar to those in other centres where intermittent catheterization has led to a marked reduction in the frequency of urinary tract infections and other urinary tract trauma associated with the use of indwelling catheters, and a high proportion of catheter-free pa-

tients.[59-62] If voluntary voiding has not been achieved by the time of discharge, patients returning home are taught to perform intermittent catheterization themselves; for patients transferred to Lyndhurst Hospital the program is continued there.

Immediate neurosurgical management of the injured spinal cord

Decisions regarding the acute management of the injured spinal cord are made on the basis of the principles outlined in Tables II to IV. Emergency treatment begins with rapid restoration of normal cardiovascular and respiratory function, immobilization of the spine and administration of steroids and mannitol.

In complete cervical or thoracic spinal cord injuries of immediate onset and more than 6 to 12 hours' duration, the experience during the past several decades has been that decompression laminectomy at that time or later is almost always unsuccessful in restoring spinal cord function.[27,63] There are probably several reasons for these poor results. First, most of the decompression procedures were performed too late in view of the autodestructive changes that occur in the cord within the first few hours of injury. Indeed, one recent series defined "early surgical therapy" as occurring "within 48 hours of injury".[63] Little wonder that surgical therapy did not help. The second reason is that in most instances cord decompression was attempted by laminectomy. It is now known that in many, if not most, cases of persisting traumatic cord compression the compression is occurring mainly anterior to the cord,[64,65] and that laminectomy provides only partial relief of cord compression caused by anteriorly situated masses.[66] Thus, the poor results of previous surgical treatment of complete spinal cord lesions have been due to late intervention from the wrong direction.

It is incorrect to conclude from these results that patients with complete spinal cord lesions cannot recover. Indeed, there are several well documented cases in which cord function has recovered, sometimes spontaneously and sometimes after surgical decompression, in cases of complete spinal cord lesions.[4,40,67-70] As we have noted, the historical data on surgical results are based mostly on cases treated after considerable delay. There is no accurate clinical information about the value of early decompression — say, within 2 hours of injury — in cases of immediate complete cord injuries. However, recent reports suggest that immediate decompression — that is, within 1 to 2 hours of injury — may improve cord recovery,[24,25] and there is support from controlled laboratory studies for early decompression in severe spinal cord injuries.[19]

Some patients with immediate complete spinal cord injuries may benefit from surgical procedures performed within a few days for indications other than attempts to improve cord recovery. For example, some patients with complete cervical injuries may benefit from anterior discotomy and fusion for decompression of the cervical roots and early stabilization of the spine to allow early mobilization.[65,68,71,72] The exact indications for this procedure remain uncertain because of the danger of major surgical procedures on patients with severe cervical cord injuries, as pointed out so well by Munro.[73] When this procedure is performed in our unit it is delayed until several days after the injury to allow normalization of respiratory and cardiovascular function. In cases of complete injury at the thoracolumbar junction or in the lumbar region with persisting compression, immediate or early decompression by an anterior or a posterior approach should also be considered, not to improve cord recovery but to promote anterior horn cell and nerve root recovery in the conus medullaris and cauda equina.

In most cases of incomplete lesions with progressive deterioration and demonstrated persisting compression early decompression should be performed.[74,75] However, even in the absence of progressive deterioration many patients with an incomplete lesion, a stable deficit and persisting compression derive significant improvement from early decompression, especially from an anterior or an anterolateral approach.[68,76-78] Similar patients not treated surgically may also recover, however.[4,69,79,80] Unfortunately, no controlled study of cases of incomplete spinal cord lesions has been reported.

Persisting cord compression can be relieved in several ways (Table IV). The anterior or anterolateral approach is highly recommended for relief of acute disc herniation or removal of posteriorly protruding segments of vertebral bodies, as in burst fractures. The posterior approach, with laminectomy, is preferred in instances in which posterior compression by bone fragments is suspected or demonstrated. In selected cervical cord injuries decompression can be quickly accomplished by skeletal traction,[81] sometimes assisted by general anesthesia or muscle relaxation. In reducing cervical dislocations or fracture–dislocations by skeletal traction the incremental addition of weight, with frequent clinical, ra-

Table IV—Indications for and methods of immediate decompression of the spinal cord or cauda equina

Indications for decompression
With immediate complete neurologic deficit
Cervical or thoracic lesion: no proven indications.
Thoracolumbar or lumbar lesion: to aid nerve root recovery by removal of persisting compression.
With incomplete neurologic deficit
At all levels in selected patients with persisting compression demonstrated on plain roentgenograms or myelogram.
— Usually necessary in those with a progressive deficit.
— Optional in those with a stable deficit.
— Usually unnecessary in those with an improving deficit.
Methods of decompression
Reduction of dislocation
Closed traction.
Closed traction with anesthesia or relaxant.
Operative reduction.
Posterior decompression: cervical, thoracic or lumbar laminectomy.
Anterior decompression: cervical.
Anterolateral decompression: thoracic or lumbar.

diologic and electrophysiological monitoring and pharmacologic therapy as required, is preferred. Frequent roentgenography to demonstrate the degree of reduction and distraction is critical. The rate at which increments are made and the total amount of weight employed must be individualized. These precautions are necessary to minimize the known risk of neurologic deterioration during traction.[82]

Two new methods of management have been used for complete spinal cord injuries — local cord cooling, or hypothermic irrigation, and myelotomy. Experimental studies of cord cooling, including ours, have confirmed that it is of some value in improving cord recovery,[46,83] but not to the extent found in the initial studies by Albin and colleagues.[22,23] To date we have treated 11 patients with hypothermic or normothermic irrigation of the cord.[84,85] All had complete cord injuries, and treatment was instituted within a few hours of injury. There was no functionally significant motor recovery, in contrast to the results reported by others.[86,87] However, three patients had useful sensory recovery, and therefore we plan to continue performing cord irrigation in certain patients with complete spinal cord lesions who have no other serious injuries and are admitted within a few hours of injury.

Myelotomy for internal decompression of the cord has been of interest since Allen[6] first advocated it, in 1911. Since then it has been tested in other laboratories, including ours, and the results have been favourable.[88,89] Recently there have been some preliminary reports of its use in patients, but the results have been difficult to evaluate.[7,90] Further study of this therapeutic modality is required.

Immediate management of the injured spinal column

In the acute phase it is safest to assume that all patients with spinal cord injuries have an unstable spinal column. Spinal stability can only be deemed certain after repeated clinical and radiologic studies. Immobilization of the spine in the acute phase is essential in patients with spinal cord and spinal column injury. Traditionally, acute immobilization in cases of unstable cervical injuries has been by skull traction with tongs,[81] and in thoracic and lumbar injuries it has been by postural control and positioning in bed with supports.[69,91] Spinal instability is the principal reason for prolonged bed rest in conventional programs of management for patients with spinal cord injuries. However, early mobilization of the patient is highly desirable for several reasons, especially to reduce the chance of complications of prolonged bed rest, such as pressure sores, pneumonia and thrombophlebitis. Early mobilization also improves the patient's morale by allowing earlier participation in rehabilitation efforts and programs to teach independence. Fortunately many patients with unstable spinal column injuries can now be mobilized rapidly with the spine safely and securely immobilized. Spinal immobilization can now be accomplished by numerous techniques, some of which, like the halo, have only recently been used for the management of patients with spinal cord injuries.[92]

Halo devices were originally introduced for patients with paralyzed neck and trunk muscles mainly due to poliomyelitis, and were used to maintain immobilization of the spine for several months following operative spinal fusion.[93] Later they were used for patients with traumatic lesions of the cervical spine without accompanying cord injury.[94,95] To date we have used halo devices in more than 50 patients with acute cervical spine injuries, approximately half of whom have had serious cervical cord injuries, and our experience has been highly favourable.[96] Halo devices have almost completely replaced skull tongs in our unit because they are much more versatile. We now use the halo in the acute stage for traction and immobilization for the same indications for which we previously used tongs. The halo has the added advantage that it can be converted to a halo–vest or halo–body jacket assembly for continuing immobilization of the spine while the patient is mobilized. The halo–vest does not provide rigid immobilization in all instances, however, and considerable care is required in its use.[97]

Spinal immobilization for unstable thoracic and lumbar injuries can now also be more readily accomplished with internal fixation devices such as Harrington rods. Although initially developed for the treatment of scoliosis,[98] Harrington rods provide excellent immobilization of the injured vertebral column and at the same time allow earlier mobilization of the patient.[99-101] It is not the purpose of this review to discuss fully the indications for operative spinal fixation or fusion. However, it should be noted that some highly experienced workers in the field of spinal cord injuries have strongly advocated nonoperative treatment to achieve spinal stability in most patients with these injuries.[69,73,102,103] They have cited a very low frequency of instability after nonoperative treatment of spinal fractures. Although operative procedures are sometimes undertaken primarily for spinal stability, in our unit most are undertaken to improve neurologic recovery, and in such instances the opportunity is usually taken to stabilize the spine concurrently.

Paraplegic units and acute spinal cord injury units

The most important reason for the increase in life expectancy and the reduction in the frequency of complications such as uremia and pressure sores in patients with spinal cord injuries has been the development of paraplegic units. One of the first units was at Stoke Mandeville, England; it began during World War II. There has been a highly significant Canadian contribution in this field, with the first paraplegic unit in Canada being opened at Lyndhurst Lodge (now Hospital) in Toronto in 1945. Now there are paraplegic units in most developed countries[91,102,104] providing expert care for patients with spinal cord injuries during the subacute and rehabilitation phases. They offer multidisciplinary teams of physicians, nurses and paramedical personnel, including physiotherapists, occupational therapists, social workers and vocational counsellors specially trained in caring for and rehabilitating such patients. With the assistance of such a team of interested personnel, many pa-

tients with spinal cord injuries can now look forward to an almost normal life expectancy and a return to gainful employment.[105-107]

During the past 10 years acute spinal cord injury units have been developed in many countries. Three reasons may be given to account for this development. First, the idea of an acute care unit was a natural extension of the idea of paraplegic units, for it was expected that the benefits to the patients in terms of rehabilitation and reduction of the frequency of complications would be even greater if applied as early as possible in the acute stage.[108] Second, the previously described laboratory research in spinal cord injuries indicated that early treatment by a variety of measures could improve spinal cord recovery, but that such treatment must be initiated as soon as possible after trauma. For example, the experiments on cord cooling showed that cooling was effective if applied 4 hours after trauma[22] but was ineffective at 8 hours.[23] The fact that patients with certain cord injuries could recover if treated early but would have complete paraplegia if treated late dispelled some of the nihilism concerning early therapy for severe spinal cord injuries. The third reason for the development of acute spinal cord injury units was the realization that acute care, which includes many new diagnostic and therapeutic modalities, is sufficiently complex that it must be planned and organized on a regional basis in order to reach most patients as soon as possible after trauma. We strongly agree with Bucy's recommendation that patients be transported to "an adequately staffed and equipped spinal cord injury center within two hours after the accident has occurred".[109] In addition, it has been suggested that such units may reduce the initial costs of care;[110,111] the costs in the first year are considerable.[112]

In the United Kingdom, Europe and Australia[102] many of the existing paraplegic units were able to accept patients in the acute phase because they were in general hospitals. In the United States and Canada this was often not the case, and therefore many acute care centres have been developed. In the United States the Department of Health, Education, and Welfare, the National Institutes of Health and the Veterans Administration have taken steps to develop new acute care centres or to reorganize existing paraplegic units for the management of the acute phase of spinal cord injuries.[109,113-115] In Canada the need for acute care centres was also recognized early and advocated by many. For example, in 1973 Gingras[116] stated that "acute centres should be developed in association with certain neurosurgical units", and the report of Botterell and associates[117] in 1975 to the Ontario Ministry of Health strongly advocated the development of acute spinal cord injury units.

At present there are two acute spinal cord injury units in Canada. The unit at Sunnybrook Medical Centre was started in 1974 as an extension of the paraplegic unit at the hospital and has been funded by a demonstration model grant from the Ontario Ministry of Health. The second unit began in Vancouver in 1975 at Shaughnessy Hospital.[118] Since 1974 at Sunnybrook there has been a progressive reduction in the interval between trauma and admission to the unit, and at present most patients are admitted within 5 hours of trauma. This is still far too long, and it is expected that the helicopter evacuation program in Ontario will reduce this figure. In Switzerland, where air rescue is used for almost all spinal cord injuries, the average time required for rescue has been reduced from 4½ hours to 50 minutes.[104]

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