

## **MANAGEMENT OF ACUTE SPINAL CORD INURIES IN AN INTENSIVE CARE UNIT OR OTHER MONITORED SETTING**

### **RECOMMENDATIONS**

Standards: There is insufficient evidence to support treatment standards.

Guidelines: There is insufficient evidence to support treatment guidelines.

Options:

- Management of patients with acute SCI, particularly patients with severe cervical level injuries, in an intensive care unit or similar monitored setting is recommended.
- Use of cardiac, hemodynamic, and respiratory monitoring devices to detect cardiovascular dysfunction and respiratory insufficiency in patients following acute cervical spinal cord injury is recommended.

### **RATIONALE**

The intensive care unit (ICU) setting has traditionally been reserved for critically ill patients who require aggressive medical care and exceptional medical attention. Most contemporary medical centers have multiple critical care units; each designed to provide discipline-specific observation and intensive care to patients in need. Select institutions have created Acute Spinal Cord Injury Centers and offer multidisciplinary care including ICU care to patients who have sustained acute spinal cord injuries. (2,11,12,16,21,22,28,30,31,32,34) Several reports describe improved patient management and lower morbidity and mortality following acute SCI with intensive care unit monitoring and aggressive medical management. (11,12,16,22,30,31,32,34) Despite this interest in and commitment to more comprehensive care for the patient with acute spinal cord injury (SCI) over the last 30 years by selected individuals and centers, many patients who sustain acute spinal cord injuries are not managed in an ICU

setting, nor are they routinely monitored for cardiac or respiratory dysfunction. There exist divergent management strategies for acute SCI patients within regions, communities, even within institutions depending on the training and experiences of the clinicians providing care. Recently completed randomized clinical trials investigating pharmacological agents in the treatment of acute SCI patients did not suggest a specific, common medical management paradigm to guide patient care provided by participating investigators, other than the timing and dosage of the pharmacological agents being tested. (4-7,9,10). These studies included large numbers of seriously injured acute SCI patients managed outside the ICU setting, most without continuous cardiac or respiratory monitoring.

## **QUESTIONS**

- 1). Do patients with acute spinal cord injuries benefit from care in the ICU setting?
- 2). Is monitoring of cardiac, hemodynamic and pulmonary performance of benefit to patients who have sustained acute SCI?

## **SEARCH CRITERIA**

A National Library of Medicine computerized literature search from 1966 to 2001 was undertaken using Medical Subject Headings in combination with “spinal cord injury”: medical management, non-operative management, surgical management, hypotension, respiratory insufficiency, pulmonary complications and intensive care unit. Approximately 3400 citations were acquired. Non English-language citations were deleted. Titles and abstracts of the remaining publications were reviewed and relevant articles were selected to develop this guideline. We focused on four specific topics concerning human patients with acute spinal cord

injuries: management in an intensive care unit (18 articles reviewed), cardiac instability (8 articles reviewed), hypotension (22 articles reviewed) and respiratory/pulmonary dysfunction (20 articles reviewed). Additional references were culled from the reference lists of the remaining papers. Finally, members of the author group were asked to contribute articles known to them on the subject matter that were not found by other search means. Articles describing non-human laboratory investigations germane to the topic and related general review articles referenced in the Scientific Foundation are included in the bibliography. Articles describing economics, epidemiology, anesthesia, monitoring techniques, penetrating cord injuries, nursing care, infectious or urologic complications, chronic complications or patients with remote spinal cord injuries were deleted. These efforts resulted in 17 manuscripts, all of which are reports of case series (Class III medical evidence), which form the foundation for this review. These articles are summarized in Evidentiary Table format.

## **SCIENTIFIC FOUNDATION**

The pathophysiology of acute spinal cord injury is complex and multifaceted. It involves a primary mechanical injury by way of compression, penetration, laceration, shear and/or distraction. The primary injury appears to initiate a host of secondary injury mechanisms including; (1) vascular compromise leading to reduced blood flow, loss of autoregulation, loss of microcirculation, vasospasm, thrombosis and hemorrhage, (2) electrolyte shifts, permeability changes, loss of cellular membrane integrity, edema, and loss of energy metabolism, and (3) biochemical changes including neurotransmitter accumulation, arachidonic acid release, free-radical and prostaglandin production and lipid peroxidation. (1,13,25,26,27,29) These mechanisms if unchecked result in axonal disruption and cellular death. A number of

contemporary reviews describe these theories and provide experimental evidence in their support (1,25,26,27).

Animal models of SCI suggest that ischemia of the spinal cord underlies much of the mechanism of posttraumatic SCI and is the important common denominator resulting in neurologic deficit after primary injury. (1,26,27) Ischemia appears to be related to both local and systemic vascular alterations after severe injury. Local vascular alterations are due to the direct spinal cord injury and focal, post-injury vasospasm, both of which lead to loss of autoregulation of spinal cord blood flow. (1,8,13,23,24,26,27,29) Systemic vascular alterations of blood flow to the spinal cord after acute SCI observed in both animal studies and in human SCI patients include reduced heart rate, cardiac rhythm irregularities, reduced mean arterial blood pressure, reduced peripheral vascular resistance and compromised cardiac output. (1,8,15,17,18,21,24,26,27,29,32) Any of these untoward hemodynamic occurrences can contribute to systemic hypotension following severe injury (15,16,17,21,26,27,30,32). Systemic hypotension in the setting of acute spinal cord injury, with coincident loss of spinal cord autoregulatory function, compounds local spinal cord ischemia by further reducing spinal cord blood flow and perfusion (1,26,27,29).

Respiratory insufficiency and pulmonary dysfunction is common after traumatic spinal cord injury, particularly when the injury occurs at cervical spinal cord levels. (11,12,14,18,19,20,22,30) Severely injured patients demonstrate marked reductions in expected vital capacity, inspiratory capacity and may experience relative hypoxemia, all of which contribute to global hypoxemia and can exacerbate spinal cord ischemia after acute injury. (14,18,19,20,22)

It appears that the earlier cardiac and/or ventilatory/pulmonary dysfunction is detected, the more likely effective, often life-saving treatment can be initiated. It is for these reasons that the issues of early ICU care and cardiac and pulmonary monitoring for human patients following acute SCI have been raised.

Several clinical series have been reported in which human patients with acute SCI have been managed in intensive care unit environments with attention to heart rate, cardiac function, pulmonary performance and mean arterial blood pressure. (2,3,11,12,14-22,30-34) Zach et al, in 1976 provided a preliminary report on their prospective medical management paradigm in the treatment of 117 consecutive acute SCI patients in the Swiss Paraplegic Centre of Basle, Switzerland. (34) All patients were treated in the intensive care unit with central venous pressure monitoring and were administered dexamethasone, 0.5 mg/kg for 4 days, with a tapering dose through 10 days, and volume expansion with Rheomacrodex 40, 500 ml/day for 7 days. Patients were stratified by injury level, degree of deficit (Frankel grade) and by time of admission after injury. The authors reported that 62% of cervical level SCI patients they managed in this way improved at last follow-up, including eight of 18 Frankel grade A patients, two by two grades and a third patient by three grades. No patient with a cervical injury worsened, 38% were unchanged from admission. Patients with thoracic T1-T10 level SCI fared less well; 38% improved, none worsened and 62% were without change, including 22 of 26 Frankel grade A patients. Two Frankel grade A patients experienced a complete recovery. Seventy percent of acute T11-L1 level SCI improved with this treatment paradigm, none worsened and 30% were unchanged from admission. Of patients who arrived within 12 hours of injury, 67% improved compared to their admission neurologic exam. Of patients admitted between 12 and 48 hours of

injury, only 59% improved. When admission occurred after 48 hours of injury, improvement was seen in only 50% of patients. The authors concluded that early transfer and “immediate medical specific treatment of the spinal injury” appeared to improve neurologic recovery.

Hachen, in 1977, reported the ten-year experience with acute traumatic tetraplegia from the National Spinal Injuries Centre in Geneva.(12) He described 188 acute SCI patients treated in a ten-year period in the intensive care unit setting following immediate transfer from the scene of the injury. The Centre reported a marked reduction in mortality rates following acute cervical SCI compared to annual statistics from 1966. Mortality for complete tetraplegia was reduced from 32.5% to 6.8% over the ten-year period. Mortality for patients with incomplete tetraplegia fell from 9.9% in 1966 to 1.4% in 1976. Most early deaths in the Centre’s experience were related to pulmonary complications. The likelihood of severe respiratory insufficiency was related to the severity of the cervical spinal cord injury. Seventy percent of patients with complete lesions experienced severe respiratory insufficiency in the Centre’s experience, compared to 27% of patients with incomplete lesions. The improvement in mortality rates described was directly related to early monitoring and treatment of respiratory insufficiency in the ICU setting. Hachen stressed that facilities for continuous monitoring of central venous pressure, arterial pressure, pulse, respiration rate and pattern, and oxygenation-perfusion parameters must be available for all patients with neurologic injuries following acute SCI, particularly those injuries above the C6 level.

In 1979 Gschaedler et al, described the comprehensive management of 51 patients with acute cervical spinal cord injuries in the intensive care unit setting in Colmar, France.(11) Forty percent of the patients they managed had multiple organ system injuries. They reported a low mortality rate (7.8%), and described several severely injured patients who made important

neurologic improvements, including one Frankel grade A patient to grade D, and two Frankel grade B patients to grade D. They cited early transport after injury and comprehensive intensive medical care with attention to and avoidance of hypotension and respiratory insufficiency as essential to the improved management outcome they experienced.

McMichan, Michel and Westbrook reported in 1980 their prospective assessment of pulmonary complications identified in 22 patients with cervical level acute SCI managed in the ICU setting.(20) They compared their results with 22 retrospective patients with similar injuries. Use of a new, aggressive pulmonary treatment paradigm resulted in zero deaths and fewer respiratory complications compared to those experienced by the retrospective group (nine deaths). They concluded that vigorous pulmonary therapy initiated early after acute SCI was associated with increased survival, a reduced incidence of pulmonary complications and a decreased need for ventilatory support.

Ledsome and Sharp measured pulmonary function in 16 cervical level complete acute SCI patients and compared initial values to those obtained in the same patients at one, three, five weeks and three and five months after injury.(14) In their 1981 report, they noted profound reductions in forced vital capacity (FVC) and expiratory flow rates immediately after injury. Patients with a FVC less than 25% of expected had a high incidence of respiratory failure requiring ventilator support. This was especially true of patients with injuries at C4 or above. They found a significant increase in FVC at five weeks post-injury and an approximate doubling of FVC at three months, irrespective of the level of cervical cord injury. Importantly, they identified hypoxemia ( $PO_2 < 80\text{mm Hg}$ ), in the majority of their patients (74% of those who did not require ventilator support), despite adequate alveolar ventilation ( $PCO_2$  normal despite low FVC). They attributed this to a ventilation perfusion imbalance that occurs immediately after

acute SCI. Systemic hypoxemia was identified by blood gas measurements and was effectively treated with the addition of supplemental oxygen in most patients.

Piepmeyer, Lehmann and Lane identified cardiovascular instability following acute cervical spinal cord injury in 45 patients they managed in the ICU setting in New Haven, CT.(21) Twenty-three patients had Frankel grade A injuries, eight were grade B, seven grade C and seven grade D. They discovered a high incidence of cardiovascular irregularities in these patients and identified a direct correlation between the severity of the cord injury and the incidence and severity of cardiovascular problems. Three patients returned to the ICU setting during the two-week observation period of the study due to cardiac dysfunction, despite a period of initial stability. Twenty-nine of the 45 patients had an average daily pulse rate of less than 55 bpm, 32 had spells during which their pulse rate was below 50 bpm for prolonged periods of time. Hypotension was common after acute SCI in their series, but most patients responded well to volume replacement. Nine patients required vasopressors to maintain a systolic pressure > 100 mm Hg., therapy which ranged from hours to five days duration. Cardiac arrest occurred in five patients (11%). All had Frankel grade A injuries. Three arrests occurred during endotracheal suctioning. The authors found that the first week post-injury was the timeframe during which patients were most vulnerable to cardiovascular instability. Patients with the most severe neurological injuries were most likely to experience cardiovascular instability after acute SCI. These events occurred despite the absence of complete autonomic disruption. Hypoxia and endotracheal suctioning were associated with cardiac arrest in the majority of instances. They concluded that careful monitoring of severely injured acute SCI patients in the intensive care unit setting reduces the risk of life-threatening emergencies.

In 1984 Tator and colleagues described their experience with 144 patients with acute SCI managed between 1974 and 1979 at a dedicated spinal cord injury unit at Sunnybrook Medical Centre in Toronto, Canada.(31) They compared their results to a cohort of 358 SCI patients managed between 1948 and 1973 prior to the development of the acute care SCI facility. All 144 patients managed from 1974 to 1979 were treated in an intensive care unit setting with strict attention to the treatment of hypotension and respiratory failure. Their medical paradigm was developed on the principle “that avoiding hypotension is one of the most important aspects of the immediate management of acute cord injury”. Hypotension was “treated vigorously” with crystalloid and transfusion of whole blood or plasma for volume expansion. Patients with respiratory dysfunction were treated with ventilatory support as indicated. They reported reduced mean time of injury to admission and treatment, 4.9 hours, compared to greater than 12 hours from the 1948-1973 experience. Neurological improvement was observed in 41 of 95 patients (43%) managed under the aggressive ICU medical paradigm. Fifty-two patients demonstrated no improvement (55%). Only two patients deteriorated (2%). The authors reported lower mortality, reduced morbidity, shorter length of stay and lower cost of treatment with their contemporary comprehensive management paradigm compared to the 1948-1973 experience. They cited improved respiratory management in their ICU as one of the principal factors responsible for reduced mortality and credited the avoidance of hypotension, sepsis and urologic complications for reduced morbidity after injury. These improved management results were realized despite the fact that 28% of the acute SCI patients they treated had additional injuries that increased their risk of morbidity and mortality.

Lehman et. al, in a follow-up study in 1987, reported on 71 acute SCI patients they managed in the intensive care unit at the Yale/New Haven Medical Center.(15) Patients were

admitted within 12 hours of injury and were stratified by level and severity of neurological injury (Frankel scale). No patient had an associated head injury, a history of diabetes mellitus, a pre-existing cardiac disorder or a history of cardiac medication use. All were monitored and aggressively treated to avoid hypotension. The authors found that all patients with severe cervical spinal cord injuries, Frankel grades A and B, had persistent bradycardia, defined as a heart rate < 60 beats per minute for duration of at least one day. Thirty-five per cent of Frankel grade C and D patients were identified to have persistent bradycardia. Only 13% of thoracic and lumbar SCI injuries had this finding. Similarly, marked bradycardia, <45 beats/min, was frequent in the severe cervical injury group (71%), and less common in the milder cervical injury (12%) and thoracolumbar injury (4%) patients. Many times sinus node slowing was profound enough to produce hemodynamic compromise and systemic hypotension. Bolus injections of atropine or placement of a temporary pacemaker were often performed. This therapy was required by 29% of the severe cervical injury patients and by none in the two other injury groups. Episodic hypotension unrelated to hypovolemia was identified in 68% of the severe cervical injury group and in none of the other two injury groups. Thirty-five percent of the severe cervical injury group patients required the use of intravenous pressors to maintain an acceptable blood pressure. Five of 31 patients (16%) in the severe injury group experienced a primary cardiac arrest, three of which were fatal. All five patients had Frankel grade A SCI. No patient in their study experienced a significant cardiac rate disturbance or spontaneous episode of hypotension beyond 14 days of injury. The authors concluded that potentially life-threatening cardiac arrhythmias and episodes of hypotension regularly accompanied acute severe injury to the cervical spinal cord within the first 14 days of injury. These events were not solely

attributable to disruption of the autonomic nervous system. Detection and treatment was best accomplished in the ICU setting.

Wolf et. al., in 1991 described their experience with bilateral facet dislocation injuries of the cervical spine at the University of Maryland in Baltimore.(33) Fifty-two patients with acute cervical trauma were described employing an aggressive treatment paradigm that included ICU care, aggressive resuscitation, invasive monitoring and hemodynamic manipulation to maintain mean blood pressure above 85 mm Hg. for five days. Thirty-four patients had complete neurological injuries, 13 had incomplete injuries and five patients were intact. The authors attempted closed reduction within four hours of patient arrival to their center and performed early open reduction on patients who could not be reduced by closed means, including closed reduction under anesthesia. All but three patients underwent surgery for stabilization and fusion. The authors report neurological improvement at discharge in 21% of complete SCI patients and in 62% of patients with incomplete cervical SCI on admission. No intact patient deteriorated. Only 52% one-year follow-up was provided. The authors concluded that their protocol of aggressive, early medical and surgical management of patients with acute SCI improved outcome following injury. Treatment in the ICU setting, hemodynamic monitoring with maintenance of mean arterial pressure and early decompression of the spinal cord by open or closed means appeared to reduce secondary complications following acute SCI in their study.

Levi and coworkers treated 50 acute cervical SCI patients in the ICU at the University of Maryland in Baltimore according to an aggressive management protocol which included invasive hemodynamic monitoring and volume and pressor support to maintain a hemodynamic profile with adequate cardiac output and mean blood pressure > 90 mm Hg.(16) Their 1993 report described 31 patients with Frankel grade A injuries on admission, eight patients with Frankel

grade B injuries and 11 patients in Frankel C and D grades. Eight patients had shock at the time of admission (systolic BP < 90 mm.), and 82% of patients had volume resistant hypotension requiring pressors within the first seven days of treatment. This was 5.5 times more common among patients with complete motor injuries. The authors reported that the overall mean PVRI for the 50 patients they studied was less than the normal range, and it was less than the normal value in 58% of patients. Half of their acute SCI patients had lower than normal SVRI values. No patient with a complete motor deficit (Frankel grades A and B) and marked PVRI/SVRI deficits experienced neurologic recovery at six weeks. Forty percent of patients managed by protocol including several with complete injuries improved, 42% remained unchanged and nine patients died (18%), at six weeks post-injury. There was minimal morbidity associated with invasive hemodynamic monitoring. The authors concluded that hemodynamic monitoring in the ICU allows early identification and prompt treatment of cardiac dysfunction and hemodynamic instability and can reduce the potential morbidity and mortality following acute SCI.

Vale et al, in 1997 reported their experience with a non-randomized, prospective pilot study in the assessment of aggressive medical resuscitation and blood pressure management in 77 consecutive acute SCI patients treated at the University of Alabama in Birmingham. (32) There was no control group. All patients were managed in the ICU with invasive monitoring, (Swan Ganz catheters and arterial lines) and blood pressure augmentation to maintain MAP > 85 mm Hg. for seven days post-injury. They reported ten patients with complete cervical SCI (ASIA grade A), 25 with incomplete cervical injuries (ASIA grades B, C and D), 21 patients with complete thoracic SCI and eight patients with incomplete thoracic level SCI (grades B, C and D). The average admission MAP for grade A cervical SCI patients was 66 mm Hg. Nine of ten required pressors following volume replacement to maintain an MAP of 85 mm Hg. Fifty-

two percent of incomplete cervical SCI patients required pressors to maintain MAP at 85 mm Hg. Only nine of 29 patients with thoracic level SCI required the use of pressors. The authors reported minimal morbidity with the use of invasive monitoring or with pharmacological therapy to augment MAP. At one-year follow-up (mean 17 months) neurological recovery was variable and typically incomplete. Three of ten ASIA grade A cervical SCI patients regained ambulatory capacity and two regained bladder function. Incomplete cervical SCI patients fared better. Twenty-three of these patients regained ambulatory function at 12 month follow-up, only four of who had initial exam scores consistent with ambulation. Twenty-two of 25 (88%) patients regained bladder control. Thirty-one of 35 cervical SCI patients and 27 of 29 thoracic level SCI patients were treated surgically. The authors statistically compared selection for and timing of surgery with admission neurological function and compared surgical treatment, early and late, with neurological outcome and found no statistical correlation. They concluded that the enhanced neurological outcome identified in their series after acute spinal cord injury was optimized by early and aggressive volume resuscitation and blood pressure augmentation and was in addition to and/or distinct from any potential benefit provided by surgery.

#### **SUMMARY:**

Patients with severe acute SCI, particularly cervical level injuries, or patients with multi-system traumatic injury, frequently experience hypotension, hypoxemia, pulmonary dysfunction and many exhibit cardiovascular instability, despite early acceptable cardiac and pulmonary function after initial resuscitation. These occurrences are not limited to acute SCI patients with complete autonomic disruption. Life-threatening cardiovascular instability and respiratory insufficiency may be transient and episodic and may occur in patients who appear to have stable

cardiac and respiratory function early in their post-injury course. Patients with the most severe neurological injuries after acute SCI appear to have the greatest risk of these life-threatening events. Monitoring allows the early detection of hemodynamic instability, cardiac rate disturbances, pulmonary dysfunction and hypoxemia. Identification and treatment of these events appears to reduce cardiac and respiratory related morbidity and mortality. Management in an intensive care unit or similar setting with cardiovascular and pulmonary monitoring have an impact on neurological outcome after acute SCI. Patients with acute spinal cord injuries appear to be best managed in the intensive care unit setting for the first seven to fourteen days after injury, the time frame during which they appear most susceptible to significant fluctuations in cardiac and pulmonary performance. This appears to be particularly true for severe cervical SCI patients, specifically acute ASIA grades A and B.

#### **KEY ISSUES FOR FUTURE INVESTIGATION:**

The length of stay in the intensive care unit setting necessary to provide optimal management of patients with acute SCI is unknown. The available evidence suggests that most untoward and potentially life-threatening cardiac and respiratory events occur within the first week or two following injury. Patients with less severe acute spinal cord injuries may require less time in a monitored setting than those patients with more severe injuries. These issues could be addressed in a prospective cohort study, or potentially a retrospective case control study.

## EVIDENTIARY TABLE

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
Lu K et al, 2000 Spine	Retrospective review of apnea in 36 ASCI patients	CLASS III	Delayed apnea most likely in ASCI patients with severe, diffuse ASCI. Apnea most likely within first 7-10 days
Botel et al, 1997, Spinal Cord	225 ASCI treated in ICU. Only 87 admitted within 24 hrs of injury	CLASS III	Significant numbers of multiply injured and head injured patients. No complete injury rec. Improved outcome when admitted to ICU early after injury
Vale et al, 1997, J Neurosurg	Prospective assessment of 77 ASCI treated in ICU, aggressive Hemodynamic support, MAP > 85	CLASS III	Improved outcome with aggressive medical care, distinct from potential benefit from surgery at 1 year follow up
Levi et al, 1993, Neurosurgery	50 patients treated in ICU, aggressive medical treatment, MAP > 90	CLASS III	Improved outcome with aggressive hemodynamic support at 6 weeks post-injury.
Tator et al, 1993, Surg. Neurology	201 ASCI patients, ICU care, hemodynamic support compared to 351 prior patients	CLASS III	Less severe cord injuries due to immobilization, resuscitation and early transfer to ICU setting.
Levi et al, 1991, Neurosurgery	103 ASCI, 50 incomplete (Group A), 53 complete (Group B), ICU care hemodynamic support, MAP > 85	CLASS III	Improved neurological outcome, no significant difference between early and late surgery in either group.
Wolf et al, 1991, J Neurosurg	52 patients with locked facets reduced within 4 hours, ICU care, MAP > 85. 49 operated upon, 23 day 1, 26 delayed (8.7d mean).	CLASS III	Closed reduction 61% Closed (a) 15% 52% f/u at 1 year, in general improved neurological outcome.
Lehmann et al, 1987, JACC	71 consecutive ASCI patients, ICU care, monitoring of cardiac/hemodynamic parameters	CLASS III	Bradycardia, 100%, Hypotension (<90 syst), 68% Life threatening bradyarrhythmias, 16% incidence related to severity of SCI
Reines HD et al, 1987 Neurosurgery	123 cases. ASCI patients in ICU, aggressive pulmonary treatment	CLASS III	Respiratory insufficiency major cause of morbidity and mortality after ASCI. Aggressive ICU care, pulmonary treatment reduces incidence.
Piepmeier et al, 1985, Central Nerv. Syst Trauma	45 ASCI patients, all managed in ICU setting with cardiac, hemodynamic monitoring	CLASS III	Cardiac dysrhythmia, hypotension and hypoxia common in first 2 weeks after ASCI. Incidence related to severity of injury.

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
Bose, et al, 1984, Neurosurgery	28 patients with ASCI, 22 managed in ICU setting Group I: medical treatment Group II: med/surg treatment	CLASS III	Improved neurologic outcome at discharge for Group II but better scores initially. Group I with intrinsic cord injury vs. Group II compression on myelo and/or instability.
Tator, et al, 1984, Canadian J Surg	144 ASCI patients ICU care, hemodynamic support, compared to prior series	CLASS III	Improved neurological outcome, less mortality with early transfer, avoidance of hypotension, and ICU care
Ledsome JR et al, 1981 Am Rev Respir Dis	Reassessment of pulmonary function in ASCI patients, comparison over time.	CLASS III	Reduced VC, flow rates and hypoxia after ASCI. Incidence related to severity of SCI. Marked improvement in pulmonary functions three months post-injury.
McMichan JC et al, 1980 JAMA	Prospective study of pulmonary complications in 22 ASCI patients, compared to 22 prior patients managed with aggressive ICU care.	CLASS III	No deaths in series vs. 9 of 22 deaths in prior group. ICU care and vigorous pulmonary therapy improves survival, reduces complications.
Gschaedler et al, 1979 Paraplegia	51 ASCI managed in ICU, aggressive medical treatment, avoid hypotension	CLASS III	Improved morbidity and mortality with early transfer, avoidance of hypotension,, respiratory insufficiency.
Hachen, 1977 J Trauma	188 ASCI managed in centre ICU, aggressive treatment of hypotension, respiratory insufficiency.	CLASS III	Reduced morbidity and mortality with early transfer, attentive ICU care and monitoring, and aggressive treatment of hypotension and respiratory failure.
Zach, et al, 1976 Paraplegia	117 ASCI at Swiss Center, ICU setting aggressive BP, volume therapy. Rheomacrodex x 5d Dexamethasone x 10d	CLASS III	Improved neurological outcome with aggressive medical treatment. Better outcome for early referrals.

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