

**Section on Disorders of the Spine and Peripheral Nerves**  
of the  
**American Association of Neurological Surgeons**  
and the  
**Congress of Neurological Surgeons**



American  
Association of  
Neurological  
Surgeons



**Guidelines for the Management of Acute  
Cervical Spine and Spinal Cord Injuries**

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### **AUTHOR GROUP**

#### **Co-Chairs**

**Mark N. Hadley, MD, FACS**

Professor, University of Alabama at Birmingham  
Division of Neurological Surgery

**Beverly C. Walters, MD, M.Sc. FRCSC, FACS**

Associate Professor, Brown Medical School  
Department of Neurosurgery

**Paul A. Grabb, MD**

Associate Professor, University of Alabama at Birmingham  
Division of Neurological Surgery  
Children's Hospital of Alabama

**Nelson M. Oyesiku, MD**

Associate Professor, Emory University  
Department of Neurosurgery

**Gregory J. Przybylski, MD**

Associate Professor, Northwestern University Medical School  
Department of Neurosurgery

**Daniel K. Resnick, MD**

Assistant Professor, University of Wisconsin – Madison  
Department of Neurosurgery

**Timothy C. Ryken, MD**

Assistant Professor, University of Iowa  
Department of Neurosurgery

#### **Secretarial Support**

Debbie H. Mielke

Administrative Associate

University of Alabama at Birmingham

Division of Neurological Surgery

## **INTRODUCTION**

Spinal cord injuries occur approximately 14,000 times per year in North America. The majority involves the cervical spinal region. Most patients, although not all, will have cervical spinal fracture-dislocation injuries as well. Patients who sustain cervical spinal cord injuries usually have lasting, often devastating neurological deficits and disability. Tens of thousands more patients per year will sustain traumatic cervical spinal injuries without spinal cord injury. The management of these patients and their injuries, cord and vertebral column, is typically not standardized or consistent within a single institution, from one center to another or among centers within geographic regions. Treatment strategies are usually based on institutional or personal provider experiences, physician training and the resources available at the treatment facility. Management can affect outcome in these patients; therefore, clinicians worldwide strive to provide the “best and most timely care”. Many times we may not be fully aware of what the “best care” may be or whether “timeliness” matters. In many circumstances “best care” likely encompasses a variety of treatment strategies, all with acceptable success rates and reasonable inherent risks.

The Section on Disorders of the Spine and Peripheral Nerves of the American Association of Neurological Surgeons and the Congress of Neurological Surgeons has long been interested in getting answers to some of the difficult management issues associated with acute spinal cord injuries. Identification of “best care” strategies is desired for all aspects of the care of acute cervical injury patients including pre-hospital care and transport, neurological and radiographic assessment, medical management of spinal cord injury, closed reduction of cervical fracture-dislocations and specific treatment options, both operative and non-operative, for each specific cervical injury type known to occur from the occiput through thoracic level one. The leadership of the Spine Section charged this committee to generate guideline documents on the management of patients with acute cervical spine and cervical spinal cord injuries. Our committee undertook this task in May 2000.

Twenty-two topics were identified and multiple questions were generated around which recommendations would be formed. We followed a meticulous process founded in evidence-based medicine. We searched for and relied on published scientific evidence rather than expert opinion or traditional practices. The author group first convened in September 2000. One year later we have completed our task.

Our hopes are that these guidelines will define the variety of assessment or treatment options available to a clinician in the management of an individual patient, provide direction within the broad scope of clinical practice, highlight what is known about specific issues and importantly, define what is not known, stimulating additional research.

## METHODOLOGY OF GUIDELINE DEVELOPMENT

### Introduction

The evolution of medical evidence has occurred rapidly over the last fifty years. From initial reports, anecdotal in nature, to large-scale randomized controlled trials, medical evidence is variable. From the evidence, and influenced by personal experience, clinicians choose paths of disease management. The medical specialties have pioneered the use of evidence produced from experimental trials to support clinical practice decisions. The surgical specialties have lagged behind the development of large-scale studies of surgical procedures and perioperative management. However, the high cost of medical care along with practice variation from region to region has given rise to an interest in developing strategies for linking practice to underlying evidence. In the course of this endeavor it has become clear that the variability of the evidence must somehow be reflected in any recommendations derived from it.

In the 1980s, criteria to be used in selecting evidence for developing treatment recommendations were developed. In a formal document, Clinical Practice Guidelines: Directions for a New Program, the Institute of Medicine addressed such guideline issues as “definition of terms, specification of key attributes of good guidelines, and certain aspects of planning for implementation and evaluation.” (1) The key intent of the document is to promote standardization and consistency in guideline development. In the course of the document, several key concepts in guideline development were espoused. They include:

1. A thorough review of the scientific literature should precede guideline development.
2. The available scientific literature should be searched using appropriate and comprehensive search terminology.

3. The evidence should be evaluated and weighted, reflecting the scientific validity of the methodology used to generate the evidence.
4. There should be a link between the available evidence and the recommendations with the strength of the evidence being reflected in the strength of the recommendations, reflecting scientific certainty (or lack thereof).
5. Empirical evidence should take precedence over expert judgment in the development of guidelines.
6. Expert judgment should be used to evaluate the quality of the literature and to formulate guidelines when the evidence is weak or non-existent.
7. Guideline development should be a multidisciplinary process, involving key groups affected by the recommendations.

The Guidelines for the Management of Acute Cervical Spine and Spinal Cord Injuries were developed using the evidence-based approach reflected in the above recommendations, rather than a consensus-based approach using the input of experts in a given field who make recommendations based upon a literature review and their personal experience. The author group involved in the development of these guidelines for treatment of patients with acute cervical spinal injury employed a strict process of literature review, ranking the published papers by strength of study design. Every effort was made to avoid influence by personal or professional bias by being objective in following a methodology defined in advance. The methodology chosen for this Guideline is evidence-based and follows the recommendations of the Institute of Medicine (IOM) Committee to Advise the Public Health Service on Clinical Practice Guidelines (1) as outlined in detail in the development process description below.

## **GUIDELINE DEVELOPMENT METHODOLOGY**

### **Literature search**

Extensive literature searches were undertaken for each clinical question addressed. The searches involved the available English-language literature for the past twenty-five years, using the computerized database of the National Library of Medicine. Human studies were looked for, and the search terms employed reflected the clinical question in as much detail as relevant, as described in the individual sections. Abstracts were reviewed and clearly relevant articles were selected for evaluation.

### **Evaluating strength of the therapy literature**

Each paper found by the above-mentioned techniques was evaluated as to study type (e.g., therapy, diagnosis, clinical assessment). For therapy, evidence can be generated by any number of study designs. The strongest study protocol, when well designed and executed, is by far the randomized controlled trial (RCT). The prospectivity, presence of contemporaneous comparison groups, and adherence to strict protocols observed in the RCT diminish sources of systematic error (called *bias*). The randomization process reduces the influence of unknown aspects of the patient population that might affect the outcome (*random error*).

The next strongest study designs are the non-randomized cohort study and the case-control study, also comparing groups who received specific treatments, but in a non-randomized fashion. In the former study design, an established protocol for patient treatment is followed and groups are compared in a prospective manner, providing their allocation to treatment is not determined by characteristics that would not allow them to receive either treatment being studied. These groups would have a disorder of interest, e.g., spinal cord injury, and receive different interventions, and then the differences in outcome would be studied. In the case-control

study, the study is designed with the patients divided by outcome (e.g., functional ability) and their treatment (e.g., surgery vs. no surgery) would be evaluated for a relationship. These studies are more open to systematic and random error and thus are less compelling than an RCT. However, the RCT with significant design flaws that threaten its validity loses its strength and may be classified as a weaker study.

Least strong evidence is generated by published series of patients all with the same or similar disorder followed for outcome, but not compared as to treatment. In this same category is the case report, expert opinion, and the RCT so significantly flawed that the conclusions are uncertain. All of these statements regarding study strength refer to studies on treatment. But patient management includes not only treatment, but also diagnosis and clinical assessment. These aspects of patient care require clinical studies that are different in design, generating evidence regarding choices of diagnostic tests and clinical measurement.

### **Evaluating strength of the diagnostic test literature**

To be useful, diagnostic tests have to be reliable and valid. Reliability refers to the test's stability in repeated use, in the same circumstance. Validity describes the extent to which the test reflects the "true" state of affairs, as measured by some "gold standard" reference test. Accuracy reflects the test's ability to determine who does and does not have the suspected or potential disorder. Overall, the test must be accurate in picking out the true positives and true negatives, with the lowest possible false positive and false negative rate. These attributes are represented by sensitivity, specificity, positive predictive value, and negative predictive value. These may be calculated using a Bayesian 2 X 2 table as follows:

		GOLD STANDARD		
		Patient has injury	Patient has no injury	
TEST RESULT	Positive: Appears to have injury	TRUE POSITIVE (a)	FALSE POSITIVE (b)	(a) + (b)
	Negative: Appears to have no injury	FALSE NEGATIVE (c)	TRUE NEGATIVE (d)	(c) + (d)
		(a) + (c)	(b) + (d)	(a) + (b) + (c) + (d)

Using the above table, the components of accuracy can be expressed and calculated as follows:

Sensitivity	$a/a+c$	If a patient has a positive X-ray, how likely is he to have a C-spine injury?
Specificity	$d/b+d$	If a patient has a negative X-ray, how likely is he to not have a C-spine injury?
Positive predictive value	$a/a+b$	If a patient has a C-spine injury, how likely is he to have a positive test?
Negative predictive value	$d/c+d$	If a patient does not have a C-spine injury, how likely is he to have a negative test?
Accuracy	$a+d/a+b+c+d$	

It is the characteristic of diagnostic tests that these attributes do not always rise together, but generally speaking, these numbers should be greater than 70% to consider the test useful. The issue of reliability of the test will be discussed below when describing patient assessment.

## Evaluating strength of the patient assessment literature

There are two points when patient assessment is key in the patient management paradigm. There is initial assessment, e.g., patient's condition in the trauma room, and the ultimate, or outcome, assessment. All patient assessment tools, whether they are radiographic, laboratory, or clinical, require that the measurement be reliable. In the case of studies carried out by mechanical or electronic equipment, these devices must be calibrated regularly to assure reliability. In the instance of assessments carried out by observers, reliability is assured by verifying agreement between various observers carrying out the same assessment, and also by the same observer at different times. Because a certain amount of agreement between observers or observations could be expected to occur by chance alone, a statistic has been developed to measure the agreement between observations or observers beyond chance. This is known as an index of concordance and is called the *kappa* statistic, or simply *kappa*. (3) Once again, the Bayesian 2 X 2 table can be utilized to understand and to calculate kappa.

		OBSERVER #1		
		YES	NO	
OBSERVER #2	YES	AGREE (a)	DISAGREE (b)	(a) + (b) = $f_1$
	NO	DISAGREE (c)	AGREE (d)	(c) + (d) = $f_2$
		(a) + (c) = $n_1$	(b) + (d) = $n_2$	(a) + (b) + (c) + (d) = N

Using these numbers, the formula for calculating kappa is:

$$k = \frac{N(a+d) - (n_1f_1 + n_2f_2)}{N^2 - (n_1f_1 + n_2f_2)} \text{ or } k = \frac{2(ad - bc)}{n_1f_2 + n_2f_1}$$

Translating the numbers generated using these formulas to meaningful interpretations of the strength of the agreement between observers or observations is accomplished using these guidelines (5):

Value of k	Strength of Agreement
<0	Poor
0 - .20	Slight
.21 - .40	Fair
.41 - .60	Moderate
.61 - .80	Substantial
.81 - 1.00	Almost perfect

Each paper on clinical assessment was examined for its adherence to the rules of reliability and the exact kappa was noted and linked to the strength of recommendations, as described below.

### Linking evidence to guidelines

The concept of linking evidence to recommendations has been further formalized by the American Medical Association (AMA) and many specialty societies, including the Congress of Neurological Surgeons (CNS), American Association of Neurological Surgeons (AANS) and the American Academy of Neurology (AAN). (1, 2, 6, 7) This formalization involves the designation of specific relationships between the strength of evidence and the strength of recommendations, avoiding ambiguity. In the paradigm for therapeutic maneuvers, evidence is classified into that derived from the strongest clinical studies (well-designed, randomized, controlled trials), generating **Class I** evidence. **Class I** evidence is used to support

recommendations of the strongest type, called practice **Standards**, indicating a *high degree of clinical certainty*. Non-randomized cohort studies, randomized controlled trials with design flaws, and case-control studies (comparative studies with less strength) are designated **Class II** evidence. These are used to support recommendations called **Guidelines**, reflecting a *moderate degree of clinical certainty*. Other sources of information, including observational studies such as case series and expert opinion, as well as randomized controlled trials with flaws so serious that the conclusions of the study are truly in doubt (**Class III** evidence), support practice **Options** reflecting *unclear clinical certainty*. These categories of evidence are summarized in the table below.

Classification of Evidence on Therapeutic Effectiveness

Class I	Evidence from one or more well-designed, randomized controlled clinical trials, including overviews of such trials.
Class II	Evidence from one or more well-designed comparative clinical studies, such as nonrandomized cohort studies, case-control studies, and other comparable studies, including less well designed randomized controlled trials.
Class III	Evidence from case series, comparative studies with historical controls, case reports, and expert opinion, as well as significantly flawed randomized controlled trials.

The general term for all of the recommendations is **Practice Parameters**. Because so few practice Standards exist, the term more commonly used to describe the whole body of recommendations is practice guidelines. Thus, we have named this document ***Guidelines for the Management of Acute Cervical Spine and Spinal Cord Injuries***.

One of the practical difficulties encountered in implementing this methodology is that a poorly designed randomized controlled trial might take precedence over a well-designed case-control or non-randomized cohort study. The authors of this document have attempted to avoid

this pitfall by carefully evaluating the quality of the study as well as its type. All of these criteria apply to practice guidelines (parameters) for *treatment*. To assess literature pertaining to *prognosis, diagnosis, and clinical assessment*, completely different criteria must be used.

Criteria for prognosis have been developed and were widely used in the publications of Prognostic Indicators in Severe Traumatic Brain Injury. (Walters, Chesnut). No issues of prognosis were addressed in the current document.

For diagnosis, papers are evaluated differently yet again. The issues addressed by papers on diagnosis are related to the ability of the diagnostic test to successfully distinguish between patients who have and do not have a disease or pertinent finding. This speaks to the validity of the test and is illustrated below.

**Classification of Evidence on Diagnosis**

Class I	Evidence provided by one or more well-designed clinical studies of a diverse population using a “gold standard” reference test in a blinded evaluation appropriate for the diagnostic applications and enabling the assessment of sensitivity, specificity, positive and negative predictive values, and, where applicable, likelihood ratios.
Class II	Evidence provided by one or more well-designed clinical studies of a restricted population using a “gold standard” reference test in a blinded evaluation appropriate for the diagnostic applications and enabling the assessment of sensitivity, specificity, positive and negative predictive values, and, where applicable, likelihood ratios.
Class III	Evidence provided by expert opinion, studies that do not meet the criteria for the delineation of sensitivity, specificity, positive and negative predictive values, and, where applicable, likelihood ratios.

For clinical assessment, there needs to be both reliability and validity in the measure. This means that the assessment is done reliably between observers, and by the same observer at a different time. For validity, the clinical assessment, like diagnostic tests described above, need

to adequately represent the true condition of the patient. This latter aspect is difficult to measure, so most clinical assessments are graded according to their reliability.

**Classification of Evidence on Clinical Assessment**

Class I	Evidence provided by one or more well-designed clinical studies in which interobserver and intraobserver reliability is represented by a Kappa statistic of .80 or greater.
Class II	Evidence provided by one or more well-designed clinical studies in which interobserver and intraobserver reliability is represented by a Kappa statistic of .60 or greater.
Class III	Evidence provided by one or more well-designed clinical studies in which interobserver and intraobserver reliability is represented by a Kappa statistic of less than .60.

For each question addressed in these guidelines, articles were examine and the study type was assessed and assigned a classification according to the scheme outlined above. These designations are listed in the evidentiary tables of each of the chapters.

**GUIDELINES DEVELOPMENT PROCESS**

A group of individuals with interest and expertise in the treatment of cervical spinal injured patients and/or guideline practice parameter development was assembled under the auspices of and with the support of the Joint Section on Disorders of the Spine and Peripheral Nerves of the AANS/CNS. The group reflected expertise in spinal neurosurgery, neurotrauma, and clinical epidemiology. The issues chosen for inclusion in the document were those considered pertinent to the acute management of patients with cervical spine and/or spinal cord injury (e.g., transport, medical management, treatment of specific fracture/dislocation patterns, vascular injury, and prophylaxis for thromboembolic events).

A Medline search from January 1966 to January 2001 was carried out using the search terms described in each individual section. The search was limited to human subjects and included English language literature only for all but one of the sections. Additional papers were found through the reference lists in the articles found, as well as from other sources known to the author group. Papers were rejected on the basis of irrelevance to the clinical question at hand. Case reports were included if there was insufficient material from case series. Individuals then brought additional articles of relevance from other sources. All articles were evaluated according to the medical evidence-based scheme outlined above. For therapy, diagnosis, and clinical assessment, recommendations were derived. The drafts were revised and members of the author group different from the primary authors rewrote the drafts and the final product was agreed upon by consensus. On occasion, the assessed quality of the study design may have been so contentious and the conclusions so uncertain, that the author group designated a lower classification than might have been expected without such detailed review.

In every way, the author group attempted to adhere to the Institute of Medicine criteria for searching, assembling, evaluating, and weighting the available medical evidence and linking it to the strength of the recommendations presented in this document.

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## PRE-HOSPITAL CERVICAL SPINAL IMMOBILIZATION FOLLOWING TRAUMA

### RECOMMENDATIONS

Standards: There is insufficient evidence to support treatment standards.

Guidelines: There is insufficient evidence to support treatment guidelines.

Options:

- It is suggested that all trauma patients with a cervical spinal column injury or with a mechanism of injury having the potential to cause cervical spinal injury should be immobilized at the scene and during transport using one of several available methods.
- A combination of a rigid cervical collar and supportive blocks on a backboard with straps is very effective in limiting motion of the cervical spine and is recommended. The longstanding practice of attempted cervical spinal immobilization using sandbags and tape alone is not recommended.

### RATIONALE

The early management of the patient with a potential cervical spinal cord injury begins at the scene of the accident. The chief concern during the initial management of patients with potential cervical spinal injuries is that neurologic function may be impaired due to pathologic motion of the injured vertebrae. It is estimated that 3% to 25% of spinal cord injuries occur after the initial traumatic insult, either during transit or early in the course of management.(11,12,40,46,78,92) Multiple cases of poor outcome from mishandling of cervical spinal injuries have been reported.(9,49,78,92) As many as 20% of spinal column injuries involve multiple non-continuous vertebral levels, therefore the entire spinal column is potentially at risk. (36,37,65,70) Consequently, complete spinal immobilization has been used in pre-hospital spinal

care to limit motion until injury has been ruled out. (20,25,38,65,70,73,95, 98,101) Over the last 30 years there has been a dramatic improvement in the neurologic status of spinal cord injured patients arriving in emergency departments. During the 1970's the majority (55%) of patients referred to Regional Spinal Cord Injury Centers arrived with complete neurological lesions. In the 1980's, however, the majority (61%) of spinal cord injured patients arrived with incomplete lesions.(44) This improvement in the neurologic status of patients has been attributed to the development of Emergency Medical Services (EMS) initiated in 1971, and the pre-hospital care (including spinal immobilization) rendered by EMS personnel. (20,43,44,100) Spinal immobilization is now an integral part of pre-hospital management and is advocated for all patients with potential spinal injury following trauma by EMS programs nationwide and by the American College of Surgeons.(1,2,13,20,30,67,88,95)

Recently, the use of spinal immobilization for all trauma patients, particularly those with a low likelihood of traumatic cervical spinal injury has been questioned. It is unlikely that all patients rescued from the scene of an accident or site of traumatic injury require spinal immobilization.(31,48,66,74) Some authors have developed and advocate a triage system based on clinical criteria to select patients for pre-hospital spinal immobilization.(10,30,71)

Several devices are available for pre-hospital immobilization of the potential spine injured patient. However, the optimal device has not yet been identified by careful comparative analysis. (14,18,25,51,58,61,89,97) The recommendations of the American College of Surgeons consist of a hard backboard, a rigid cervical collar, lateral support devices, and tape or straps to secure the patient, the collar and the lateral support devices to the backboard. (94,95) A more uniform, universally accepted method for pre-hospital spinal immobilization for patients with potential spinal injury following trauma may reduce the cost and improve the efficiency of pre-hospital

spinal injury management.(10,30,71) While spinal immobilization is typically effective in limiting motion, it has been associated with morbidity in a small percentage of cases. (6,15,16,24,53,84,93,98)

These issues are the subject of this review on the use and effectiveness of pre-hospital spinal immobilization.

## **SEARCH CRITERIA**

A National Library of Medicine computerized literature search from 1966 to 2001 using the medical subject heading "spinal immobilization" was performed. The search was limited to human subjects and the English language. A total of 39 articles were accessed. A second search combining the exploded terms "spinal injuries" and "immobilization" yielded 122 articles. A third search combining the exploded terms "spinal injuries" and "transportation of patients" yielded 47 articles. A fourth search combining the exploded terms "spinal injuries" and "emergency medical services" produced 119 articles. Additional references were culled from the reference lists of the remaining papers. Finally, the author group was asked to contribute articles known to them on the subject matter that were not found by other search methods. Duplicate references were discarded. The abstracts were reviewed and articles unrelated to the specific topic were eliminated. This process yielded a total of 100 articles for this review which are listed in the bibliography. Articles used to formulate this guideline are summarized in Evidentiary Table format.

## SCIENTIFIC FOUNDATION

Pathologic motion of the injured cervical spine may create or exacerbate cervical spinal cord or cervical nerve root injury.(36,37,38,65,70,91) This potential has lead to the use of spinal immobilization for trauma patients who have sustained a cervical vertebral column injury or experienced a mechanism of injury that could result in cervical spinal column injury. (2,25,33,31,38,65,70,71,73,95,101)

Kossuth is credited with pioneering the currently accepted methods of protection and immobilization of the cervical spine during extrication of acute injury victims.(54,55) Farrington championed the concept of pre-hospital immobilization. (34,35) Dick and Land noted in their review of spinal immobilization devices that techniques of pre-hospital spinal immobilization appeared in standard EMS texts and in the American Academy of Orthopedic Surgeons Committee on Injuries Emergency text as early as 1971.(20,28) Initially, the preferred method to immobilize the cervical spine was the use of a combination of a soft collar and a rolled up blanket.(18) This was followed by the introduction of a more rigid extrication collar by Hare in 1974. Hare's contribution launched an era of innovation for devices for spinal immobilization.(25)

Currently, spinal immobilization is one the most frequently performed procedures in the pre-hospital care of acute trauma patients in North America. (2,3,20,25,36,38,65,70,73,96,101) While clinical and biomechanical evidence demonstrate that spinal immobilization limits pathologic motion of the injured spinal column, there is no Class I or Class II medical evidence to support spinal column immobilization in all patients following trauma. While immobilization of an unstable cervical spinal injury makes good sense, and Class III evidence reports exist of neurological worsening with failure of adequate spinal immobilization, there have been no case-controlled studies or randomized trials that address the impact of spinal immobilization on clinical

outcomes following cervical spinal column injury. (2,25,30,38,40,46,48,65,66,70,92) The issue is important; tens of thousands of trauma victims are treated with spinal immobilization each year, yet few will have spinal column injuries or instability.(37,71,79)

Other considerations in the use of pre-hospital spinal immobilization include the cost of equipment, the time and training of EMS personnel to apply the devices, and the unnecessary potential morbidity for patients who do not need spinal immobilization following trauma.(6,15,16,24,25,53,56,80,84,93,98) As with many interventions in the practice of medicine, spinal immobilization has been instituted in the pre-hospital management of trauma victims with potential spinal injuries based on the principles of neural injury prevention and years of clinical experience, but without supportive scientific evidence from rigorous clinical trials. For a variety of both practical and ethical reasons it is likely impossible to obtain this information in clinical trials in contemporary times.

In 1989 Garfin stated that "no patient should be extricated from a crashed vehicle or transported from an accident scene without spinal stabilization". In his review, he credited stabilization of the cervical spine as a key factor in the decline in the percentage of complete spinal cord injury lesions from 55% in the 1970's to 39% in the 1980's and in the significant reduction in the mortality of multiple injury patients with cervical spinal injuries.(38) Unfortunately, there is no Class I or Class II medical evidence to support his claims.

Few articles have directly evaluated the effect of pre-hospital spinal immobilization on neurological outcome after injury. Several Class III evidence reports cite the lack of immobilization as a cause of neurological deterioration among acutely injured trauma patients transported to medical facilities for definitive care. (9,38,49,59,78) The most pertinent study is a

retrospective case series of Toscano et al, who in 1988 reported that 32 of 123 trauma patients (26%) they managed sustained major neurological deterioration in the period of time between injury and admission. The authors attributed neurological deterioration to patient mishandling and cited the lack of spinal immobilization after traumatic injury as the primary cause. Their report supports the need for pre-hospital spinal immobilization of trauma patients with potential spinal column injuries.(92)

In contrast, a collaborative, 5-year retrospective chart review reported by the University of New Mexico and the University of Malaya challenges this position. Hauswald et al analyzed only patients with acute blunt spinal or spinal cord injuries. At the University of Malaya, none of the 120 patients they managed were immobilized with spinal orthoses during transport. All 334 patients managed at the University of New Mexico were initially treated with spinal immobilization. Both hospitals were reportedly comparable with respect to physician training and clinical resources. Two independent physicians blinded to the participating hospital characterized the neurological injuries into two groups: disabling and non-disabling. Data were analyzed using logistic regression techniques, with hospital, patient age, gender, anatomic level of injury, and injury mechanism as variables. Neurological deterioration after injury was less frequent in Malayan patients with spinal injuries who were not treated with formal spinal immobilization during transport (OR 2.03; 95% CI 1.03-3.99;  $p = 0.04$ ), compared to patients in New Mexico who were managed with spinal column immobilization techniques. Even when the analysis was limited to cervical spine injuries, no significant protective effect from spinal immobilization was identified. The authors theorized that since the initial injury is of tremendous force, further movement of the spine by the patient or rescuers is insufficient to cause further injury. However, they noted that because of the small sample size, the benefit of spinal immobilization might not

have been statistically measurable in their study.(48) This report has been challenged and several flaws identified: Patients who died at the scene or during transport were excluded from analysis. Injuries were not matched by severity of neurological injury or by type of spinal column injury. The mechanisms of injury differed dramatically in the two populations. Malayan patients were immobilized or held immobile during transport but spinal orthoses as immobilization devices were not employed. For these reasons and others, the conclusions drawn by the authors are considered spurious at best. (25,74)

Evidence in the literature evaluating the effectiveness of pre-hospital spinal immobilization is sparse. The Hauswald paper was published in 1998 after a significant period of time during which universal spinal immobilization following trauma had been applied in the United States and North America. Ethical and practical issues preclude the execution of a contemporary clinical trial designed to study the effectiveness of pre-hospital spinal immobilization compared to no immobilization, primarily because spinal immobilization for trauma patients is perceived as essential with minimal risk, and is already widely employed. Intuitively, the use of pre-hospital spinal immobilization is a rational means of limiting spinal motion in spinal injured patients in an effort to reduce the likelihood of neurological deterioration due to pathological motion at the site(s) of injury.

The consensus opinion from all articles reviewed (Class III evidence), from an anatomic and biomechanical perspective and from time-tested clinical experience with traumatic spinal injuries is that all patients with cervical spinal column injuries, or those with the potential for a cervical spinal injury following trauma, should be treated with spinal column immobilization until injury has been excluded or definitive management has been initiated. While there is insufficient medical evidence to support a treatment standard or a treatment guideline, practitioners are

strongly encouraged to provide spinal immobilization to spine injured patients (or those with a likelihood of spinal injury) until definitive assessment can be accomplished.

Orledge and Pepe in their commentary on the Hauswald findings point out some limitations of their paper, but also suggest that it raises the issue of a more selective evidence-based protocol for spinal immobilization.(74) Should all trauma patients be managed with spinal immobilization until spinal injury has been excluded, or should immobilization be selectively employed for patients with potential spinal injury based on well-defined clinical criteria? Which clinical criteria should be used? Following the Hauswald report, prospective studies in support of the use of clinical findings as indicators for the need for pre-hospital spinal immobilization after trauma have been reported.(31-33) Several EMS systems now employ clinical protocols to help guide which patients should be managed with spinal immobilization after trauma.(4,41)

Domeier et al, in a multicenter prospective study of 6,500 trauma patients found that the application of clinical criteria (altered mental status, focal neurologic deficit, evidence of intoxication, spinal pain or tenderness, or suspected extremity fracture), was predictive of the majority of patients who sustained cervical spinal injuries requiring immobilization. The predictive value of their criteria held for patients with high or low risk mechanisms of injury.(30,31,33) They suggested that clinical criteria rather than the mechanism of injury be evaluated as the standard by which spinal immobilization be employed.

Brown et al examined whether EMS providers could accurately apply clinical criteria to clear the cervical spines of trauma patients prior to transport to a definitive care facility. (10) The criteria included the presence of pain or tenderness of the cervical spine; the presence of a neurological deficit; an altered level of consciousness; evidence of drug use or intoxication

(particularly alcohol, analgesics, sedatives, or stimulants); and/or the presence of other significant trauma that might act as a distracting injury. Immobilization of the cervical spine was initiated if any one of six criteria was present. The clinical assessment of trauma patients by EMS providers was compared to the clinical assessment provided by emergency physicians. The providers (EMTs and ER MDs) were blinded to each other's assessments. Agreement between EMS and MD providers was analyzed by the kappa statistic. Five hundred seventy-three patients were included in the study. The assessments matched in 79% of the cases (n = 451). There were 78 patients (13.6%) for whom the EMS clinical assessment indicated spinal immobilization, but the MD assessment did not. There were 44 patients (7.7%) for whom the MD clinical assessment indicated spinal immobilization, but the EMS assessment did not. The kappa for the individual components ranged from 0.35 to 0.81. The kappa value for the decision to immobilize was 0.48. The EMS clinical assessments were generally more in favor of immobilization than the physician clinical assessments. The authors concluded that EMS and MD clinical assessments to rule out cervical spinal injury after trauma have moderate to substantial agreement. The authors recommended, however, that systems that allow EMS personnel to decide whether to immobilize patients after trauma should provide attentive follow-up of those patients to ensure appropriate care and to provide immediate feedback to the EMS providers.<sup>(10)</sup> Meldon et al, in an earlier study, found significant disagreement between the clinical assessments and subsequent spinal immobilization of patients between EMS technicians and MDs. They recommended further research and education before widespread implementation of this practice.<sup>(69)</sup>

Clinical criteria to select appropriate patients for spinal immobilization are being studied in Michigan (4) and have been implemented in Maine (41) and San Mateo County, California.<sup>(84)</sup> Recommendations regarding the adoption of EMS protocols for pre-hospital spinal

immobilization awaits definitive studies of safety and efficacy.(21) EMS personnel who make these assessments require intensive education and careful, quality assurance scrutiny to ensure that trauma patients with potential spinal injuries are appropriately triaged and managed.

While awaiting further studies, the available Class III studies supports the use of spinal immobilization for all patients with the potential of a cervical spinal injury following trauma.

### Methods of Pre-hospital Spinal Immobilization

Pre-hospital spinal immobilization is effective in limiting spinal motion during patient transport.(3,25,38,65,70,101) Various devices and techniques exist to provide immobilization of the cervical spine. Attempts to define the best method of spinal immobilization for pre-hospital transport have been hampered by physical and ethical constraints. (14,25,51,58,61,89,97)

The methods of measuring the efficacy of spinal immobilization devices vary among investigators. Comparative studies of the various devices have been performed on normal human volunteers but none have been tested in a large number of patients with spinal injury. It is difficult to extrapolate normative data to injured patients with spinal instability.(14,17,22,25,27,47,50,51,56,62,63,75,89,96,97)

Several methods have been used to measure movement of the cervical spine. They range from clinical assessment, plumb lines, photography, radiography, cinematography, CT and MRI. Roozmon et al, summarized the problems inherent in each method and concluded that there was no satisfactory noninvasive means of studying neck motion, particularly if one is to quantify movement between individual vertebral segments.(82)

The position in which the injured spine should be placed and held immobile - the "neutral position", is poorly defined.(23,26,72,84,87) Schriger defined the neutral position as, "the normal

anatomic position of the head and torso that one assumes when standing and looking ahead." This position correlates to 12 degrees of cervical spine extension on a lateral radiograph.(84) Schriger comments that the extant radiographic definition of neutral position was based on radiographic study of patients who were visually observed to be in neutral position. (84) Schriger et al used this position in their evaluation of occipital padding on spinal immobilization backboards.(87) De Lorenzo et al, in their MRI study of 19 adults, found that a slight degree of flexion equivalent to 2 cm of occiput elevation produces a favorable increase in spinal canal/spinal cord ratio at levels C5 and C6, a region of frequent unstable cervical spine injuries.(26) Backboards have been used for years for extrication and immobilization of spine injured patients. Schriger questioned the ability of a flat board to allow neutral positioning of the cervical spine. They compared spinal immobilization employing the flat backboard with and without occipital padding in 100 adults. Clinical observation and assessment were used to determine the neutral position of the cervical spine. The authors found that the use of occipital padding in conjunction with a rigid backboard places the cervical spine in optimal neutral position compared to positioning on a flat backboard alone.(87,88) McSwain determined that more than 80% of adults require 1.3 cm to 5.1 cm of padding to achieve neutral positioning of the head and neck with respect to the torso and noted that body habitus and muscular development alter the cervical-thoracic angle, thus affecting positioning.(68) This makes it impossible to dictate specific recommendations for padding.

In general, spinal immobilization consists of a cervical collar; supports on either side of the head; and the long and short backboards with associated straps to attach and immobilize the entire body to the board.(25) Garth proposed performance standards for cervical extrication collars but these standards have not been uniformly implemented.(39) There are a variety of different

cervical collars. Several studies compare collars alone or in combination with other immobilization devices employing a wide range of assessment criteria.(14,15,17,22,89,97)

Podolsky et al in 1983, evaluated the efficacy of cervical spine immobilization techniques utilizing goniometric measures.(77) Twenty-five healthy volunteers lying supine on a rigid emergency department resuscitation table were asked to actively move their necks as far as possible in six ways: flexion, extension, rotation to the right and left, and lateral bending to the right and left. Control measurements were made with no device and measurements were repeated following immobilization in a soft collar, hard collar, extrication collar, Philadelphia collar, bilateral sandbags joined with 3-inch-wide cloth tape across the forehead attached to either side of the resuscitation table, and the combination of sandbags, tape, and a Philadelphia collar. Hard foam and hard plastic collars were superior at limiting cervical spine motion compared to soft foam collars. Neither collars alone nor sandbags and tape in combination provided satisfactory restriction of cervical spine motion. Sandbags and tape immobilization was significantly better than any of the other methods of attempted cervical spinal immobilization used alone, for all six cervical spinal movements. The authors found that sandbags and tape in combination with a rigid cervical collar was the best means of those evaluated to limit cervical spine motion, particularly because the addition of a Philadelphia collar to the sandbag and tape construct was significantly more effective in reducing neck extension ( $p < 0.01$ ), from 15 degrees to 7.4 degrees, a change of 49.3%. Collar use had no significant additive effect for any other motion of the cervical spine. Sandbags as adjuncts to cervical spinal immobilization require more attention from care providers rather than less.(52) Sandbags are heavy and if the extrication board is tipped side to side during evacuation and transport, the sandbags can slide, resulting in lateral displacement of the victim's head and neck with respect to their torso. Sandbags can be taped in proper position to the

extrication board, but because they are small compared to the patient, this can be difficult and/or ineffective. Finally, they must be removed prior to initial lateral cervical spine x-ray assessment because they can obscure the radiographic bony anatomy of the cervical spine. For these reasons and the findings by Podolsky et al, sandbags and tape alone to attempt to immobilize the cervical spine is not recommended.(52,76)

In 1985, Cline compared methods of cervical spinal immobilization used in pre-hospital transport. They found that strapping the patient to a standard short board was superior to cervical collar use alone.(18) They noted no significant differences between the rigid collars they tested. McCabe and colleagues compared four different collars for their ability to restrict motion in flexion-extension, and lateral bending using radiographic assessment. They found that the Polyethylene-1 collar provided the most restriction of motion of the cervical spine, particularly with flexion.(62) Rosen in 1992 compared limitation of cervical spinal movement of four rigid cervical collars in 15 adults utilizing goniometric measurements. The vacuum splint cervical collar provided the most effective restriction of motion of the cervical spine of the four devices they tested.(86)

Graziano compared pre-hospital cervical spine immobilization methods by measuring cervical motion radiographically in the coronal and sagittal planes in 45 immobilized adults. The Kendrick's extrication device and the Extrication Plus-One device were nearly as effective in limiting cervical motion as the short immobilization board in their study. Both devices were superior to a rigid cervical collar alone.(42)

Cohen in 1990 described the Russell Extrication device (RED) for immobilization of patients with potential spine injuries. The RED was comparable to the short immobilization board for pre-hospital spinal immobilization.(19) Chandler et al compared a rigid cervical extrication

collar with the Ammerman halo orthosis in 20 males. The Ammerman halo orthosis in combination with a rigid spine board provided significantly better cervical spinal immobilization than a cervical collar and spine board. The Ammerman halo orthosis and spine board was equivalent to the standard halo vest immobilization device.(17)

Perry et al evaluated three cervical spine immobilization devices during simulated vehicle motion in six adults. Neck motion was assessed by three neurologists and neurosurgeons as to whether motion was "clinically significant". They found that substantial head motion occurred during simulated vehicle motion regardless of the method of immobilization.(75) They observed that the efficacy of cervical spine immobilization was limited unless the motion of the head and the trunk was also controlled effectively. Mazolewski et al tested the effectiveness of strapping techniques to reduce lateral motion of the spine of adults restrained on a backboard. Subjects were restrained on a wooden backboard with four different strapping techniques. The backboard was rolled to the side, and lateral motion of the torso was measured. The authors found that additional strapping securing the torso to backboard reduced lateral motion of the torso.(61)

Finally, the traditional method of moving a patient onto a long backboard has typically involved the logroll maneuver. The effectiveness of this transfer technique has been questioned.(29,83) Significant lateral motion of the lumbar spine has been reported to occur.(64,90) Alternatives to the logroll maneuver include the HAINES method and the multi-hand or fireman lift method.(45,93,95) In the HAINES method (acronym for High Arm IN Endangered Spine), the patient is placed supine, the upper arm away from the kneeling rescuer is abducted to 180 degrees, the near arm of the patient is placed across the patient's chest, and both lower limbs are flexed. The rescuer's hands stabilize the head and neck and the patient is rolled away onto an extrication board or device.(45) The multihand or fireman lift method involves

several rescuers on either side of the patient each of whom slides their arms underneath the patient and lifts the patient from one position to the another onto an extrication board or device.

The above review depicts the evolution of and underscores the diversity of techniques available for providing pre-hospital spinal immobilization of spinal injured patients during transport. These studies are limited by the fact that none of the studies evaluates the full range of available devices using similar criteria. Overall, it appears that a combination of rigid cervical collar immobilization with supportive blocks on a rigid backboard with straps to secure the entire body of the patient is most effective in limiting motion of the cervical spine after traumatic injury.(95) The longstanding practice of attempted spinal immobilization using sandbags and tape alone is insufficient.

#### Safety of Pre-hospital Spinal Immobilization Devices

Despite obvious benefits, cervical spinal immobilization has a few potential drawbacks. Immobilization can be uncomfortable, it takes time to apply, application may delay transport and it is associated with modest morbidity.(6,15,16,24,84,93,98)

Chan et al studied the effects of spinal immobilization on pain and discomfort in twenty-one non-injured adults. Subjects were placed in backboard immobilization for 30-minutes and symptoms were chronicled. All subjects developed pain which was described as moderate to severe in 55% of volunteers. Occipital headache, sacral, lumbar and mandibular pain were the most frequent complaints.(16) In a later study, Chan and others compared spinal immobilization on a backboard to immobilization with a vacuum mattress-splint device in 37 normal adults. The authors found that the frequency and severity of occipital and lumbosacral pain was significantly greater during backboard immobilization than on the vacuum mattress-splint device. Johnson and

Hauswald performed a prospective, comparative study of the vacuum splint device versus the rigid backboard. The vacuum splint device was significantly more comfortable than the rigid backboard and was faster to apply. The vacuum splint device provided better immobilization of the torso. The rigid backboard with head blocks was slightly better at immobilizing the head. Vacuum splint devices, however, are not recommended for extrication because they are reportedly not rigid enough, and they are more expensive. At a cost of approximately \$400, the vacuum splint device is roughly three times more expensive than a rigid backboard. (16)

Hamilton studied the comfort level of 26 adults on a full-body vacuum splint device compared to a rigid backboard, with and without cervical collars. Subjects graded their immobilization and discomfort. No statistically significant difference was found between the vacuum splint device and collar combination compared to the backboard-collar combination for flexion and rotation. The vacuum splint-collar combination provided significantly superior immobilization in extension and lateral bending than the backboard-collar combination. The vacuum splint alone, provided superior cervical spinal immobilization in all neck positions except extension, compared to the rigid backboard alone. A statistically significant difference in subjective perception of immobilization was noted, with the backboard alone less effective than the other three alternatives. In conclusion, the vacuum splint device, particularly when used with a cervical collar is an effective and comfortable alternative to a rigid backboard (+/- collar) for cervical spinal immobilization.(47)

Barney et al evaluated pain and discomfort during immobilization on rigid spine boards in 90 trauma patients and found that rigid spine boards cause discomfort.(5) Padding the rigid board improves patient comfort without compromising cervical spine immobilization.(99) Minimizing

the pain of immobilization may decrease voluntary movement and therefore decrease the likelihood of secondary injury.(15)

Cervical collars have been associated with elevations in intracranial pressure (ICP). Davies, prospectively analyzed ICP in a series of injured patients using the Stifneck rigid collar. ICP rose significantly ( $P < 0.001$ , mean 4.5 mmHg) when the collar was firmly in place. They cautioned that since head-injured patients may also require cervical spinal immobilization, it is essential that secondary insults producing raised ICP are minimized.(24) Kolb also examined changes in ICP after the application of a rigid Philadelphia collar in 20 adult patients. ICP averaged 176.8 mm H<sub>2</sub>O initially and increased to an average of 201.5 mm H<sub>2</sub>O after collar placement. Although the difference in ICP of 24.7 mm H<sub>2</sub>O was statistically significant ( $p = .001$ ), it remains uncertain that it has clinical relevance. Nonetheless, this modest increase in pressure may be important in patients who already have elevated intracranial pressure.(53) Plaisier et al in 1994 prospectively evaluated craniofacial pressure with the use of four different cervical orthoses. They found small changes in craniofacial pressure (increases) but no significant differences between the four collar types.(76)

Spinal immobilization increases the risk of pressure sores. Linares found pressure sores were associated with immobilization (patients who were not turned during the first two hours post-injury). The development of pressure sores was not related to mode of transportation to hospital or the use of a spinal board and sandbags during transportation.(57) Mawson et al prospectively assessed the development of pressure ulcers in 39 spinal cord-injured patients who were immobilized immediately after injury. The length of time on a rigid spine board was significantly associated with the development of decubitus ulcers within 8 days of injury ( $P = 0.01$ ). (60) Rodgers et al reported a marginal mandibular nerve palsy due to compression by a hard collar.

The palsy resolved uneventfully during the next two days.(81) Blaylock et al found that prolonged cervical spinal immobilization may result in pressure ulcers.(8) Improved skin care (keeping the skin dry), proper fitting (avoid excessive tissue pressure) and the appropriate choice of collars (those that do not trap moisture and do not exert significant tissue pressure) can reduce this risk.(7,8)

Cervical spinal immobilization may also increase the risk of aspiration and may limit respiratory function. Bauer et al, examined the effect of the Zee Extrication Device and the long spinal board on pulmonary function. They tested pulmonary function in 15 healthy, non-smoking men using forced vital capacity (FVC), forced expiratory volume in one second (FEV1), the ratio FEV1:FVC, and forced mid-expiratory flow (FEF 25%-75%). They found a significant difference ( $P < .05$ ) between pre-strapping and post-strapping values for three of the four functions tested when on the long spinal board. Similarly significant differences were found for three of the four parameters using the Zee Extrication Device. These differences reflect a marked pulmonary restrictive effect of appropriately applied entire body spinal immobilization devices.

Totten et al, evaluated the effect of whole-body spinal immobilization on respiration in 39 adults. Respiratory function was measured at baseline, once immobilized with a Philadelphia collar on a rigid backboard, and when immobilized on a Scandinavian vacuum mattress with a vacuum collar. The comfort levels of each of the two methods were assessed on a visual analog scale. Both immobilization methods restricted respiration by an average of 15%. The effects were similar under the two methods, although the FEV1 was lower on the vacuum mattress. The vacuum mattress was significantly more comfortable than the wooden backboard.(93)

In conclusion, cervical spine immobilization devices are generally effective at limiting cervical spinal motion, but may be associated with important but usually modest morbidity.

Cervical spinal immobilization devices should be used to achieve the goals of safe extrication and transport yet should be removed as soon as it is safe to do so.

## **SUMMARY**

Spinal immobilization can reduce untoward movement of the cervical spine and can reduce the likelihood of neurological deterioration in patients with unstable cervical spinal injuries following trauma. Immobilization of the entire spinal column is necessary in these patients until a spinal column injury (or multiple injuries) or a spinal cord injury has been excluded or until appropriate treatment has been initiated. While not supported by Class I or Class II medical evidence, this effective, time-tested practice is based on anatomic and mechanical considerations in attempt to prevent spinal cord injury and is supported by years of cumulative trauma and triage clinical experience.

It is unclear whether the spines of all trauma victims must be immobilized during pre-hospital transport. Many patients do not have spinal injuries and therefore do not require such intervention. The development of specific selection criteria for those patients for whom immobilization is indicated remains an area of investigation.

The variety of techniques employed and the lack of definitive evidence to advocate a uniform device for spinal immobilization, make immobilization technique and device recommendations difficult. It appears that a combination of rigid cervical collar with supportive blocks on a rigid backboard with straps is effective at achieving safe, effective spinal immobilization for transport. The longstanding practice of attempted cervical spinal immobilization using sandbags and tape alone is not recommended.

Cervical spine immobilization devices are effective but can result in patient morbidity. Spinal immobilization devices should be used to achieve the goals of spinal stability for safe extrication and transport. They should be removed as soon as definitive evaluation is accomplished and/or definitive management is initiated.

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

The optimal device for immobilization of the cervical spine following traumatic vertebral injury should be studied in a prospective fashion.

A reliable in-field triage protocol to be applied by EMS personnel for patients with potential cervical spine injuries following trauma needs to be developed.

## EVIDENTIARY TABLES

First Author Reference	Description of Study	Data Class	Conclusions
Markenson, <i>Pre-Hospital Emerg Care</i> 1999	An evaluation of the Kendrick extrication device for pediatric spinal immobilization.	Class III	KED provides excellent static and dynamic immobilization.
Perry, <i>Spine</i> , 1999	A experimental evaluation of 3 immobilization devices compared during simulated vehicle motion Neck motion was judged by 3 physicians.	Class III	Substantial amounts of head motion can occur during simulated vehicle motion regardless of the method of immobilization Movement of trunk can have equal effect as head motion on motion across the neck.
Bauer, <i>Ann Emerg Med</i> 1998	A study of the effect of spinal immobilization devices on pulmonary function in 15 men	Class III	Significant restriction of pulmonary function may result from spinal immobilization.
Mawson, <i>Am J Phys Med Rehabil</i> 1998	A prospective study to determine the association between immobilization and pressure ulcers in 39 SCI patients.	Class III	Time spent on backboard is significantly associated with pressure ulcers developing within 8 days.
Hauswald et al, <i>Academic Emerg Med</i> 1998	5-year retrospective chart review of patients with acute traumatic SCI from 2 centers. None of the 120 patients at the University of Malaya had spinal immobilization with orthotic devices during transport. All 334 patients at the University of New Mexico did. The hospitals were comparable. Neurological injuries were assigned to 2 categories, disabling or not disabling, by 2 blinded physicians. Data were analyzed using multivariate logistic regression. There was less neurological disability in the Malaysian patients (OR 2.03; 95% CI 1.03-3.99; p = 0.04). Results were similar when the analysis was limited to patients with cervical injuries (OR 1.52; 95% CI 0.64-3.62; p = 0.34).	Class III	Out-of-hospital immobilization has little effect on neurological outcome in patients with blunt spinal injuries.  The association between spinal column movement and the potential for SCI remains unclear.
Blaylock, <i>Ostomy Wound Management</i> 1996	A prospective study to determine the association between immobilization and pressure ulcers in 32 SCI patients.	Class III	Pressure sores developed mostly in patients who were turned after three hours. Most of those without sores were turned less than two hours after immobilization.
Johnson, <i>Am J Emerg Med</i> 1996	Measured immobilization and comfort on 10-point scale. The vacuum splint was compared with backboard.	Class III	Vacuum splints are more comfortable and faster to apply than backboards and provide a similar degree of immobilization. Vacuum splints not rigid enough for extrication and are more expensive.

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
Rodgers, <i>J Orthop Trauma</i> 1995	Case report of marginal mandibular nerve palsy due to compression by a cervical hard collar	Class III	The collar was removed; the palsy resolved uneventfully during the next 2 days.
Chan, <i>Ann Emerg Med</i> 1994	A prospective study of the effects of spinal immobilization on pain and discomfort in 21 volunteers after 30-minutes. All subjects developed pain.	Class III	Standard spinal immobilization may be a cause of pain in an otherwise healthy subject.
Liew, <i>ANZ Journal of Surgery</i> 1994	Two case reports of significant occipital pressure ulceration associated with the use of hard cervical collar.	Class III	Pressure ulcers may occur with the use of hard cervical collars.
Mazolewski, <i>Ann Emerg Med</i> 1994	A study to test the effectiveness of strapping techniques in reducing lateral motion on a backboard in laboratory in 19 adults.	Class III	Strapping should be added to the torso to reduce lateral motion on a backboard.
Plaisier, <i>J Trauma Inj Inf Crit Care</i> 1994	A prospective evaluation of craniofacial pressure of four different cervical orthoses in 20 adults. Pressure was measured at the occiput, mandible, and chin. Opinions on comfort were also collected.	Class III	The Newport or Miami J have favorable skin pressure patterns and superior patient comfort.
Raphael, 1994	A randomized, single-blind, crossover study of 9 patients scheduled for elective spinal anesthesia the cerebrospinal fluid pressure in the lumbar subarachnoid space was measured with and without a 'Stifneck' cervical collar applied.	Class III	There was a significant elevation of cerebrospinal fluid pressure in seven of the patients studied when the cervical collar was applied ( $p < 0.01$ ).
Chandler, <i>Ann Emerg Med</i> 1992	A comparison of the rigid cervical extrication collar with Ammerman halo orthosis in 20 men.	Class III	Ammerman halo orthosis and spine board provided significantly better immobilization, equivalent to halo vest.
Rosen, <i>Ann Emerg Med</i> 1992	A comparison of 4 cervical collars In 15 adult volunteers, by goniometry.	Class III	Vacuum splint cervical collar restricted range of motion of the cervical spine most effectively.
Schafermeyer, 1991	A study to assess the restrictive effects of two spinal immobilization strapping techniques on the respiratory capacity of 51 children.	Class III	Spinal immobilization significantly reduced respiratory capacity as measured by FVC in healthy patients 6 to 15 years old. There is no significant benefit of one strapping technique over the other.
Schriger, <i>Ann Emerg Med</i> 1991	A study comparing the flat backboard with occipital padding in achieving neutral position in 100 healthy volunteers.	Class III	Occipital padding places the cervical spine in more neutral alignment

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
Cohen, <i>Paraplegia</i> , 1990	A study analyzing the Russell Extrication device (RED) in 64 patients.	Class III	RED an effective spinal immobilization device with advantages over currently available devices.
Barney, <i>Ann Emerg Med</i> , 1989	Evaluated pain and discomfort during immobilization on rigid spine boards in 90 patients.	Class III	Spine boards may cause discomfort.
Toscano, 1988	Prevention of neurological deterioration before admission to hospital Retrospective review of 123 patients, 32 of 123 sustained major neurological deterioration from injury to admission	Class III	Appropriate handling of patients with spinal injury after trauma can reduce major neurological deterioration due to pathological motion of vertebral column.
Graziano, <i>Ann Emerg Med</i> , 1987	A radiographic comparison of pre-hospital cervical immobilization methods with the short board in 45 volunteers	Class III	The SBT proved to be significantly better (P < .05)
Linares, <i>Orthopedics</i> , 1987	A study of 32 SCI patients to determine whether pressure sores are associated with prolonged immobilization.	Class III	There is a strong association between 1-2 hrs of immobilization and the development of pressure sores.
McGuire, <i>Spine</i> , 1987	A radiographic evaluation of motion of the thoracolumbar spine in a cadaver with an unstable thoracolumbar spine, and a patient with a T12-L1 fracture dislocation.	Class III	Extreme motion at an unstable thoracolumbar spine segment can occur during the logroll maneuver The backboard and the Scoop stretcher offered adequate stabilization for thoracolumbar spine instability.
McCabe, <i>Ann Emerg Med</i> , 1986	A radiographic comparison of the 4 cervical collars in 7 adults.	Class III	Polyethylene - 1 provided most restriction in flexion.
Cline, <i>J Trauma</i> , 1985	A radiographic comparison of 7 methods of cervical immobilization in 97 adults.	Class III	The short-board technique appeared to be superior to all the three collars studied. The collars provided no augmentation of immobilization over that provided by the short board alone.

First Author Reference	Description of Study	Data Class	Conclusions
Podolsky, <i>J Trauma-Infection and Critical Care</i> , 1983	Static trial using goniometry comparing soft collar, hard collar, extrication collar, Philadelphia collar, bilateral sandbags and tape, and the combination of sandbags, tape and the Philadelphia collar in 25 normal adult volunteers.	Class III	Hard foam and plastic collars superior to soft collars Sandbags and tape in combination with a rigid cervical collar was the best means of those evaluated to limit cervical spine motion. The addition of a Philadelphia collar was significantly more effective in reducing neck extension ( $p < 0.01$ ), from 15 degrees to 7.4 degrees, a change of 49.3%. The combination of sandbags and tape alone does not allow sufficient restriction of extension.

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# **TRANSPORTATION OF PATIENTS WITH ACUTE TRAUMATIC CERVICAL SPINE INJURIES**

## **RECOMMENDATIONS**

Standards: There is insufficient evidence to support treatment standards.

Guidelines: There is insufficient evidence to support treatment guidelines.

Options: Expeditious and careful transport of patients with acute cervical spine or spinal cord injuries is recommended from the site of injury using the most appropriate mode of transportation available to the nearest capable definitive care medical facility.

## **RATIONALE**

Definitive assessment, resuscitation, and care for the patient with an acute traumatic cervical spinal injury cannot be rendered at the accident scene. Optimal care for patients with spinal injury includes initial resuscitation, immobilization, extrication and early transport of the patient to a medical center with the capability for diagnosis and treatment (3-5,9,11). Delay in transportation to a definitive treatment center is associated with less favorable outcome, longer hospitalizations, and increased costs (7,8,11).

Several modes of transportation are available to transport the spinal injury patient, primarily land (ambulance) and air (helicopter or fixed wing plane). Selection of the ideal mode of transportation for an individual patient depends on the patient's clinical circumstances, distance, geography, and availability. The goal is to expedite efficient, safe, and effective transportation, without an unfavorable impact on patient outcome. These issues provide the rationale to establish guidelines for transportation of patients with acute traumatic cervical spine and spinal cord injuries.

## **SEARCH CRITERIA**

A National Library of Medicine computerized literature search from 1966 to 2001 was undertaken using Medical Subject Headings in combination with “spinal injury” and “transport”. The search was limited to the English language. The first search term (keyword and exploded subject heading) yielded 8,493 articles. The second search term (as keyword) yielded 12,437 articles. A search combining both search terms provided 44 articles. All 44 abstracts were 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. A total of 13 articles were directly relevant to the subject of transportation of spinal injured patients. All articles provide Class III medical evidence. Pertinent articles are summarized in Evidentiary Table format.

## **SCIENTIFIC FOUNDATION**

Safe, rapid transport of the spinal injured patient to a medical facility for definitive care has long been a fundamental tenet of Emergency Medical Service (EMS) care delivery. No randomized clinical trials to establish the necessity or effectiveness of this strategy have been performed. A search of the literature does not provide Class I or Class II medical evidence in support of this practice.

One of the basic principles of pre-hospital spinal care is the early transfer of the injury victim to a center with the resources and expertise to manage the patient with an acute cervical spine or spinal cord injury (3-5,9,11). Early complications can be prevented and improved neurological outcomes have been reported when early transfer to a specialized spinal cord injury

(SCI) center is accomplished (5,11). During transportation every effort must be made to limit untoward spinal motion and to preserve neurological function (12).

Several options exist for the transportation of patients to a definitive care facility. The selection of the mode of transportation is based on the patient's clinical status and what is reasonable and available to achieve the goals of rapid transfer while maintaining effective medical support of the patient and proper spinal immobilization for patients at risk.

In 1974, Hachen described the creation of a nationwide emergency transportation protocol for spinal injury patients implemented in Switzerland in 1968 (5). All SCI patients in Switzerland were immediately transported to The National Spinal Injuries Centre in Geneva by the Swiss Air Rescue Organization. In the ten-year follow-up of this protocol published in 1977, Hachen reported that early transport from the site of the accident to the SCI center under close medical supervision was associated with no patient death during transport. Prior to 1968 multiple deaths occurred during transport secondary to acute respiratory failure before definitive care could be provided. After 1968, patients were transported rapidly with an onboard anaesthetist who provided respiratory, cardiac, and hemodynamic monitoring, resuscitation, and nasotracheal intubation as necessary. The average time for the rescue operation was reduced from 4.5 hours to 50 minutes. There was a significant reduction in cardiovascular and respiratory morbidity and mortality. The mortality rate for complete quadriplegic patients dropped from 32.5% in 1966 to 6.8% in 1976; and for incomplete cervical cord injury patients from 9.9% to 1.4% during the same time period. Hachen concluded that survival and outcome of patients with acute spinal cord injuries was enhanced by a well-organized medical system, rapid medical-supervised transfer by helicopter to a specialized center followed by definitive care in a SCI facility for aggressive management in the intensive care unit setting (5,6).

Zach, et al, in 1976 described their experience with 117 acute SCI patients managed per prospective protocol in the Swiss Paraplegic Centre in Basle, Switzerland. All patients were treated in the ICU setting with aggressive medical management and cardiac and blood pressure support. Outcome was stratified by initial injury and time of admission after injury. Sixty-two percent of cervical spinal cord injuries managed in this fashion improved at last follow-up. No patient with a cervical level injury worsened, 38% were unchanged. Of patients who arrived within 12 hours of injury, 67% improved compared to their initial neurological condition. Fifty-nine percent of patients admitted between 12 and 48 hours of injury showed neurological improvement. When admission occurred after 48 hours of injury improvement was seen in only 50% of patients. The authors concluded that early transport and “immediate medical specific treatment of the spinal injury” appeared to facilitate neurological recovery (13).

In 1984, Tator et al reported their experience with 144 patients with acute spinal cord injuries treated between 1974 to 1979 at the Acute Spinal Cord Injury Unit (ASCIU) at Sunnybrook Medical Centre in Toronto, Canada. They found a marked reduction in both morbidity and mortality following acute spinal cord injury for the group of patients managed from 1974 to 1979 compared to a similar group of patients managed from 1947 to 1973, before the creation of a dedicated, regional Spinal Cord Injury Unit. Reasons cited for these improvements included earlier transport to the ASCIU following trauma and better definitive management upon arrival (10).

In a subsequent 1993 publication comparing ASCIU patients managed from 1974 to 1981 to their historical population of patients managed from 1947 to 1973, Tator and colleagues noted a statistically significant difference in duration of time from injury to arrival, 5 hours for ASCIU patients compared to 13 hours for the pre-ASCIU group. They found a significant decrease in

the severity of spinal cord injury, (65% complete cervical lesions compared to 46% for ASCIU patients) and noted fewer complications, shorter hospital stays, and lower expenses for patients managed under the new ASCIU paradigm. Their findings support the advantages of early transport to a regional, specialized SCI center for definitive comprehensive care of patients with spinal cord injuries (10, 11).

Burney, et al, reviewed the means of transport and type of stabilization used for all patients with acute spinal cord injuries transferred to the University of Michigan Medical Center from 1985 to 1988 to determine the effect of these variables on impairment and neurological improvement. Sixty-one patients were reviewed. Twenty-five patients were transported by ground ambulance (41%), 33 by helicopter (54%), and three patients by fixed-wing aircraft (5%). Forty-three patients (70.5%) had cervical spinal injuries, 11 (18%) thoracic spine injuries, and seven (11.5%) lumbar spinal injuries. Fifty-one patients (84%) were transferred within 24 hours of injury. A variety of standard methods of stabilization were used during transport. No patient suffered an ascending neurological injury as a result of early transport. Level of function improved before discharge in 26 of 61 patients (43%). Patients transported to the Medical Center within 24 hours of injury were more likely to show improvement (25 of 51) than those transported after 24 hours (1 of 10). There was no significant difference in the probability of improvement between ground (8 of 25) or air (18 of 36) transport. The authors concluded that acute SCI patients could be safely transported by air or ground using standard precautions. They found that distance and the extent of the patient's associated injuries were the best determinants of the mode of transport (3).

Rural areas reportedly account for 70% of fatal accidents and rural mortality rates for victims of motor vehicle accidents are four to five times greater than those found in urban areas.

A prospective cohort study by Boyd, et al, examined the effectiveness of air transport of major trauma patients when transferred to a trauma center from a rural emergency room (2). The study consisted of 872 consecutive trauma patients admitted after long-distance transfer. The authors found that air transport was associated with a 25.4% reduction in predicted mortality ( $Z = 3.95$ ;  $p < 0.001$ ). The benefit of helicopter EMS transport was realized only in major trauma patients with a probability of survival of less than 90%. Thus, the benefits identified with early helicopter EMS transport were directly related to injury severity. It is unclear whether these findings can be extrapolated to spinal injured and/or spinal cord injury patients, since the authors did not stratify injuries by body systems in their report.

Neither land nor air transport has been reported in the literature to negatively impact the outcome of spinal injured patients when properly executed. One note of caution was offered by Armitage, et al. They described four spinal injured patients who developed respiratory distress/failure during airplane transport. They noted that since patients with cervical spinal cord injuries may have severely reduced pulmonary performance, measures to optimize oxygenation, humidification and pulmonary function in cervical spinal cord injury patients should be undertaken, particularly during air transport. (1)

## **SUMMARY**

The patient with an acute cervical spinal or spinal cord injury should be expeditiously and carefully transported from the site of injury to the nearest capable definitive care medical facility. The mode of transportation chosen should be based upon the patient's clinical circumstances, distance from target facility, geography to be traveled, and should be the most rapid means available. Patients with cervical spinal cord injuries have a high incidence of airway

compromise and pulmonary dysfunction, therefore respiratory support measures should be available during transport. Several studies cited suggest improved morbidity and mortality of spinal cord injured patients after the advent of sophisticated transport systems to dedicated SCI centers. These studies all provide Class III medical evidence on this issue.

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

Development and refinement of transportation protocols for patients with cervical spine and spinal cord injury should be undertaken and could be accomplished using a large prospectively collected data set. From these data case-control or comparative cohort studies could be structured to generate Class II evidence.

## EVIDENTIARY TABLE

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
Tator et al, 1993, <i>Surg Neurology</i>	A study of 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.
Armitage et al, 1990, <i>BMJ</i>	Case reports of four patients who developed respiratory problems during airplane transport.	Class III	Airplane air is less humid and measures to optimize humidity and pulmonary function travel in high cervical injury patients may be required
Boyd et al, 1989 <i>J Trauma-Injury Infection &amp; Crit Care</i>	A prospective cohort study to determine the effectiveness of air transport for major trauma patients when transferred to a trauma center from a rural emergency room.	Class III	Patients with severe multiple injury from rural areas fare better with helicopter EMS than ground EMS
Burney et al, 1989 <i>J Trauma-Injury Infection &amp; Crit Care</i>	Retrospective review of the means of transport and type of stabilization used for all patients with ASCI.	Class III	Acute SCI patients can be safely transported by air or ground using standard precautions. Distance and extent of associated injury are the best determinants of mode of transport.
Tator et al, 1984 <i>Can J of Surg</i>	A retrospective review of results of innovations between 1974 to 1979 at Sunnybrook Medical Centre in Toronto.	Class III	Patients transferred to the SCI unit earlier, with consequent marked reduction in complications and cost of care.
Hachen, 1977 <i>J Trauma</i>	A study of 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 <i>Paraplegia</i>	A study of 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.
Hachen, 1974 <i>Paraplegia</i>	Retrospective review of effectiveness of emergency transportation of spinal injury patients in Switzerland. Between 1965-1974 all SCI patients were immediately transported by air to SCI center. Mortality reduced to zero, during transport. Average time for the rescue operation reduced from 4.5 hours to 50 minutes. h Significant reduction in cardiovascular and respiratory morbidity.	Class III	Mortality and morbidity of patients with acute spinal injury is reduced by a well-organized medical response with smooth and rapid transfer by helicopter to a specialized SCI center.

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# CLINICAL ASSESSMENT FOLLOWING ACUTE CERVICAL SPINAL CORD INJURY

## RECOMMENDATIONS

### Neurological Examination:

Standards: There is insufficient evidence to support neurological examination standards.

Guidelines: There is insufficient evidence to support neurological examination guidelines.

Options: The ASIA international standards for neurological and functional classification of spinal cord injury is recommended as the preferred neurological examination tool for clinicians involved in the assessment and care of acute spinal cord injury patients.

### Functional Outcome Assessment

Standards: There is insufficient evidence to support functional outcome assessment standards.

Guidelines: The Functional Independence Measure (FIM) is recommended as the functional outcome assessment tool for clinicians involved in the assessment and care of acute spinal cord injury patients.

Options: The modified Barthel Index (MBI) is recommended as a functional outcome assessment tool for clinicians involved in the assessment and care of acute spinal cord injury patients.

## RATIONALE

Acute traumatic spinal cord injury affects 12,000 to 14,000 people in North America each year. The functional consequences of an acute spinal cord injury (ASCI) are variable, therefore

the initial clinical presentation of patients with ASCI is a key factor in determining triage and therapy and predicting prognosis. Consistent and reproducible neurological assessment scales are necessary to define the acute injury patient's neurological deficits and to facilitate communication about patient status to caregivers. Prognostic information provided by comparing injury victims to the outcomes of historical patients with similar injuries is of value to patients and families. The evaluation of new therapies proposed for the treatment of ASCI require the use of accurate, reproducible neurological assessment scales and reliable functional outcome measurement tools, not only to measure potential improvement following therapy, but to determine its functional significance. For these reasons, the clinical neurological assessment and the determination of functional abilities are important aspects of the care of patients with ASCI. The purpose of this review of the medical literature is to determine which neurological assessment scales and which functional impairment tools have the greatest utility in the care of patients with acute spinal cord injuries.

## **SEARCH CRITERIA**

A computerized search of the database of the National Library of Medicine of the literature published from 1966 to 2001 was performed. The search was limited to the English language and the human literature. The terms "spinal cord injury" or "spinal injury" were combined with the terms "classification or assessment", yielding 17,923 references. Another search using the terms "scale" or "weights and measures" or "index" or "abstracting and indexing" combined with the terms "spinal cord injury" or "spinal injury" yielded 337 references. These 337 references and the previous 17,923 references from the broader search were imported into a database and duplicates were eliminated. Papers germane to this topic were selected by reviewing their titles and abstracts. 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.

A total of fifty-three articles were accessed, reviewed, graded, and included in this review. There is no Class I medical evidence in the literature on this topic. There are two Class II comparative analyses of functional outcome scales. Twenty-seven pertinent manuscripts are summarized in Evidentiary Table format.

## **SCIENTIFIC FOUNDATION**

A variety of assessment systems are available for the documentation of neurological status of patients following ASCI. They include the Frankel Scale, the modified Frankel Scale, Lucas and Ducker's Neurotrauma Motor Index, the Sunnybrook, the Botsford, and the Yale scales, the NASCIS scale, the ASIA scale and the ASIA/International Medical Society of Paraplegia international standards for neurological and functional classification of spinal cord injury scale.(1-4,6-10,13,15,16,28,37,41,47)

Several of these assessment scales have been refined through serial iterations.(1-4,28,41,42,47) A few are widely used while others have not attained general acceptance and recognition. Ideally, the clinical neurological assessment of acute SCI victims should be uniform, reproducible and thorough, yet easy to use. The assessment tool must be detailed and precise to specifically document a given patient's injury and must provide descriptive measurement scales that allow determination of loss or gain of function with time and therapy. Finally, there must be measurement of the patient's functional abilities relative to their neurological examination to document whether losses or gains have meaningful significance to the patient and to accurately determine outcome. Whatever assessment system is used it must

have inter-rater reliability. Difficulties exist when clinicians utilize poorly defined measurement tools or different methods of neurological assessment to describe the same patient, hindering the definition (potentially the management) of that patient by different clinicians and the comparison of that patient with other patients with similar injuries. The accurate assessment of both the neurological status and the functional skills of acute spinal cord injury patients is essential for patient management, the conduct of research studies and comparisons of clinical therapeutic trials.

Numerous assessment scales have been used to evaluate patients with SCI. Scales may be divided into two general types. The first type is exam specific and focuses on the neurological deficits suffered as a result of SCI. These scales use the motor and sensory examination primarily (or exclusively), to assign a numerical value or letter grade.(1-2,6-12,15,28,37,41,47) The second type of scale focuses on functional skills, including a patient's ability to care for his or her self, participate in personal hygiene, transfer or ambulate.(3-4,14,22-24,26,29,34-35,38,42,45) In general, the first type of scale is used for the acute assessment of patients with SCI, whereas both assessment scales are used to define the chronically injured patient. More contemporary assessment scales incorporate both neurological examination scores and functional outcome scores in their overall definition of individual patients.(3,4,51)

### **Neurological Examination scales**

Frankel et al provided the first report of a stratified neurological scale employed to characterize patients with acute traumatic spinal cord injuries in 1969.(28) The authors used a five grade scale, A to E, to define spinal cord injuries in 682 patients managed at the Stoke Mandeville Hospital between 1951 and 1968. Grade A patients had complete motor and sensory lesions, Grade B patients had sensory only function below the level of injury. Grade C patients

had motor and sensory function below the level of injury but the motor function was useless. Grade D patients had motor useful, but not normal function below the level of spinal cord injury and Grade E patients had recovery, no motor, sensory or sphincter disturbance. The Frankel scale, as it became known, was widely adopted for use in the description of spinal cord injury patients and in assessment of their therapy (outcome) in the 1970's and 1980's.(1,2,28,29,41) It was easy to use, was based solely upon motor and sensory function and required very little patient assessment before classification into one of five grades. However, differentiation between patients classified into grades C and D was imprecise. These were broad injury groups with considerable range within each injury grade. The sensitivity of the Frankel scale to change in serial measurements, particularly among patients in grades C and D was poor. Significant improvement in patient function could occur over time without the patient advancing a Frankel grade.(1,2,7) Modifications of the Frankel scale were offered in an attempt to solve this shortcoming, however, the use of the Frankel scale as either an acute assessment tool or an outcome measure has been largely abandoned due to its lack of sensitivity. (3,4,6,7,13,20,42,47,48,50,51)

Institutions and investigators have proposed a variety of neurological assessment scales for spinal cord injury patients over the years. (1-4,6-12,15,28,37,41,47) In 1978, Bracken et al described the spinal cord injury severity scale developed at Yale University School of Medicine.(8) This scale combines motor and sensory function in selected muscle groups and dermatomes. Its primary focus is to distinguish between complete and incomplete spinal cord injuries. The sensory severity scale ranged from one point to seven points, and the motor severity scale ranged from one point to five points. The authors reported a strong correlation between the two scales and noted that "change scores" in motor and sensory function correlated

with outcome at discharge compared to admission. Their proposed assessment scales did not assess bowel or bladder function and suffered from grouping all patients into one of five possible motor scales and into one of seven possible sensory scales. It was difficult to memorize and hard to apply at the bedside.

In 1979, Lucas and Ducker at the Maryland Institute for Emergency Medical Services (MIEMS) developed a scoring system for patients with ASCI.(37) Their scale was based on motor function at and below the level of injury (Lucas and Ducker's Neurotrauma Motor Index), and was used to evaluate over 800 patients collected by the Nationwide Spinal Cord Injury Registry. It was later modified for a prospective study of spinal cord injury treatment regimes employed at MIEMS. The authors chose 14 muscles for examination and used mathematical analysis to predict a motor outcome score based upon the initial motor examination and an empirically derived understanding of the recovery rate of individual injury subtypes. The scoring system was limited in that many patients were excluded from the analysis (only 436 of over 800 patients were analyzed), the standard error of the predicted recovery score was large, and the calculations were cumbersome. Their scoring system was later modified by the American spinal Cord Injury Association (ASIA) into a motor index score.(23,42)

In the early eighties three different SCI neurological assessment scales were introduced, not one of which gained popular acceptance. (15,34,47). In 1980, Klose et al described the University of Miami Neuro-Spinal Index (UMNI).(34) It was composed of two sub-scales, one motor and the other sensory. The motor scale was scored on a zero to five-point scale for 44 muscle groups, resulting in a possible range of scores from zero to 220. Sensory scoring was a three-point scale for pinprick and vibratory sensation in 30 dermatomes on each side of the body. Initial inter-rater reliability was high among three physical therapists that examined ten

neurologically stable patients in the rehabilitation setting. Further studies were planned to determine the efficacy of UMNI in the acute setting and as an outcome tool. The Yale Scale was reported in 1981 by Cherazi et al at the Yale New Haven Medical Center.(15) The scale employed the British Medical Research Council's (BMRC) gradation (zero to five points) of muscle strength using 10 selected muscle groups from each side of the body. Sensory function was scored on a zero to two-point scale for superficial pain, position sense and deep pain. Bladder and bowel functions were not scored. In 1982, the Sunnybrook Cord Injury Scales for assessing neurological injury and recovery from spinal cord injury were proposed by Tator et al. (47) A ten point numerical neurological assessment scale was offered. It represented an improvement upon the Frankel scale in how sensory losses were classified. However, like the Frankel scale, motor grading was not very sensitive. The differentiation between grades three to five and grades six to eight that corresponded to Frankel grades C and D remained relatively imprecise and consisted of large, heterogeneous groups of patients. Bladder and bowel functions were not assessed.

In 1984, the American Spinal Injury Association (ASIA) generated standards for the neurological classification of spinal injury patients.(1) The neurological assessment used a ten-muscle group motor index score (zero to five points scale) and incorporated the Frankel classification as the functional abilities assessment tool. The sensory examination was not scored but the most cephalad level of normal sensation was noted. These standards were revised in 1989 to provide better, more specific sensory level determinations.(2) In 1991, Priebe and Waring examined the inter-observer reliability of the revised ASIA standards (1989 version).(44) Sample patients in quiz format were given to house-staff and faculty of a department of Physical Medicine and Rehabilitation. They were asked to classify each patient with respect to sensory

level, motor level, zone of injury and Frankel classification according to the 1984 ASIA standards. Two months later they were asked to complete another quiz using the 1989 ASIA standards. While the percent correct answers improved using the 1989 ASIA standards the authors conclude that inter-observer reliability was “less than optimal” with a kappa coefficient of 0.67, indicating agreement between observers but only within the range of fair agreement.(44)

Botsford et al introduced a new functionally oriented neurological grading system that incorporated motor and sensory function, rectal tone and bladder control.(7) The motor assessment scored on a zero to five-point scale assessed flexor and extensor groups at major joints (hence a “more functional” motor assessment). Sensory function was graded on a zero to ten-point scale and was divided into five categories. Voluntary rectal contraction was scored on a ten-point scale (zero, five or ten points). Bladder function was divided into normal and not normal and assigned 5 points. The authors applied their proposed grading system to a historical group of patients who had initially been assessed and classified according to the Frankel scale. They concluded that the new grading system was more sensitive for the detection of improvement in the neurological examination and in functional performance over time.(7)

Two national acute spinal cord injury studies (NASCIS I and II) were accomplished in the late 1980’s and early 1990’s in examination of methylprednisolone as a treatment for patients with acute spinal cord injuries.(9-12) Investigators at multiple centers in North America used a motor assessment scale (NASCIS scale) that evaluated motor function in fourteen muscle groups graded from one point to six points (NASCIS I),(9-10) or zero to five points (NASCIS II).(11-12) Scores for the right and left sides of the body were obtained independently. Sensory function was divided into pin-prick and light touch in dermatomes C2 through S5 and was scored on a zero to three-point scale. Functional abilities were not assessed in the NASCIS I and II

studies. Like most other neurological assessment scoring systems before the NASCIS scales, there was no documentation of inter-observer reliability, despite the large numbers of patients examined and entered into the NASCIS I and II trials.

In 1992, ASIA generated new standards for neurological and functional classification of spinal cord injury in conjunction with the International Medical Society of Paraplegia (IMSOP).(3) These standards replaced the revised 1989 version. The new assessment recommendations included motor index scores, sensory examination scores (zero to two point scale), the ASIA impairment scale (modified Frankel classification) and incorporated the Functional Independence Measure (FIM). FIM is a functional assessment tool and is used to assess the impact of SCI on the patient's functional abilities. It quantifies the extent of individual disability and complements the neurological assessment by providing scoring for activities of eating, grooming, bathing, dressing upper body, dressing lower body and toileting. (20-23) Improvements in neurological function over time or with treatment (as documented by neurological examination scales) can be measured in terms of functional or meaningful improvement to the patients with the addition of FIM in the assessment battery.

Davis et al measured the inter-rater reliability of Frankel classification and the Sunnybrook scale by experienced personnel who were provided with concise definitions.(19) They demonstrated high inter-rater reliability of the Frankel classification and Sunnybrook scales (Pearson correlation coefficients 0.71 to 0.91), with 94% to 100% intra-rater agreement. Kappa values were not provided. The authors concluded that both assessment systems corresponded well to total sensory and motor function in SCI patients but were insensitive to ambulation skills and bladder function.(19)

In 1993, Bednarczyk and Sanderson reported on the ability of three different classification systems to describe spinal cord injury patients and to compare the correlation between the three scales when provided by a single trained provider.(5) They compared the NASCIS scale with the ASIA scale and the BB (wheelchair basketball) Sports Test. The authors found that that the ASIA scale had the greatest discrimination in grouping subjects with SCI into mixed injury categories and into incomplete injury categories. The BB Sports Test had a positive correlation with the ASIA scale (Spearman's rho correlation coefficient, 0.81). The NASCIS scale had a negative correlation with both the ASIA scale (-0.66) and the BB Sports Test (-0.48). In contrast, El Masry et al retrospectively assessed 62 consecutive ASCI patients and compared ASIA and NASCIS motor scores with conventional motor examinations. They found that both motor assessment scales were representative of the conventional motor scores reported for these patients and could be used to quantify motor deficits and recovery following ASCI.(26)

Lazar et al, in 1989, evaluated the relationship between early motor status and functional outcome after SCI prospectively in 52 quadriplegic and 26 paraplegic patients.(36) Motor status was measured within 72 hours of injury and quantified with the ASIA motor index score (MIS). Functional status was evaluated with the modified Barthel index (MBI). A senior physical therapist completed MIS and MBI assessments on each patient upon admission to the spinal cord intensive care unit and every 30 days during rehabilitation. The authors found that early motor function correlated well with average daily improvement in functional status including self-care and mobility ( $p = .001$ ). The initial MIS strongly correlated with the functional status of quadriplegics at admission ( $p = .001$ ), at 60 days, and at rehabilitation discharge ( $p = .001$ ), but had poor correlation in paraplegic patients. The MIS correlated significantly with the MBI self-care sub-score at 60 days and at discharge ( $p = .01$ ), but not with the MBI mobility sub-score.

They concluded that the MIS a useful tool in predicting function during rehabilitation, although individual differences in ambulation, particularly for patients with paraplegia, limit the predictive utility of this index.(36)

Waters et al, in 1994, compared the strength of 36 acute SCI patients as determined by the ASIA motor score with motor scores based on biomechanical aspects of walking to predict ambulatory performance.(49) The authors found that the ASIA scoring system compared favorably with the biomechanical scoring system, and was a relatively simple clinical measure that correlated strongly with walking ability. In 1995, Marino et al compared the ASIA motor level (ML) and the upper extremity motor score (UEMS) to the neurological level (NL) of injury in fifty quadriplegics.(40) At 12 months post-injury, Quadriplegia Index of Function (QIF) assessments were obtained. Spearman's correlations were calculated. The authors found that the ML was more highly correlated to the UEMS and the QIF than the NL. The UEMS had the highest correlation to the QIF feeding score, 0.78. They concluded that the ASIA ML and UEMS better reflect the severity of impairment and disability. Similarly, Ota et al compared the ASIA motor scores and neurological level of injury (NL) with FIM in 100 Frankel grade A and B patients. They found that the motor score reflected the patients' disability as determined by FIM better than the ASIA level of injury. (43)

Wells and Nicosia compared the utility and limitations of five different spinal cord injury scoring systems applied by a single skilled observer in the assessment of 35 consecutive SCI patients; the Frankel classification, the Yale Scale, the ASIA motor index score, the Modified Barthel Index (MBI) and FIM scores.(51) The authors found that the Frankel classification correlated strongly with the Yale scale and the ASIA motor index scores but poorly with MBI and FIM. These three assessment scales shared a focus on impairment measurement. The MBI

and the FIM score correlated strongly, but weakly with the other scales and shared a focus on disability. They concluded that one classification system or scale alone does not adequately describe SCI patients in both the acute and follow-up settings. They favored a combination of two scales to characterize ASCI patients, one based on neurological impairment and the other on functional disability.(51)

Jonsson et al, evaluated inter-observer reliability of the 1992 ASIA standards for neurological and functional classification of spinal cord injury.(33) Two physicians and two physiotherapists classified 23 SCI patients according to the 1992 recommendations. Kappa values for pin-prick scores varied from 0 to 0.83 (poor to very good), from 0 to 1.0 for light touch scores and from 0 to 0.89 for motor function. They found weak inter-rater reliability for scoring patients with incomplete spinal cord injuries. Cohen et al performed further tests of reliability of the 1992 ASIA standards.(17) One hundred and six professionals in the field of SCI completed a pretest and posttest in which they classified two SCI patients by sensory and motor levels, zone of partial preservation (ZPP), ASIA impairment scale and completeness of injury. Percent 'correct' was calculated for each tested criterion. The authors reported that participants had very little difficulty in correctly classifying the patient with a complete SCI but had variable success characterizing the patient with an incomplete SCI. They concluded that further refinement of the 1992 ASIA standards and more training in their application was required.(17)

In 1996, ASIA/IMSOP provided a revised version of the international standards for neurological and functional classification of spinal injury (an update of their 1992 recommendations).(4) Further refined by input from numerous international organizations, the combination of the ASIA impairment scale, the ASIA motor index score, the ASIA sensory scale

and FIM is considered to be the most representative assessment and classification tool for patients with acute spinal cord injuries. It was felt to be an improvement on the pre-existing 1992 standards, which were subject to criticism.(4,13,17,18,25,33,42-44)

### **Functional outcome scales**

Functional outcome scales are nonspecific measures of human performance ability relevant to medical rehabilitation, that is, how a person functions with activities of everyday life. Several scales have been developed in an effort to accurately characterize an injury victim's functional skills and disabilities in order to quantify his or her functional independence.(3,4,14,20,22,24,25,27,29,30,34,35,38,39,42,43,45,46) They attempt to determine a patient's ability or inability to live independently. Scales for functional rating include, the Barthel Index (BI), Modified Barthel Index (MBI), the Functional Independence Measure (FIM), the Quadriplegic index of Function (QIF), the spinal cord independence measure (SCIM), the Walking Index for spinal cord injury (WISCI), and the spinal cord injury functional ambulation inventory (SCI-FAI). (3,4,14,20,22,24,25,27,29,30,34,35,38,39,42,43,45,46) They are applicable to a wide range of nervous system disorders, however the QIF, the SCI-FAI and the SCIM are more specific for patients with SCI.(14,27,29) All of these scales have been successfully used to characterize to SCI patients. (3,4,14,20,22,24,25,27,29,30,34,35,38,39,42, 43,45,46)

Among many available functional assessment scales, the BI has been one of the most popular.(35,38,52) It has been utilized for both the characterization of individual patients, and in the evaluation of the efficacy of various rehabilitation programs. The BI has ten ratable patient skill items. Values are assigned to each item (zero, five points or ten points) based on the amount of physical assistance required to perform each task. A BI total score ranges from zero

to 100 points (0: fully dependent; 100: fully independent). In the original version, each item is scored in three steps.(38) The modified Barthel Index (MBI) with a five-step scoring system, appears to have greater sensitivity and improved reliability than the original version, without examination difficulty or an increase in implementation time. Shaw et al found the internal consistency reliability coefficient for the MBI to be 0.90, compared to 0.87 for the original index.(45) In another study, Kucukdeveci et al, evaluated the reliability and validity of the MBI in 50 inpatient rehabilitation SCI patients in Turkey.(35) Patients were assessed by the MBI at admission and discharge. Reliability was tested using internal consistency, inter-rater reliability and the intra-class correlation coefficient. Construct validity was assessed by association with impairments (ASIA) and by Rasch analysis. Internal consistency was 0.88. The level of agreement between two raters was sufficient with Kappa levels of 0.5. The intra-class correlation coefficient was 0.77. However, Rasch analysis revealed that bladder and bowel items of the MBI misfit the construct. The authors concluded that adaptation of the modified Barthel Index is useful in assessment of SCI patients in Turkey as long as its limitations are recognized.(35)

The FIM was developed to provide uniform assessment of severity of patient disability and medical rehabilitation outcome.(20-23) It is an 18-item, 7-level scale designed to assess severity of patient disability, estimate burden of care and determine medical rehabilitation functional outcome. The FIM has emerged as a standard assessment instrument for use in rehabilitation programs for disabled persons.(4,20-23,25,30-32,42,43,51,53) In 1993, Dodds et al assessed FIM with respect to validity and reliability in characterizing 11,102 general rehabilitation patients in the Uniform Data System from the Pacific Northwest.(25) They compared admission and discharge FIM scores and assessed for validation using several

hypotheses. The authors found high overall internal consistency and that FIM identified significant functional gains in patients over time. FIM discriminated patients on the basis of age, comorbidity, and discharge destination. The authors concluded that FIM had high internal consistency, adequate discriminative capabilities and was a good indicator of burden of care.(25)

Hamilton et al have assessed inter-rater agreement of the seven-level FIM in two separate reports (31,32). In the 1991 report, two or more pairs of clinicians assessed each of 263 patients undergoing inpatient medical rehabilitation at 21 US hospitals subscribing to the uniform data system (UDS) for medical rehabilitation.(31) Criteria were intra-class correlation coefficient (ICC) (ANOVA) for total FIM, and FIM sub-scores greater than or equal to 0.90 (five of six sub-scores must be .90 or more; no ICC could be 0.75 or less). Kappa values (unweighted) for individual FIM items had to be greater than or equal to 0.45 for at least 15 of the 18 items. The total FIM ICC was 0.97. Sub-score ICC's were: self-care; 0.96, sphincter control; 0.94, mobility (transfers); 0.96, locomotion; 0.93, communications; 0.95 and social cognition; 0.94. The FIM item Kappa mean was 0.71, (range 0.61 to 0.76). The authors concluded that the 7- level FIM appears to have good clinical inter-rater agreement based on their methods of analysis.(28) In 1994, Hamilton et al reported inter-rater reliability among clinicians from 89 US rehabilitation facilities within the UDS. One thousand eighteen patients were characterized.(32) Using similar analysis methods reported earlier, total FIM ICC was 0.96. Sub-score ICC's ranged from 0.89 to 0.96. FIM item Kappa coefficients ranged from 0.53 to 0.66. For a subset of institutions that met UDS reliability criteria, kappa values ranged from 0.69 to 0.84. The authors concluded that FIM is reliable when used by trained and tested inpatient medical rehabilitation clinicians. Stineman et al utilized the FIM instrument and a function-based strategy to generate functional outcome benchmarks among 3604 spinal cord injury patients they reviewed.(46) They found

that the majority of patients whose motor-FIM scores at admission were above 30 were able to groom, dress the upper extremity, manage bladder function, use a wheelchair and transfer from bed to chair by the time of discharge from rehabilitation. Most patients with scores above 52 attained independence in all but the most difficult FIM tasks such as bathing, tub transfers and stair climbing. The authors concluded that these “FIM item attainment benchmarks” may be useful in counseling patients, predicting outcome and anticipating post-discharge patient care needs.

The Quadriplegia Index of Function (QIF) was developed in 1980 because the Barthel Index was deemed too insensitive to document the small but significant functional gains made by quadriplegics (tetraplegics) during medical rehabilitation.(29,53) The QIF is comprised of variables that are each weighted and scored (transfers, grooming, bathing, feeding, dressing, wheelchair mobility, bed activities, bladder and bowel program and understanding of personal care). A final score ranging from zero to 100 points is derived that characterizes each patient’s functional abilities and serves as a reference for future assessment. Gresham et al tested the QIF on a group of 30 complete quadriplegic patients at admission to and discharge from inpatient medical rehabilitation.(29) Resultant scores were compared to those simultaneously obtained by the Barthel Index and the Kenny Self-Care Evaluation (KSCE). The QIF was found to be more sensitive for patient functional improvement (46%) than that defined by the BI (20%) or the KSCE (30%). The QIF was also tested for reliability. Ratings by three different nurses, working independently, were found to be significantly positively correlated for all sub-scores ( $p < .001$ ). Gresham et al concluded that the QIF provides a useful option in choosing a functional assessment instrument for quadriplegic patients.(29)

Yavuz et al compared ASIA scores, the QIF and FIM in twenty-nine subjects with cervical SCI.(53) The same examiner used all three scales at admission to and discharge from the rehabilitation center. They identified strong correlation of ASIA scores to both FIM and the QIF. Feeding and dressing categories of QIF showed an even stronger correlation to ASIA motor scores, however statistical significance was the same for corresponding categories of FIM and QIF. The percent of recovery on ASIA motor scores was significantly correlated only to gain in QIF scores, not FIM. The authors recommended that additions to the FIM may be useful, especially in the feeding, dressing and bed activity categories in order to improve sensitivity.(53)

Catz et al developed a new disability scale specific for patients with spinal cord pathology, SCIM and compared it to FIM in the assessment and characterization of 30 patients. (14) Two pairs of trained staff members recorded scores one week after admission and every month thereafter during hospitalization. The authors found remarkable consistency between each pair of raters for all tasks assessed, Kappa coefficient between 0.66 and 0.98. The authors found the SCIM more sensitive than FIM to changes in function of spinal cord lesion patients: SCIM detected all functional changes detected by FIM, but FIM missed 26% of changes detected by SCIM scoring. The authors concluded that SCIM may be a useful instrument for assessing functional changes in patients with lesions of the spinal cord.

The Walking index for SCI (WISCI) was proposed as a scale to measure functional limitations in walking of patients following SCI.(24) It incorporates gradations of physical assistance and devices required for walking following paralysis of the lower extremities secondary to SCI. The purpose of the WISCI is to document changes in functional capacity with respect to ambulation in a rehabilitation setting. A pilot study of the WISCI was completed utilizing video clips of patients walking. Raters at eight international centers completed the

assessment skills. The concordance for the pilot data was significant. Inter-rater reliability revealed 100% agreement. The authors conclude that the WISCI showed good validity and reliability but needed further evaluation before it can serve as a useful tool for clinical studies. (24)

Finally, the SCI-FAI is a functional observational gait assessment instrument developed at the University of Miami that addresses three key domains of walking function in individuals with SCI: gait parameters/symmetry, assistive device use and temporal-distance measures.(27) The authors assessed its validity and reliability in a study of 22 patients with incomplete spinal cord injuries examined by four trained raters. Inter-rater reliability was good for all four raters, ICC range = 0.850 to 0.960. A moderate correlation (Pearson  $r = 0.58$ ) was found between change in gait score and lower extremity strength. The authors concluded that the SCI-FAI is a reliable, valid and relatively sensitive measure of walking ability in individuals with SCI.

## **SUMMARY**

A variety of injury classification schemes have been utilized to describe patients who have sustained spinal cord injuries. There are two general types of assessment scales, neurological examination scales and functional outcome scales. The most accurate and meaningful description of spinal cord injury patients, in the acute setting and in follow-up, appears to be that accomplished by using a neurological scale in conjunction with a functional outcome scale. At present, the most utilized and studied neurological assessment scales are the ASIA scores including the motor index scores, sensory scores and the ASIA Impairment scale. After multiple revisions and several refinements these scales are easy to apply, and are reliable.

The 1996 ASIA recommendations for international standards of neurological and functional classification of spinal cord injury include the ASIA scales, as noted, and the

Functional Independence Measure (FIM). FIM as a functional outcome tool has been studied extensively. It appears to be the best functional outcome scale used to describe disability among SCI patients, both early and late after injury. It is easy to administer and is valid and reliable. Inter-rater agreement with FIM has been high in several studies with reported Kappa values of 0.53 to 0.76.

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

Any future investigation of or clinical trial involving spinal cord injury patients must include both a neurological examination scale and a functional outcome assessment. Therapeutic trials of spinal cord injury patients should include reliable neurological and functional scoring systems and should verify the validity and inter-rater reliability of those scoring scales as part of the investigational paradigm.

## EVIDENTIARY TABLE: Neurological Examination Scales

First Author Reference	Description of Study	Data Class	Conclusions
Jonsson, 2000, <i>Spinal Cord</i>	A study of the inter-rater reliability of the ASIA ISCSCI-92. Physicians and physiotherapists classified 23 patients according to the ISCSCI-92 and calculated Kappa values.	Class III	This study indicates a weak inter-rater reliability for scoring incomplete SCI lesions using the 1992 ASIA standards.
Cohen. 1998, <i>Spinal Cord</i>	This study was a test of the ASIA ISCSCI-92. Participants completed a pretest and posttest in which they classified two patients who had a SCI.	Class III	Further revision of the ASIA 1992 standards and more training was needed to ensure accurate classification of spinal cord injury.
El Masry, 1996, <i>Spine</i>	A study to assess the reliability of the ASIA and NASCIS motor scores. The motor scores of 62 consecutive acute SCI patients were retrospectively reviewed.	Class III	The differences in correlation coefficients between the ASIA motor score and the NASCIS motor score were not statistically significant. The ASIA and NASCIS motor scores can both be used for the neurological quantification of motor deficit and motor recovery.
Wells, 1995, <i>J Spinal Cord Med</i>	A comparison of the Frankel Scale, Yale Scale, Motor Index Score, MBI, Functional Independence Measurement (FIM) in 35 consecutive acute SCI patients.	Class III	The best assessment tool is a combination of two scales, one based on neurological impairment and the other on functional disability.
Waters, 1994, <i>Arch Phys Med Rehab</i>	An assessment of strength using motor scores derived from ASIA compared with motor scores based on biomechanical aspects of walking in predicting ambulatory performance in 36 SCI patients.	Class III	The ASIA scoring system compared favorably with the biomechanical scoring system. ASIA motor score strongly correlates with walking ability.
Davis, 1993, <i>Spine</i>	A prospective study of 665 acute SCI patients to determine the reliability of the Frankel and Sunnybrook scales.	Class III	Demonstrated high inter-rater reliability of Frankel and Sunnybrook scales. Both scales correspond to total sensory and motor function but are insensitive to walking and bladder function.
Bednarczyk, 1993, <i>J Rehab Research &amp; Dev</i>	A study comparing ASIA scale, NASCIS scale and wheelchair basketball (BB) Sports Test in 30 SCI patients classified by the same examiner.	Class III	ASIA Scale showed the greatest discrimination in grouping subjects with ASCI. NASCIS scale had negative correlation with ASIA scale and BB sports test.
Botsford, 1992, <i>Orthopedics</i>	Description of a new functionally oriented scale with assessment of motor and sensory function, rectal tone and bladder function.	Class III	Botsford scale was sensitive for the detection of improvement in function over time following SCI.

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
Priebe, 1991, <i>Am J Phys Med &amp; Rehab</i>	A study of the interobserver reliability of the 1989 revised ASIA standards assessed by quiz given to 15 physicians.	Class III	The interobserver reliability for the revised ASIA (1989) standards were improved compared to previous versions, but less than optimal. Changes were recommended.
Bracken, 1990 <i>New England Journal of Med</i>	Multi-center North American trial examining effects of methylprednisolone or naloxone in ASCI. (NASCIS II)	Class III for neurological assessment	Motor scores of 14 muscles on 0-5 point scale, right side of body only. Sensory scores of pin prick and light touch, 1-3 point scale, bilateral. No inter-rater reliability comparison.
Lazar, 1989, <i>Arch Phys Med &amp; Rehab</i>	A prospective study of the relationship between early motor status and functional outcome after SCI in 78 patients. Motor status was measured by the ASIA Motor Index Score and functional status was evaluated with the Modified Barthel Index.	Class III	The MIS correlated well with functional status for quadriplegic patients, poorly for paraplegic patients. Individual differences in ambulation limit its predictive utility.
Bracken, 1985 <i>J Neurosurg</i>	Multi-center North American trial examining effects of methylprednisolone in ASCI. (NASCIS I)	Class III for neurological assessment	Motor scores of 14 muscles on 1-6 point scale. Right side of body only. Sensory scores of pinprick and light touch, 1-3 point scale, bilateral. No inter-rater reliability comparison.
Tator, 1982, <i>Early Management of Spinal Cord Injury</i>	Initial description of the Sunnybrook Scale, a 10 grade numerical neurological assessment scale.	Class III	Improvement from the Frankel scale. Motor grading subdivided but not very sensitive.
Cherazi, 1981, <i>J Neurosurg</i>	Initial description of the Yale scale and its use in a group of 37 patients with SCI.	Class III	Provides assessment of the severity of SCI.
Lucas, 1979, <i>American Surgeon</i>	Initial description of a motor classification of patients with SCI and its use in 800 patients.	Class III	Allows the clinical researcher to evaluate current treatments and assess the potential of new treatment regimes.
Bracken, 1977 <i>Paraplegia</i>	Description of 133 ASCI patients classified using motor and sensory scales developed by Yale Spinal Cord Injury Study Group.	Class III	Considerable discrepancy between motor and sensory impairment scales among patients with greater motor than sensory loss.
Frankel, 1969 <i>Paraplegia</i>	The first clinical study of the Frankel scale to assess neurologic recovery in 682 patients treated with postural reduction of spinal fractures.	Class III	First neurological examination scale for ASCI.

## EVIDENTIARY TABLE: Functional Outcome Scales

First Author Reference	Description of Study	Data Class	Conclusions
Field-Fote, 2001, <i>J Rehabil Med</i>	SCI-FAI offered as functional assessment scale for gait assessment.	Class III	Reliable and relatively sensitive measure of walking ability in patients with SCI. Interrater reliability good. No kappa values offered.
Kucukdeveci, 2000, <i>Scan J of Rehab Med</i>	To determine the reliability and validity of the MBI in Turkey.	Class III	Adaptation of the modified Barthel Index successful in Turkey as long as its limitations are recognized. Kappa values > 0.5.
Ditunno, 2000, <i>Spinal Cord</i>	WISCI offered as index for ambulation skills following SCI in pilot study.	Class III	Good reliability, excellent interrater reliability but needs assessment in clinical settings.
Yavuz, 1998, <i>Spinal Cord</i>	Assessment of the relationship of two functional tests, FIM and QIF, to ASIA scores.	Class III	Strong correlation between FIM and QIF to ASIA scores.
Catz, 1997, <i>Spinal Cord</i>	SCIM offered as new disability scale for spinal cord lesions. Thirty patients assessed with SCIM and FIM.	Class III	SCIM more sensitive than FIM.
Hamilton, 1994, <i>Scan J of Rehab Med</i>	Assessment of interrater agreement of FIM in 1018 patients in 89 UDS hospitals.	Class II	Kappa values for 7 level FIM ranged from 0.53 to 0.66. Kappa values higher in subset of UDS hospitals with experienced rehab clinicians, 0.69 to 0.84.
Dodds, 1993 <i>Arch Phys Med Rehabil</i>	Assessment of reliability of FIM in characterizing 11,102 UDS rehab patients.	Class III	FIM has high internal consistency, adequate discriminative capabilities, and was good indicator of burden of care.
Hamilton, 1991, <i>Arch Phys Med Rehabil</i>	Interrater agreement assessment of FIM in 263 patients in 21 UDS hospitals.	Class II	Kappa values for 7 level FIM ranged from 0.61 to 0.76, mean 0.71.
Shah, 1989, <i>Journal of Clin Epidemiology</i>	Description of Modified Barthel Index (MBI).	Class III	The MBI has greater sensitivity and improved reliability than the original version, without additional difficulty or implementation time.
Gresham, 1986 <i>Paraplegia</i>	Assessment of QIF as functional scale, compared to Barthel Index.	Class III	The QIF was more sensitive and reliable than the Barthel Index.

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# **RADIOGRAPHIC ASSESSMENT OF THE CERVICAL SPINE IN ASYMPTOMATIC TRAUMA PATIENTS**

## **RECOMMENDATIONS**

Standards: Radiographic assessment of the cervical spine is not recommended in trauma patients who are awake, alert, and not intoxicated, who are without neck pain or tenderness, and who do not have significant associated injuries that detract from their general evaluation.

Guidelines: None

Options: None

## **RATIONALE:**

Spinal cord injury is a potentially devastating consequence of acute trauma and can occur with improper immobilization of an unstable cervical spine fracture. Immobilization of an injury victim's cervical spine following trauma is now standard care in the vast majority of Emergency Medical Services (EMS) systems. Immobilization of the cervical spine is maintained until spinal cord or spinal column injury is ruled out by clinical assessment and/or radiographic survey. Radiographic study of the cervical spine of every trauma patient is costly and results in significant radiation exposure to a large number of patients, very few of whom will have a spinal column injury. The purpose of this review is to define which radiographic studies are necessary in the assessment of the cervical spine in asymptomatic patients following trauma.

## **SEARCH CRITERIA:**

A National Library of Medicine computerized literature search from 1966 to 2001 was performed using Medline and keywords "spinal cord injury", "spinal fractures", or "spinal

injuries”. This resulted in 7994 matches. Combination with the keyword “cervical” resulted in 1844 matches. These references were limited to human studies and the English language, resulting in 1268 articles. Combination with the keywords “clearance,” “diagnosis,” or “radiography” yielded 184 matches. The titles and abstracts of these 184 articles were reviewed. All papers focusing on clinical decision-making with regard to the diagnosis of cervical spine injury in adult victims of trauma were included. 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. The bibliography developed by the EAST practice parameter workgroup for cervical spine clearance was reviewed, (15) as was the reference list developed by the NEXUS (National Emergency X-radiography Utilization Study) group.(5,7)

Nine large, prospective cohort studies were identified. These nine studies provide Class I evidence. There were no randomized controlled trials in the literature addressing this issue. Numerous smaller studies, case series, and retrospective cohort studies were identified, which provide corroborating Class II and Class III evidence. This guideline was generated from these articles. The thirteen articles most germane to this issue are summarized in Evidentiary Table format.

## **SCIENTIFIC FOUNDATION**

A missed cervical spine injury can result in devastating neurological injury. For this reason, radiographic assessment of the cervical spine is liberally employed in patients following acute trauma. Cervical spine radiographs are relatively inexpensive and are easy to obtain. Computed tomography and magnetic resonance imaging may also be used to evaluate the selected spine in

certain circumstances. These studies are more expensive yet remain widely available. Since the overall incidence of cervical spinal column injury in the general trauma population is low, a large number of patients are exposed to unnecessary ionizing radiation and may be immobilized unnecessarily, sometimes for lengthy periods of time. For these concerns and others (financial, resource allocation and utilization, etc.), the issue of radiographic assessment of asymptomatic patients following trauma has been raised. A number of investigators have proposed that asymptomatic patients do not require radiographic assessment of the cervical spine following trauma. (2,5-7,9,14,16)

Asymptomatic patients following trauma are defined as those patients who meet all of the following criteria:

- 1) Are neurologically normal. (These patients must be GCS 15 and must not have any of the following: a) disorientation to person, place, or time; b) inability to remember 3 objects at 5 minutes; c) delayed or inappropriate response to external stimuli; or d) any focal motor or sensory deficit.)
- 2) Are not intoxicated. (Patients should be considered intoxicated if they have: a) a recent history of intoxication or intoxicating ingestion; b) evidence of intoxication on clinical examination; or c) laboratory evidence for the presence of drugs which alter the level of alertness, including alcohol greater than 0.08 mg/dl.)
- 3) Do not have neck pain or midline tenderness. (Midline tenderness is present if the patient complains of pain on palpation of the posterior midline neck from the nuchal ridge to the first thoracic vertebra.)
- 4) Do not have an associated injury that is distracting to the patient. (Significant distracting injuries have been defined as: a) long bone fractures; b) visceral injuries requiring surgical

consultation; c) large lacerations, degloving, or crush injuries; d) large burns; and e) any other injury which might impair the patient's ability to participate in a general physical, mental, and neurological examination.)(5)

Based upon these criteria, approximately one third of trauma patients evaluated in emergency rooms or trauma centers are asymptomatic (range 14-58%). (2,5,7,9,14,16-18) Avoidance of radiograph assessment in this patient population will result in a decrease in unnecessary radiation exposure, less patient time immobilized and confined in an uncomfortable position, and a significant savings in both cost and resource utilization. (13)

The establishment of a treatment standard for a therapeutic intervention requires the existence of at least one randomized controlled study. However, a treatment standard for the utility of a diagnostic test can be established with evidence derived from well designed clinical studies which include a "diverse population using a 'gold standard'" reference test in a blinded evaluation appropriate for the diagnostic applications and enabling the assessment of sensitivity, specificity, positive and negative predictive values, and where applicable, likelihood ratios."(19) In assessing the role of the radiographic assessment of asymptomatic trauma patients, we may consider the clinical examination a diagnostic test. X-ray imaging studies of the cervical spine may be considered the "gold standard" in this circumstance, as we are attempting to ascertain whether or not the clinical examination can accurately predict the results of the radiographic assessment in a given population of patients. The population in question should be representative of the trauma population evaluated at any given emergency room or trauma center.

The literature reviewed included nine large cohort studies that included a representative trauma population, defined symptomatic and asymptomatic patients based upon the above criteria,

and reported the incidence of spinal injury in these groups of patients as detected by subsequent radiographic assessment alone or by imaging of the cervical spine supplemented by clinical follow-up.(2,5,6,9,14,16,17,18) All nine studies were judged to provide Class I evidence, allowing the establishment of a treatment standard. Numerous case series and retrospective cohort studies exist and provide corroborating Class II and Class III evidence. These investigations are summarized in Evidentiary Table format and will be briefly discussed below.

The largest study addressing this issue encompassed 34,069 patients evaluated at 21 emergency rooms across the United States.(5) All patients were studied with standard three-view cervical radiography supplemented with CT, MRI, or other studies as needed. Eight hundred and eighteen patients were found to have spinal injuries, 576 of which were felt to be clinically significant. Two patients of the 576 were prospectively assigned to the “asymptomatic” group. One patient had a probable injury at C2 which was not treated due to patient refusal. Clinical follow-up in this patient revealed no sequela. The other patient had a laminar fracture of C6. He subsequently developed paresthesias in the arm and underwent surgery. Taking the worst-case scenario, and assuming that both of these patients were truly asymptomatic (the second patient later developed paresthesias), and the injuries were truly significant (the first patient’s injury was probably not significant given his subsequent clinical course), the negative predictive value of an asymptomatic examination was 99.9%. In contrast, the positive predictive value of a “symptomatic” examination was 1.9%. (5)

Bayless et al in 1989 studied a consecutive series of 228 patients who suffered “significant blunt head injury”.(2) Patients were classified as symptomatic or asymptomatic upon admission to the hospital. All patients were observed for at least 24 hours in the hospital and were assessed

with at least a three-view cervical spine x-ray series. A chart review two years post-admission was performed and any subsequent hospital visits were noted. Two hundred and eleven of the 228 patients were judged to have adequate three-view cervical spine series. Of these 122 were judged asymptomatic and none had a significant spine injury. (2) Hoffman et al performed a prospective study of 974 consecutive blunt trauma patients evaluated at a university emergency room.(6) All patients underwent at least a three-view cervical spine x-ray series supplemented with CT, oblique views, or flexion/extension views based upon physician judgement. Quality assurance logs, risk management records, and hospital charts from subsequent admissions were also reviewed. Of the 974 patients included in the study, 353 were judged “asymptomatic” and none were identified to have had a cervical spine injury. (6)

Kriepke and colleagues performed a prospective study involving 860 consecutive acute trauma patients who presented to a level one trauma center.(9) All patients underwent five-view cervical radiography supplemented with CT and/or flexion/extension views when required. Three hundred and twenty four patients were judged asymptomatic and none had a cervical spine injury detected on radiographic assessment. (9) Neifeld et al prospectively studied 886 trauma patients presenting to an urban emergency room.(14) All patients were studied with a five-view cervical spine series. Of 241 patients who were asymptomatic, none had a spine injury detected radiographically. (14)

Roberge et al in 1988, reported the results of a prospective study involving all patients who received a five-view cervical spine series while in an urban emergency room.(16) Four hundred sixty-seven patients were studied. One hundred and fifty-five were judged to be asymptomatic and none were found to have a spinal injury. (16) Ross et al prospectively studied 410 trauma patients admitted to a trauma center in 1992.(17) All patients underwent a three-view

cervical spine series supplemented as needed with CT, flexion/extension views, fluoroscopy and radionuclide bone scans. Of 196 patients judged asymptomatic, none had a cervical spine injury diagnosed with these imaging modalities.(17) Roth et al in 1994 prospectively studied 682 patients evaluated at a military hospital following blunt trauma.(19) All patients underwent a three-view cervical spine x-ray assessment. A subsequent chart review revealed no missed injuries (the hospital was the only military hospital in a radius of 2,500 miles) and 45% of patients were successfully contacted 30 to 150 days following the initial evaluation for additional clinical follow-up. Of 96 asymptomatic patients, none had a cervical spine injury.(18) Recently, Gonzales studied a series of 2176 patients evaluated in an emergency room following trauma.(4) One thousand, seven hundred sixty-eight were judged “asymptomatic,” although the criteria used were slightly different than those described previously. Three injuries were later detected in this group of 1768 patients, however two of these patients were not truly asymptomatic (one had a sternal fracture, multiple rib fractures and a splenic hematoma, the other had significant facial fractures), and the third patient’s injuries were radiographically occult. The third patient’s injuries were detected by CT and were treated with a collar. (4)

In addition to these studies which provide Class I evidence, other studies have been reported which provide corroborating Class II and Class III evidence germane to this issue. (1,10,12,13) For example, Mirvis studied 241 patients with a history of blunt trauma with cervical spine x-rays supplemented with CT. Aside from a single nondisplaced transverse process fracture of C7 (which was not seen on conventional radiographs), no patient of 138 patients deemed “asymptomatic” had a significant spinal injury. (13) McNamara performed a retrospective review of 286 trauma patients evaluated in an urban emergency room. One hundred seventy-eight patients were characterized as asymptomatic. Not one of these 178 patients had a

spinal injury detected with subsequent radiographic assessment. (12) Bachulis surveyed a prospectively acquired database of 4,941 consecutive patients evaluated after blunt traumatic injury.(1) One thousand eight hundred and twenty three patients underwent radiographic assessment of the cervical spine. Ninety-four patients were found to have a spinal injury. All patients with spinal injuries were symptomatic. No asymptomatic patient had a spinal injury. (1) Lindsey reviewed 2,283 consecutive trauma patients and determined that no patient with a spinal injury was asymptomatic (10)

Case reports exist describing asymptomatic patients who have harbored potentially unstable spinal injuries.(11,20) For example, Woodring et al reviewed 216 patients who had cervical spine injuries diagnosed with CT.(20) They report that 11 of these 216 patients were not reported to be symptomatic in the medical record. It is unclear why these 11 patients were subjected to CT evaluation of the cervical spine if they were asymptomatic. Woodring also reported a 61% false negative rate with the use of plain films in this population (20) Woodring encouraged the liberal use of CT based upon the mechanism of injury. No objective definitive conclusion can be drawn from this retrospective case series of a very select patient population. One must question the utility of radiographic assessment of any patient given a 61% false negative rate.

Other authors have refuted the contention that “mechanism of injury” is a reliable predictor of cervical spine injury. (8,17) Mace reported the case of a 51-year-old man who was awake and alert, neurologically intact, who had no complaints of neck pain, nor other associated distracting injury who was found to have an unstable fracture of C2.(11) It is important to note, however, that the patient had no history of trauma but was being evaluated for a sore throat. Cervical spine x-rays were obtained to rule out a peritonsillar abscess. (11) From these reports, it

is clear that potentially unstable spinal injuries may exist in asymptomatic patients (even those presenting with sore throats). However, these injuries are so rare that they do not appear in even the largest population based studies. The experience of Davis et al is illustrative.(3) They described the etiology of 34 missed cervical spine injuries in a series of 32,117 trauma patients evaluated at a group of emergency rooms servicing San Diego county. No missed injury occurred in an asymptomatic patient in their study. (3)

## **SUMMARY**

Clinical investigations which provide Class I evidence involving nearly 40,000 patients, plus Class II and III evidence studies involving over 5000 patients, convincingly demonstrate that asymptomatic patients do not require radiographic assessment of the cervical spine following trauma. The combined negative predictive value of cervical spine x-ray assessment of “asymptomatic” patients for a significant cervical spine injury is virtually 100%.(2,4-6,9,14,16-18)

In contrast, the reported incidence of cervical spine injuries in the symptomatic patient ranged from 1.9% to 6.2% in these Class I evidence studies. Symptomatic patients require radiographic study to rule out the presence of a traumatic cervical spinal injury prior to the discontinuation of cervical spine immobilization.(2,4-6,9,14,16-18) The type and extent of radiographic assessment of symptomatic patients following trauma is the topic of a separate review.

## **KEY ISSUES FOR FUTURE INVESTIGATION**

None.

## EVIDENTIARY TABLES

First Author Reference	Description of Study	Data Class	Conclusions
<p>Hoffman <i>New England Journal of Medicine</i> 343:94-99, 2000</p>	<p>Prospective study of 34,069 patients 4309 asymptomatic 2 had “clinically significant injuries” All patients radiographed Negative predictive value of 99.9% Positive predictive value 1.9% Note: One of two “missed injuries” did not really have a “significant injury,” as he was untreated and had no sequela with clinical follow-up. The other patient developed paresthesias in his arm and was found to have a laminar fracture of C6.</p>	<p>Class I</p>	<p>Radiographs not necessary in asymptomatic patients</p>
<p>Gonzales et al, <i>Journal of the American College of Surgeons</i> 189: 152-157, 1999</p>	<p>2176 patients prospectively studied with screening examination and radiographs. One injury was detected by plain films in an otherwise asymptomatic patient, however plain films missed 13 injuries overall.</p>	<p>Class I</p>	<p>Plain film radiography does not improve sensitivity (compared to the physical examination) for the detection of cervical spine injury in asymptomatic patients.</p>
<p>Roth, <i>Arch Surg</i> 129: 643-645, 1994</p>	<p>Prospective study of 682 patients admitted to ED with trauma 96 were asymptomatic, none had injury Overall incidence of injury was 2% All patients radiographed Follow-up clinical visit between 30-150 days post injury, achieved in 43% Negative predictive value of asymptomatic exam: 100% Positive predictive value of symptomatic exam: 2.7%</p>	<p>Class I</p>	<p>Radiographs likely not necessary in asymptomatic patients</p>
<p>Lindsey <i>Southern Medical Journal</i> 86:1253-1375, 1993</p>	<p>1,686 patients studied retrospectively, 597 patients studied prospectively A total of 49 patients with cervical spine injuries were identified (overall incidence 2.1%) No patient with an injury was asymptomatic</p>	<p>Class III The total number of symptomatic and asymptomatic patients are not reported, precluding the calculation of negative or positive predictive values.</p>	<p>Asymptomatic patients do not require radiographic images</p>

<p>Hoffman <i>Ann Emerg Med</i> 21: 1454-1460, 1992</p>	<p>974 blunt trauma patients prospectively studied Overall Incidence of cervical spine injury was 2.8% Of 353 alert, asymptomatic patients, none had a significant spine injury Follow-up: Radiographs negative in all 353 Charts, quality assurance logs, and risk management records reviewed with three month follow-up Negative predictive value of asymptomatic exam: 100% Positive predictive value of symptomatic exam: 4.5%</p>	<p>Class I</p>	<p>Asymptomatic patients do not require cervical spine films</p>
<p>Ross <i>British Journal of Accident Surgery</i> 23: 317-319, 1992</p>	<p>Prospective study of 410 patients seen at trauma center 196 patients had asymptomatic examination, none had injury All patients studied with plain films, CT's used as necessary Negative predictive value: 100% Positive predictive value: 6.1%</p>	<p>Class I</p>	<p>Radiography not mandatory for asymptomatic patients Main point of paper was that mechanism of injury is not a valuable predictor of injury.</p>
<p>McNamara: <i>Journal of Emergency Medicine</i> 8:177-182, 1990</p>	<p>Retrospective review of 286 patients judged to be "high risk" by mechanism of injury 178 were asymptomatic, none had cervical spine injury 108 were symptomatic, 5 had cervical spine injury Chart follow-up performed to determine incidence of injury Negative predictive value for asymptomatic exam was 100% Positive predictive value for symptomatic exam was 4.9%</p>	<p>Class III Many patients excluded due to poor documentation, select population follow-up inadequate (films not done on everyone, no delayed chart review)</p>	<p>Cervical spine radiographs not necessary in asymptomatic patients</p>
<p>Bayless <i>Am J Emer Med</i> 7:139-142, 1989</p>	<p>Series of 228 patients, 211 with complete studies Overall incidence of significant spinal injury was 1.7% Of 122 alert, asymptomatic patients, none had a significant injury Follow-up: Radiographs negative in all 122 Charts reviewed for Any subsequent referable visits within 2 years Negative predictive value of asymptomatic Exam: 100% Positive predictive value of symptomatic examination: 3%</p>	<p>Class I</p>	<p>Asymptomatic patients do not require cervical spine films</p>

<p>Kreipke <i>Journal of Trauma</i> 29:1438-1439, 1989.</p>	<p>Prospective study of 860 patients presenting to trauma center 324 asymptomatic, none had injury All patients radiographed Negative predictive value of asymptomatic exam: 100% Positive predictive value of symptomatic exam: 4%</p>	<p>Class I</p>	<p>Radiographs not necessary in asymptomatic patients</p>
<p>Mirvis <i>Radiology</i> 170: 831-834, 1989</p>	<p>408 patients studied with standard radiographs and CT Total population seen was 4135 patients 241 patients underwent CT because of “suspicious” radiographs, failure to visualize extremes of C-spine, or for confirmation of known fracture. Of these 241, 138 patients were clinically asymptomatic CT served as “gold standard” None of these 138 patients had a clinically relevant injury (although one had a nondisplaced C7 transverse process fracture which was treated with a collar). Negative predictive value of asymptomatic exam 99.3-100% Positive predictive value of symptomatic exam 12.6%</p>	<p>Class II, select population gold standard may be false endpoint</p>	<p>Clinically relevant cervical spine injury is extremely uncommon in asymptomatic patients. Radiographs may be unnecessary.</p>
<p>Neifeld <i>Journal of Emergency Medicine</i> 6:203-207, 1988</p>	<p>Prospective study of 886 patients 244 asymptomatic patients, none had injury All patients radiographed Negative predictive value 100% Positive predictive value: 6.2%</p>	<p>Class I</p>	<p>Asymptomatic patients do not require radiographs.</p>
<p>Roberge <i>Journal of Trauma</i> 28: 784-788, 1988.</p>	<p>Prospective study involving 467 trauma patients 155 asymptomatic patients were asymptomatic, none had a spine injury 312 were symptomatic, 8 had spine injuries All patients “scheduled to follow-up” in surgery clinic, authors state that no missed injuries have been identified Negative predictive value of asymptomatic exam: 100% Positive predictive value of symptomatic exam: 2.5%</p>	<p>Class I</p>	<p>Asymptomatic patients do not require radiographs</p>
<p>Bachulis et al. <i>American Journal of Surgery</i> 153:473-478, 1987</p>	<p>1823 of 4941 trauma patients studied with plain radiographs. 94 patients found to have injuries. All were symptomatic. No asymptomatic patient had a radiographically detectable injury.</p>	<p>Class III</p>	<p>Asymptomatic patients do not require radiographs.</p>

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## **RADIOGRAPHIC ASSESSMENT OF THE CERVICAL SPINE IN SYMPTOMATIC TRAUMA PATIENTS**

### **RECOMMENDATIONS**

Standards: A three view cervical spine series (AP, lateral, and odontoid views) is recommended for radiographic evaluation of the cervical spine in patients who are symptomatic following traumatic injury. This should be supplemented with computed tomography to further define areas that are suspicious or not well visualized on the plain cervical x-rays.

Guidelines: There is insufficient evidence to support treatment guidelines.

Options:

- It is recommended that cervical spine immobilization in awake patients with neck pain or tenderness and normal cervical spine x-rays (including supplemental CT as necessary) be discontinued following either:

- a) Normal and adequate dynamic flexion/extension radiographs; or
- b) Normal MRI study obtained within 48 hours of injury.

- Cervical spine immobilization of obtunded patients with normal cervical spine x-rays (including supplemental CT as necessary) may be discontinued:

- a) Following dynamic flexion/extension studies performed under fluoroscopic guidance; or
- b) Following a normal MRI study obtained within 48 hours of injury; or
- c) At the discretion of the treating physician.

### **RATIONALE**

Trauma patients who are symptomatic, that is, complain of neck pain, have cervical spine tenderness, or have symptoms or signs of a neurological deficit associated with the cervical spine,

and trauma patients who cannot be assessed for symptoms or signs (those who are unconscious, uncooperative or incoherent, intoxicated, or who have associated traumatic injuries that distract from their assessment) require radiographic study of the cervical spine prior to the discontinuation of cervical spine immobilization. Many authors have proposed strategies and imaging techniques to accomplish x-ray clearance of the cervical spine after trauma, particularly in the symptomatic or the obtunded patient. One, three, and five view static cervical spine x-rays, computed tomography (CT), magnetic resonance imaging (MRI), bone scans, flexion/extension radiographs, dynamic fluoroscopy with or without somatosensory evoked potential monitoring, and other studies have all been described as useful for the determination of spinal injury and potential spinal instability following traumatic injury. (1-9,11-17,19-24,26-28,30-39,41-43,45,46,47-54,56,57,59-73) The purpose of this review is to determine the optimal radiographic assessment strategy necessary and sufficient to exclude a significant cervical spine injury in the symptomatic trauma patient.

## **SEARCH CRITERIA**

A National Library of Medicine computerized literature search from 1966 to 2001 was performed using Medline and keywords “spinal cord injury”, “spinal fractures”, or “spinal injuries”. This resulted in 7994 matches. Combination with the keyword “cervical” resulted in 1844 matches. These references were limited to human studies and the English language, resulting in 1268 articles. Combination with the keywords “clearance,” “diagnosis,” or “radiography” yielded 184 matches. The titles and abstracts of these 184 articles were reviewed. All papers focusing on clinical decision-making with regard to the diagnosis of cervical spine injuries in adult victims of trauma were included. Additional references were culled from the

reference lists of the remaining papers. The 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. The practice parameters and reference list developed by the EAST practice parameter workgroup for cervical spine clearance (56) was reviewed as was the reference list developed by the NEXUS (National Emergency X-radiography Utilization Study) group (31, 33). Seventy-three total references form the basis for this review.

Twenty-one manuscripts were identified which specifically provided evidence germane to the topic of this guideline. Four studies provided Class I evidence, seven provided Class II evidence, and ten were individual case series and provided Class III evidence. These 21 manuscripts are summarized in Evidentiary Table format.

## **SCIENTIFIC FOUNDATION**

Patients who are asymptomatic with respect to a potential cervical spinal injury following acute trauma do not require radiographic assessment in order to rule out a significant injury to the cervical spine (see Guideline on Radiographic Assessment of Asymptomatic Patients). Radiographic studies do not increase the sensitivity or specificity of the clinical examination in this specifically defined population of patients.(31,33) There is however, a 2% to 6% incidence of significant cervical spine injury in the symptomatic patient population following acute trauma.(4,31-33,42,53,61,62) These patients require radiographic assessment to exclude cervical spinal injury prior to the discontinuation of cervical spine immobilization. The most significant consequence of premature discontinuation of cervical spine immobilization is neurological injury. Prolonged immobilization, however, is associated with morbidity as well. Decubitus ulcers, increased cerebrospinal fluid pressure, pain and pulmonary complications have all been described

with prolonged immobilization of the cervical spine (18,44,58). For these reasons, a diagnostic algorithm that is highly sensitive and specific for the occurrence of a significant cervical spine injury, and that can be applied in an expeditious fashion is desired.

The single most common cause of missed cervical spine injury appears to be failure to adequately visualize the region of injury. This can be caused by failure to obtain radiographs, or by making judgments on technically suboptimal films. This occurs most commonly at the extremes of the cervical spine, the occiput to C2 and at the C7-T1 levels.(17,25,59) Davis described 32,117 acute trauma patients.(17) Cervical spine injuries were missed in 34 symptomatic patients. Twenty-three of these thirty-four symptomatic patients either did not have radiographs or had inadequate radiographs which did not include the region of injury. Eight patients had adequate x-ray studies that were misread by the treating physician. Only one patient had a missed injury that was undetectable on technically adequate films, even after retrospective review. The error in two patients with missed injuries was not described.(17) Davis' review and those of other investigators, confirm that it is uncommon to miss cervical spine injuries with adequate plain radiographic assessment of the occiput through T1.(1,6,9,16,17,24,43,47)

The most prevalent initial x-ray assessment of the symptomatic or obtunded patient is the three-view cervical spine series. When adequate visualization of the entire cervical spine is achieved from occiput to T1, the negative predictive value of a normal three-view cervical spine series has been reported to range from 93% to 98% in several Class I studies (1,6,47), and from 85% to 100% in class II and III studies (9,16,24,43). Although the negative predictive value of the three-view cervical spine x-ray series is quite high, the sensitivity of the three-view series is less impressive. The same Class I series referenced above report sensitivity rates for the three view cervical spine series of 84%, 62.5% and 83% respectively (1,6,47). In the best-case clinical

scenario, assuming the highest values for negative predictive value and sensitivity, approximately 98% of patients with a normal three-view cervical spine x-ray series will have a truly normal cervical spine. This same data suggests that the three-view cervical spine series will also be normal in 15% to 17% of patients who have cervical spine injuries. If we assume a 6% incidence of spinal injury in a high-risk population (the head injured multi-trauma patient, for example), then an adequate three-view cervical spine series alone would be expected to correctly identify 5 out of 6 spinal injuries in a group of 100 patients, and correctly identify 94 of 94 non-injured patients. One patient of the 100 with an injured spine would have cervical radiographs interpreted as normal. The addition of oblique views (for a five-view series) does not appear to increase the overall sensitivity of the examination.(24) Oblique views may be useful in lieu of a swimmer's view to visualize C7-T1.(36) Holliman and colleagues have questioned the utility of the AP cervical view, and argue it is not an important addition to the assessment of the acute trauma patient.(34) The data they present is Class III evidence and has not been verified by others.(34) There exist several reports that confirm that the lateral x-ray view alone will miss a substantial portion of cervical spine injuries depicted in a three-view series.(14,26,65)

In order to increase the sensitivity of the radiographic assessment of the cervical spine in trauma patients, multiple authors have described experiences with CT and MRI imaging in the acute setting. Several have reported greater sensitivity using CT to view areas not well visualized on plain films, typically the craniocervical and cervicothoracic junctions, or areas identified as suspicious on plain cervical spine x-rays.(6,9,24,48,67,68) In a small Class I study of 58 patients, Berne et al reported that helical CT of the entire cervical spine identified all clinically significant injuries in a series of patients assessed with plain films, CT, and MRI who were followed clinically for subsequent events.(6) Two injuries were missed, however neither required

any treatment. Berne et al report a negative predictive value of 95% for CT for all spinal injuries and a negative predictive value of 100% for unstable injuries.(6) Other authors report 100% sensitivity for the detection of injuries with CT limited to areas poorly visualized or identified as suspicious on plain films.(24,48,67,68) However, all studies cited provide Class II and III evidence, and most suffer from a common flaw; they treat CT as the “gold standard” for the detection of injury. While they suggest that the addition of CT imaging increases diagnostic sensitivity, the use of CT data as the “gold standard” represents a false endpoint for the true variable of clinically relevant spinal injury.

Although the incidence of significant spinal injury with a normal cervical spine series supplemented with CT is extremely low, missed injuries have been reported. Brohi reported a missed C6-C7 facet dislocation in a patient with persistent neck pain who was studied with plain films and a CT occiput through C7-T1.(11) Sweeney reported an autopsy series of three patients who died of traumatic injuries and were found to have spinal injuries undetected by plain films supplemented with CT through the region of injury.(66) Thin-cut CT images through the entire spine may increase sensitivity somewhat, (6,55) but no direct comparison between the two imaging strategies in an appropriate patient population has been performed to date.

MRI has been used to evaluate patients at risk for acute spinal injury. Results have been mixed. Benzel and colleagues studied 174 symptomatic patients with low field MRI within 48 hours of injury. Soft tissue abnormalities were visualized on MR in 62 patients. Two of these 62 were felt to have unstable injuries. Both had plain film and CT abnormalities which revealed the injuries. The 60 patients with MRI abnormalities not felt to be significant were immobilized for 1-3 months and then studied with flexion and extension radiographs. Not one was found to have an

unstable injury. Patients with “negative” MRI studies were cleared of spinal precautions and no adverse events were reported.(5) D’Alise and colleagues reported their results of a Class III evidence study of MRI in 121 obtunded patients. Ninety patients had normal studies and were cleared. Follow-up flexion/extension radiographs did not reveal a single abnormality in this group. Thirty-one patients had injuries to soft tissues of the cervical spine identified by MRI not detected by plain radiographs. Eight of these patients ultimately required surgery.(15) Katzberg and colleagues and White et al have also described increased sensitivity of MRI for the detection of soft tissue injuries of the cervical spine following trauma.(39,72)

These studies demonstrate that MRI abnormalities are visualized in a substantial number of cervical spine studies performed on patients following trauma. It is impossible to determine the true incidence of clinically significant ligamentous injury in this group examined with MR, as all patients with MRI abnormalities were treated with immobilization. The incidence of significant cervical spine injury in previous studies looking at similar patient populations is between 2% and 6%, yet the incidence of MR imaging abnormalities is reported to be between 25% and 40%. MRI appears to “overcall” significant injury. It should be noted that the optimal time frame for MRI assessment of the cervical spine is limited. MRI studies are preferred within the first 48 hours after injury. (5,15,21,39,72) Even then some injuries are poorly visualized. Emery and colleagues used MRI to study 37 patients with known cervical spinal injury and found that MRI missed ligamentous injury in two of 19 patients known to have ligamentous injury (abnormal flexion/extension films or surgical confirmation). These images were obtained an average of 10.8 days following injury (21). Klein et al, comparing CT and MRI images obtained from the same patients, demonstrated that MRI was not as effective for the recognition of bony abnormalities as was CT (41). It appears that MRI when used early after trauma in conjunction with plain

radiographs and CT is exquisitely sensitive for the detection of soft tissue abnormalities of the cervical spine. The importance of these findings for the majority of patients is, however, unknown.

Flexion extension radiographs have been used to rule out ligamentous injury of the cervical spine. In the awake patient, this maneuver is generally considered safe and effective. Numerous series have used flexion/extension films as the gold standard for the exclusion of ligamentous injury in this population and no serious adverse events have been reported (1,3,5,10,11,15). Brady et al used dynamic flexion-extension spine films to study 451 awake patients with blunt trauma evaluated in an urban emergency room.(10) Flexion/extension views detected abnormalities in 5 of 372 patients in whom static plain cervical spine films were felt to be normal. None of these patients required “invasive stabilization,” indicating that the abnormal examinations may have been false positives.(10) It should be noted, however, that false negative examinations also occur, although infrequently. Lewis et al reported one false negative examination in a series of 141 patients studied with dynamic flexion/extension films. These authors report the negative predictive value for the combination of plain films and flexion/extension films to be over 99%. (43)

The obtunded patient is not able to actively flex or extend the neck for dynamic radiographic evaluation. Dynamic fluoroscopy has been used to clear the cervical spine in these patients and results of several series are available (16,64). Ajani et al, reported an unstable cervical spine injury detected by flexion/extension radiographs in a patient with normal plain films and CT (one of 100 patients studied).(1) Davis et al used dynamic fluoroscopy to study 116 obtunded patients who had normal cervical radiographs. Only one patient was found to have an

injury not visualized on plain films or CT. The significance of this injury, a 2 mm subluxation in a patient who was treated in a collar and subsequently lost to follow-up, is questionable.(16) Sees et al studied 20 obtunded patients with normal three-view cervical spine series. They performed bedside flexion/extension under fluoroscopy and found 1 patient with C4-5 subluxation due to a facet injury not appreciated on plain films but later confirmed with CT.(64) It should be noted that 30% of the patients in the Sees et al series could not be cleared because of difficulty visualizing the lower cervical spine, whereas Davis et al, using radiology staff in the fluoroscopy suite, were able to visualize the entire spine in virtually all patients.(16,64)

Because of the high negative predictive value of plain films and supplemental CT, application of MRI or flexion/extension fluoroscopy for clearance of the cervical spine is probably not indicated for every obtunded patient. Use of these modalities should be guided by clinical judgment based on patient history and physical examination. Subgroups of obtunded trauma patients exist with a very low likelihood of cervical spine injury, and exhaustive study is not indicated for these patients. Hanson et al found that the incidence of cervical spine injury in a series of 3684 patients without high-risk criteria was 0.2%, and that all of these injuries were detected by plain radiographs supplemented with CT for poorly visualized or suspicious areas.(29) The high risk criteria cited were: a high speed motor vehicle accident (>35 mph); an automobile crash with a death at the scene; a fall from greater than 10 ft; a significant traumatic closed head injury or traumatic intracranial hemorrhage; neurological signs or symptoms referable to the cervical spine; or pelvis or multiple extremity fractures.(29) In support of this issue, Kaups and Davis did not identify a single cervical spine injury in a group of 215 victims of gunshot wounds to the head.(40) Similarly, Patton et al used MRI and flexion/extension

fluoroscopy as a supplement to x-rays to assess the cervical spines of a series of patients with isolated head injuries suffered as a result of assault.(57) They found no undiagnosed injuries.

## **SUMMARY**

In summary, no single radiographic study can adequately rule out cervical spinal injury in all symptomatic patients. A three-view spine cervical spine series supplemented with CT through areas difficult to visualize and “suspicious” areas will detect the vast majority of spinal injuries. This combination of studies represents the minimum required for clearance of the cervical spine in the symptomatic patient. The negative predictive value of this combination of studies is reported to be between 99% and 100% in several Class II and III evidence studies. (9,11,24,48,67,68)

In the awake patient, dynamic flexion/extension views (with at least 30° excursion in each direction) are safe and effective for detecting the majority of “occult” cervical spine injuries not identified on plain x-rays. The negative predictive value of a normal three view series and flexion/extension views exceeds 99%.(43) Patients who are unable to cooperate with active flexion/extension radiographs due to pain or muscle spasm may be maintained in a cervical collar until they are able to cooperate, or may be studied with MRI. A negative MRI within the first 48 hours of injury in addition to normal radiographs and supplemental CT appear to be sufficient for the clearance of the cervical spine. The significance of a positive MR study is currently unclear. It is suggested that cervical immobilization be continued in these patients until delayed flexion/extension views can be obtained.

In the obtunded patient with a normal three-view x-ray series and appropriate CT of the cervical spine, the incidence of significant spine injury is less than 1%. Based upon mechanism of injury and clinical judgement, the cervical spine in selected patients may be considered cleared

without further study. In the remainder of cases, flexion/extension performed under fluoroscopic visualization appears to be safe and effective for ruling out significant ligamentous injury, with a reported negative predictive value of over 99% (16). Because the incidence of occult injury diagnosed with dynamic flexion/extension fluoroscopy in the setting of normal plain cervical spine x-rays and CT images is low, it is probably most efficient for these procedures to be performed by staff in the department of radiology, although variances in local experience should be respected. MRI represents another option for clearance of the spine in this patient population, and a negative MRI within 48 hours of injury appears to effectively eliminate the likelihood of a significant ligamentous injury. However, MRI evaluation will result in a large number of false positive examinations, and the consequences of prolonged unnecessary immobilization in the obtunded patient are not insignificant.(18,44,58)

## **KEY ISSUES FOR FUTURE INVESTIGATION**

The significance of positive MRI findings following cervical trauma should be evaluated using flexion/extension radiographs and clinical follow-up as the gold standard.

The incidence of abnormal findings on flexion/extension fluoroscopic studies in obtunded patients should be evaluated in a prospective fashion with appropriate clinical follow-up.

A comparison between the three-view cervical spine series supplemented with selective CT through poorly visualized or suspicious areas, and CT of the entire cervical spine should be accomplished in a prospective fashion.

## EVIDENTIARY TABLES

First Author Reference	Description of Study	Data Class	Conclusions
Banit, <i>Journal of Trauma</i> 49: 450-456, 2000.	Combined retrospective/prospective study 4,460 patients evaluated 2,217 felt to require radiographs 6 month clinical follow-up and subsequent CT/MRI used as gold standard for plain radiographs (authors claim no missed injuries, credible claim) 5 view series used in all patients	Class III	In symptomatic patients, sensitivity of plain films was 88% (68/77) before institution of protocol and 84% (71/84) False positives not given Protocol had sensitivity of 100% and included use delayed exam of patients with tenderness/pain with flexion/extension radiographs (false positives not given)
Berne, <i>Journal of Trauma</i> 47:896-903, 1999	Prospective study of select population of patients (unevaluable, multi-trauma, having CT done for another reason) 58 patients, all underwent 3 view series followed by helical CT of entire spine “Suspicious but not diagnostic” examinations were evaluated with MRI, flexion/extension views, or repeated clinical examination	Class I	20/58 (34%) had injuries detected Plain films identified 12 for a sensitivity of 60%, positive predictive value of 100%, negative predictive value of 85% CT missed 2 injuries (both “stable”) for sensitivity of 90%, specificity 100%, positive predictive value of 100%, negative predictive value of 95%.
D’Alise, <i>J Neurosurg</i> , 91 (Spine 1):54-59, 1999	121 obtunded patients with normal X-rays studied with MRI CT used to study areas of MRI abnormality All patients with negative MRI underwent flexion/extension imaging immediately upon “clearance”	Class III	31/121 (26%) had injuries detected on MRI 90/121 (74.4%) had no injury and were cleared (verified with flex/ext) 8 patients determined to have spinal instability (clinical, CT, etc.) No flex/ext performed on patients with abnormal MRI Cannot determine significance of MRI findings in 23/31 patients Seems to indicate that negative MRI equi-valent to negative Flex/ext.
Katzberg, <i>Radiology</i> 213: 203-212, 1999.	Prospective study of 199 patients who underwent MRI in addition to standard radiographic study. Half of patients were selected because of suspected high probability of injury.	Class III	MRI detected injuries in a higher fraction of these patients than did conventional radiographs and CT. Significance of these injuries? Gold standard?
Klein, <i>Spine</i> , 24:771-774, 1999.	Retrospective review of 32 patients with 75 known spine fractures Blinded review of MRIs by radiologists	Class II, select population	Posterior/Anterior element injuries: Sensitivity: 11.5%/36.7% Specificity 97.0%/98% Positive predictive value 83%/91.2% Negative predictive value 46%/64% MRI NOT good for evaluating bony pathology

Tan, <i>Journal of Spinal Disorders</i> 12: 472-476, 1999	Retrospective review of 360 patients treated for blunt injury who underwent 3 view C-spine films supplemented with CT because of nonvisualization of C7-T1. CT findings considered gold standard for detection of fracture	Class III	11 injuries detected by CT which were not visible on plain films Sensitivity of inadequate plain films relative to CT for this purpose: 97%
White, <i>British J Radiology</i> :818-823, 1999	31 patients with known or suspected spine injury evaluated with MRI	Class III	Pre-vertebral hematoma picked up more often by MRI than by plain films (24/31 vs. 14/30) Suggests that sensitivity of plain films for prevertebral hematoma is 66%
Ajani, <i>Anaesth Intensive Care</i> 26: 487-491, 1998	100 consecutive patients studied prospectively All radiographed (3 view) Follow-up clinical examination, CT, MRI, and flexion extension views performed	Class I	1/6 injuries missed by X-Ray (sensitivity 84%), 7/12 X-Ray abnormalities found to be insignificant Positive predictive value 45% Negative Predictive value 98.9% 1 missed injury detected by flexion/extension views
Sees, <i>J Trauma</i> 45: 768-771, 1998.	20 patients underwent bedside flex/ext under fluoro after 3 view C-spine films normal	Class III (for fluoro) Class II for 3 view c-spine with fluoro as gold standard	One patient found to have subluxation No gold standard for flex/ext Sensitivity of plain films with flex/ext as gold standard 95%
Benzel, <i>Journal of Neurosurgery</i> 85: 824-829, 1996	174 patients suspected of having cervical spine injury (equivocal plain films/CT or positive symptoms) Underwent MRI CT's obtained through area of injury defined by MRI	Class III	36% (62/174) had MRI evidence of injury 61/62 managed with immobilization for 1-2 months All patients with negative MRIs were cleared, no instances of late instability Negative predictive value of MRI 100% Positive predictive value ? Specificity?
Davis JW, <i>Journal of Trauma</i> 39:435-438, 1995.	116 patients with GCS<13 and normal radiographs evaluated with flexion/extension views under fluoro	Class I for plain films vs. flex/ext as gold standard Class III (follow-up questionable ) for flex/ext ruling out injury.	113 patients had no abnormality detected 2 patients had "stable" facet fractures 1 patient had 2mm of subluxation and was treated in a collar (no follow up on this patient) No patient had referable neurological injury with clinical follow-up Decubiti ulcers found under collars in 44% of patients with mean collar time of 6 days NPV FE: 100%
Holliman, <i>J Emerg Med</i> 9:421-425, 1995	Retrospective series of 148 patients with known spine injuries. Lateral and odontoid films retrospectively reviewed separately from AP film. 60 sets of film available for review.	Class III	In these 60 patients, all injuries noted on AP films were also detectable on lateral or odontoid films.

Tehranzadeh, <i>Skeletal Radiol</i> 23:349-352, 1994	Retrospective review of 100 patients with blunt injury and non visualized C7-T1 on plain films CT findings considered gold standard	Class III	3 patients found to have injuries on CT not visualized by plain films Sensitivity of inadequate plain films: 97%
Borock, <i>Journal of Trauma</i> 31: 1001-1006, 1991	Used CT to evaluate cervical spine in 179 patients who were symptomatic with normal films (2), whose entire cervical spine could not be visualized (123) or who had equivocal (13) or abnormal (41) x-rays. Plain film sensitivity calculated using CT as gold standard, authors claim no missed injuries	Class II, possible false end-point	39/54 X-Ray abnormalities were verified with CT (Positive predictive value of 72%) X-rays missed both injuries in symptomatic patients and 1 C7 transverse process fracture (Negative predictive value of 97.6%)
Cohn, <i>Journal of Trauma</i> 31: 570-574, 1991	60 patients prospectively studied with lateral film in ED Full radiographic work-up (3 or 5 view) followed Results of lateral view to full series compared	Class II, possible false end-point (Class I if used only as comparison)	Lateral view missed 3/7 total injuries Lateral view positive predictive value 100% Negative predictive value 94% Sensitivity 57%
Lewis, <i>Annals of Emergency Medicine</i> 20:117-121, 1991.	Retrospective review of 141 patients with active F/E films performed after three view series was normal	Class II for plain films vs. F/E as gold standard Class II for F/E views	11/141 F/E sets read as unstable, 4 of whom had normal 3 view series (remainder were questionable) All patients with instability had pain or were intoxicated One false negative F/E study For plain films vs F/E and plain films: Sens: 71%      99% Spec: 89%      89% NPV 93%      93% PPV 67%      99%
MacDonald RL, <i>J Trauma</i> 30: 392-397, 1990	775 patients Three views compared against gold standard of all other studies performed and clinical outcome	Class I	Three view series Sensitivity: 83% Specificity: 97% Positive Predictive Value: 81% Negative Predictive value: 98%
Emery, <i>Journal of Spinal Disorders</i> 2: 229-233, 1989	MRI used to study 37 patients with known spine injuries All patients also studied with some combination of plain films/CT/tomograms/surgical exploration/clinical follow-up	Class II, select population	19 patients found to have ligamentous injury MRI detected 17/19 MRI was negative in 18/18 patients with intact ligaments Positive predictive value 100% Negative predictive value 90% Sensitivity: 89.5%
Freemyer, <i>Annals of Emergency Med</i> , 18: 818-821, 1989.	Prospective study of "high risk" symptomatic patients imaged with 5 view series Radiographic review of three views compared to five views Computed tomography used as gold standard	Class II Select population	58 patients studied 68 injuries detected in 33 patients Sensitivity of three view series was 83% compared to tomography Addition of oblique views had no effect on overall sensitivity

Reid, <i>J Trauma</i> 27:980-986, 1987	253 patients with 274 spinal injuries evaluated 38 had delay in diagnosis Evaluated cause for delay in diagnosis	Class III	20 injuries missed despite adequate films Sensitivity 92.3%
Shaffer, <i>Ann Emerg Med</i> 10:508-513, 1981	Retrospective analysis of all C-spine injuries detected in community emergency rooms Evaluated reading of lateral film to reading of three view series (three view series gold standard) Authors claim no missed injuries with 3 view series	Class III	35 injuries detected 9 cases missed with lateral view alone Sensitivity of lateral view compared to 3 views: 74%

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## INITIAL CLOSED REDUCTION OF CERVICAL SPINAL FRACTURE-DISLOCATION INJURIES

### RECOMMENDATIONS

Standard: There is insufficient evidence to support treatment standards.

Guideline: There is insufficient evidence to support treatment guidelines.

Option:

- Early closed reduction of cervical spinal fracture-dislocation injuries with cranio-cervical traction is recommended for the restoration of anatomic alignment of the cervical spine in awake patients.
- Closed reduction in patients with an additional rostral injury is not recommended.
- Patients with cervical spinal fracture dislocation injuries who are not able to be examined during attempted closed reduction, or prior to open posterior reduction, should undergo MRI prior to attempted reduction. The presence of a significant disc herniation in this setting is a relative indication for a ventral decompression prior to reduction.
- MRI study of patients who fail attempts at closed reduction is recommended.
- Pre-reduction MRI performed in patients with cervical fracture dislocation injury will demonstrate disrupted or herniated intervertebral discs in one third to one half of patients with facet subluxation. These findings do not appear to significantly influence outcome following closed reduction in awake patients and therefore the utility of pre-reduction MRI in this circumstance is uncertain.

## **RATIONALE**

Spinal cord injury is frequently associated with traumatic cervical spine fractures and cervical facet dislocation injuries because of the narrowing of the spinal canal caused by displacement of fracture fragments or subluxation of one vertebra over another. Reduction of the deformity helps to restore the diameter of the bony canal and eliminates bony compression of the spinal cord due to the vertebral fracture and/or subluxation. Theoretically, early decompression of the spinal cord after injury may lead to improved neurological outcome. Several large series of patients describe excellent results with closed reduction of cervical fractures and facet subluxations. However, descriptive series using pre-reduction MRI have reported a high incidence of cervical disc herniation in the facet dislocation patient population. Furthermore, case reports and small series of patients who worsened neurologically following closed cervical spinal reduction exist. Several of these reports implicate ventral compression of the spinal cord by displaced disc material. The purpose of this qualitative review is to address the following issues:

1. Is closed reduction safe and effective for reducing cervical spinal deformity in patients with cervical fractures or unilateral or bilateral facet dislocation injuries?
2. Is a pre-reduction MRI essential for the management of these patients?

## **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”: spinal fracture, spinal injury and human. Approximately 12,300 citations were acquired. Non-English language citations were deleted. Searching this set of publications with the keyword “cervical” resulted in 2,154 matches. Further refining the search with the terms “reduction” or “fracture”

yielded 606 articles. Titles and abstracts of each of these 606 references were reviewed. Clinical series dealing with adult patients in the acute setting were selected. Case reports and case collections were included in the review but were not included in the Evidentiary Tables. Additional references were culled from the reference lists of the remaining papers. The 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. Finally, the tables of contents of the journal *Spine* were hand searched from 1993 through May, 2000. Thirty-five papers relevant to this topic were identified and form the basis of this guideline. The larger clinical series are summarized in Evidentiary Table format.

There were no randomized clinical trials, no prospective cohort studies and no case controlled studies. There was one historical cohort study comparing awake traction-reduction with manipulation under anesthesia (MUA). The remainder of the publications consisted of case series of patients with acute or subacute unilateral or bilateral cervical facet dislocation injuries treated with open or closed reduction. Several case reports and case series of patients who deteriorated following closed reduction were identified and are included. Several studies included pre- and post-reduction MRI findings.

## **SCIENTIFIC FOUNDATION**

Closed reduction of cervical spinal deformity due to facet dislocation by manipulation was first described by Walton in 1893.(35) Crutchfield introduced tongs for inline traction-reduction in 1933 (6), and similar techniques have been successfully used for traction-reduction of cervical deformity by a large number of authors.(1-5,11,12,16,21,23,27,29-31,34,37) Observations by Evans and Kleyn popularized reduction under anesthesia, although other authors condemned the procedure as potentially dangerous compared to cranio-cervical traction-

reduction. Manipulation under anesthesia (MUA) is still a frequently practiced technique, usually employed following failure of traction-reduction but occasionally used as a primary means of achieving reduction.(9,16) Only one cohort study has been performed comparing the two modalities. Lee et al found a higher rate of success and a lower complication rate with traction-reduction as opposed to MUA.(16) The significance of their results is questionable due to the historical cohort design of the study. Lee attributed the higher complication rate in the MUA group to the effects of anesthesia on perfusion of the injured spinal cord. It is possible, however, that advances in the pharmacological and medical management of spinal cord injured patients over the 10-year period of data accrual accounted for the improved results the authors noted in the traction-reduction group.(16) For this reason, the evidence provided by this study is considered to be Class III medical evidence.

Recent reports of neurological deterioration following closed or open posterior reduction of cervical fracture-dislocation injuries has led some authors to recommend the use of pre-reduction MRI to assess for ventral cord compromise due to traumatic disc disruption. Several investigators believe that disc disruption in association with facet fracture-dislocation increases the risk of spinal cord injury by disc material following reduction.(7,8,20,25) Other authors, however, have found no relationship between findings on pre-reduction MRI, neurological outcome, or findings on post-reduction MRI.(33) The nature of the injury predisposes a large percentage of patients with cervical facet dislocation injuries to have MRI evidence of disc material ventral to the spinal cord. Rizzolo et al found evidence of disc disruption/herniation in 55% of patients studied with pre-reduction MRI.(24) The clinical relevance of these findings has not been proven. The use of pre-reduction MRI may delay reduction of the spinal deformity and therefore may delay decompression of the compromised spinal cord. Pre-reduction MRI assessment requires the transport of a patient with a highly unstable cervical spinal fracture to the

MRI suite. Recent laboratory work, as well as Class III evidence, suggests that early reduction of fracture-dislocation injuries may improve neurological outcome.(2,10,16,20,26) If stabilization of the unstable cervical spine protects against additional injury to the cervical spinal cord, then the information gained by pre-reduction MRI must be of sufficient value to warrant the delay in treatment and the associated potential morbidity of transport.

The majority of clinical series reviewed were based on patient data accrued prior to the introduction of MRI. These combined series encompass over 1200 patients treated with closed reduction in the acute or subacute (days) period following injury. The success rate for restoration of anatomical alignment by closed reduction in these studies was approximately 80%. The reported permanent neurological complication rate was less than 1.0%.(3,5,9,11,12,15,16,20-21,23,27-31,34,36) Of the 11 patients reported to develop new permanent neurological deficits with attempted closed reduction, two had root injuries, (11,12) and two had ascending spinal cord deficits noted at the time of reduction.(3,30) Seven patients were noted to have decreased American Spinal Injury Association (ASIA) scores post-reduction, however neither the nature nor the cause of the new deficits in these patients was described.(16)

Transient neurological deterioration following closed reduction has been reported. In addition to the permanent deficits noted above, temporary deficits have been described in 20 additional patients of these 1200. These deficits reversed spontaneously, or improved following reduction of weight or following open reduction.(3,11,12,16,20,31) The causes of neurological deterioration associated with closed reduction in these and other series included over-distraction (3,21,30), failure to recognize a more rostral non-contiguous lesion (30,32), disc herniation (11), epidural hematoma (17), and spinal cord edema. (19,20)

A variety of authors have provided general suggestions on how craniocervical traction for closed reduction of cervical spinal fracture-dislocation injuries is best accomplished. (14,15,18,40) No study has been undertaken to determine the superiority of one method or technique over another. Tongs (Crutchfield or Gardner-Wells) or a halo ring are applied to the injury patient's head utilizing sterile technique and local anesthesia at the tong or pin insertion sites. Most contemporary descriptions incorporate the use of an MRI compatible halo ring as the cranial fixation device for four-point fixation of the cranium, better control of the head and neck if positioning and directional traction are needed (e.g., passive directional traction in positions of flexion or extension of the neck depending on the injury type), and to facilitate halo-ring vest application once closed reduction has been accomplished.(14,15,18,40)

Hadley et al suggest closed-reduction of acute cervical spinal fracture-dislocation injuries is best accomplished as part of the early overall medical management of the potential spinal cord injured patient in the ICU setting utilizing bedside fluoroscopy, with close monitoring of each patient's clinical and neurological status, as well as cardiac, respiratory, and hemodynamic parameters. Pain control and modest sedation-relaxation is provided utilizing short-acting intravenous agents that do not impair the patient's level of consciousness or alter their hemodynamic performance parameters. (14,18)

Cranial-cervical traction is typically arbitrarily initiated with the application of weight beginning with three pounds per superior injury level. A patient with an isolated C5-6 fracture dislocation injury using this scheme would begin treatment with an initial weight of 15 pounds (3 lbs. x 5 rostral vertebral levels). Caveats to the use of this initial weight suggestion include patients with fracture-dislocations involving C2 and above and patients with ankylosing spondylitis in whom very little weight, if any, may be needed to accomplish reduction. Judgment and experience must be utilized in this setting as some more proximal cervical spinal

injuries may be distraction injuries and will not require traction, and are perhaps best managed with realignment and compression.

Weight may be added as often as every ten to fifteen minutes as long as close clinical, neurological, and radiographic monitoring is reassessed throughout the process. No upper limit of weight has been described in the literature. In general, increasing weight is applied until closed reduction and realignment occurs or until patient complaints are great (increasing, intractable pain or a subjective worsening of neurologic status), the patient's neurological examination worsens, over-distraction occurs as noted on fluoroscopy or lateral cervical spine x-rays, when it is impractical to add further weight (no additional weight can be applied to the distraction device, no additional weight available, patient sliding upward in bed) or when the clinician's judgment is that closed reduction has failed.

Once closed reduction has been accomplished, or once the determination has been made that closed reduction has failed, it is recommended that the patient be immobilized to provide stabilization of the cervical spinal injury before transport to radiology for further assessment or to the operating room for surgical management.(14,18)

Herniated disc fragments causing compression of the cervical spinal cord at the level of facet dislocation have been described by several authors.(3,8,11,16,20,22,25,28,37) Eismont reported a series of 63 patients managed with closed traction-reduction followed by open reduction if closed reduction was unsuccessful.(8) One of these patients worsened following posterior open reduction and fusion. A herniated disc was found ventral to the cord on post-procedure myelography. Herniated discs were found in three other patients who failed closed reduction, and in two patients with static neurological deficits following fracture-dislocation reduction (one open, one closed). One of these patients deteriorated after subsequent anterior

cervical discectomy and fusion (ACDF). Eismont did not report his overall results with closed reduction. However, it is clear from his case descriptions that no awake patient experienced neurological deterioration as a result of attempted closed reduction.(8) Olerud described two patients found to have disc herniations on post-reduction MRI or CT myelography.(22) Both patients deteriorated following open reduction following failure of attempted closed reduction.(22) Robertson and Ryan reported three patients who deteriorated during management of cervical subluxation injuries.(25) One of their patients worsened during transport to the hospital. That patient's vertebral injury was found to have spontaneously partially reduced. MRI revealed a disc fragment compressing the cord. A second patient deteriorated following posterior open reduction. MRI revealed disc fragments compressing the cord. The patient underwent subsequent anterior decompression. The third patient deteriorated three days following successful closed-reduction. A subsequent MRI study demonstrated disc material compressing the ventral cervical spinal cord. Five days following deterioration, the patient underwent anterior decompression. All three patients recovered to at least their pre-deterioration neurological examination.(25) Grant et al reported a single case of neurologic deterioration in their series associated with closed reduction that also occurred in a delayed fashion (six hours following reduction). This occurred in a patient subsequently found to have a herniated disc at the level of injury.(11)

Mahale et al reviewed 16 cases of neurological deterioration in patients with cervical spinal cord injuries following reduction of facet dislocations.(20) Seven of the sixteen patients developed complete cord injuries, six following open reduction and one following manipulation under anesthesia. Five patients developed partial injuries, three following manipulation under anesthesia and two following closed traction-reduction. Of the two patients who deteriorated following closed reduction, one was found to be over-distracted. Minor injuries were suffered by

the remaining four patients, including one who deteriorated when the skull traction pins slipped, one who deteriorated in a plaster brace, one who lost reduction and had neurological worsening, and one patient who underwent open reduction. Six patients were investigated with myelography following deterioration, two with MRI and one was evaluated with CT. A disc protrusion was noted in one patient, and a “disc prolapse with hematoma” was noted in another. Both of these patients were treated conservatively. The most common imaging finding in these nine patients was cord edema. (20)

The prevalence of MRI documented disc herniation in association with cervical facet injury with subluxation has caused a number of authors to recommend pre-reduction MRI in patients with these injuries. Harrington et al. reported a series of 37 patients managed with closed reduction, in whom a 97% rate of successful reduction was achieved with no neurologic morbidity.(16) Post-reduction imaging revealed disc herniations in nine patients, four of whom underwent later anterior decompression. Doran et al reported a series of 13 patients drawn from four institutions over an unspecified time period.(8) Nine patients were treated with attempted early closed reduction. Subluxations in three patients were reduced without incident; three patients failed to reduce. Closed reduction was abandoned in another three patients because of worsening pain (one patient) or arm weakness (two patients). All patients underwent MRI evaluation (four pre-reduction). Herniated discs were visualized in ten patients, bulging discs were imaged in three. No patient treated developed a permanent neurological deficit as a result of attempted closed reduction. No patient who underwent successful closed reduction deteriorated. All three who had injuries that were successfully reduced showed significant neurologic improvement despite the MRI appearance of a disc herniation in two and a disc bulge in the third. The authors concluded that pre-reduction MRI studies should be obtained prior to closed reduction in patients with cervical spine facet dislocation injuries. Because there was not

correlation between the presence of disc herniation and neurological deterioration, the recommendation for a pre-reduction MRI in patients with cervical facet dislocation injuries cannot be supported.

Vaccaro and associates studied 11 consecutive patients with pre- and post-reduction MRI. The authors found herniated discs in two patients in the pre-reduction group and in five of nine patients who underwent successful closed reduction. The presence of a herniated disc on pre-reduction MRI or post-reduction MRI did not predict neurological deterioration. No case of deterioration following successful reduction occurred.(37) Grant et al obtained post-reduction MRI studies on 80 patients treated with closed reduction and found herniated or bulging discs in 46%. They found no correlation between MRI results and neurological outcome.(11) Finally, Rizzolo et al performed MRI pre-reduction on 55 patients with cervical fractures and dislocation injuries.(28) They found evidence of disc herniation in 54% of these patients. Awake and alert patients underwent closed traction-reduction. There were no instances of neurological deterioration in this group. The authors did not attempt closed reduction in patients who were not awake.

In summary, a review of the literature reveals only two documented cases (11,25) of neurological deterioration associated with attempted closed reduction of cervical spine fracture-dislocation injuries due to cord compression from disc herniation. Both of these cases were characterized by deterioration hours to days following closed reduction. A number of large clinical series have failed to establish a relationship between the presence of a pre-reduction herniated disc and subsequent neurological deterioration with attempted closed traction-reduction in awake patients.

## SUMMARY

Closed reduction of fracture-dislocation injuries of the cervical spine by traction-reduction appears to be safe and effective for the reduction of spinal deformity in awake patients. Approximately 80% of patients will have their injuries reduced with this technique. The overall permanent neurological complication rate of closed reduction is approximately 1%. The associated risk of a transient injury with closed reduction appears to be 2% to 4%. Closed traction-reduction appears to be safer than manipulation under anesthesia.

There are numerous causes of neurological deterioration in patients with unstable cervical spinal injuries. These include inadequate immobilization, unrecognized rostral injuries, over-distraction, loss of reduction, and cardiac, respiratory and hemodynamic instability. Therefore, the treatment of cervical spine fracture-dislocation injuries must be supervised by an appropriately trained specialist.

Although pre-reduction MRI will demonstrate disc herniation in up to half of patients with facet subluxation injuries, the clinical importance of these findings is questionable. Only two case reports exist which document neurological deterioration due to disc herniation following successful closed traction-reduction in awake patients. Both occurred in delayed fashion after closed reduction. In addition, several investigators have demonstrated the lack of correlation between the MRI finding of disc herniation and neurological deterioration in this patient population. The use of pre-reduction MRI has therefore not been shown to improve the safety or efficacy of closed traction-reduction in awake patients. MRI prior to fracture-dislocation reduction may result in unnecessary delays in accomplishing fracture realignment and decompression of the spinal cord. As Class III evidence exists in support of early closed reduction of cervical fracture-dislocation injuries with respect to neurological function, pre-reduction MRI in this setting is not necessary. The ideal timing of reduction is unknown, but

many investigators favor reduction as rapidly as possible after injury to maximize the potential for neurological recovery.

Patients who fail attempted closed reduction of cervical fracture injuries have a higher incidence of anatomic obstacles to reduction including facet fractures and discs herniation. Patients who fail closed reduction should undergo more detailed radiographic study prior to attempts at open reduction. The presence of a significant disc herniation in this setting is a relative indication for an anterior decompression procedure, either in lieu of or preceding a posterior procedure.

Patients with cervical fracture dislocation who cannot be examined, due to head injury or intoxication, cannot be assessed for neurologic deterioration during attempted closed traction-reduction. For this reason, an MRI prior to attempted reduction is recommended as a treatment option.

## **KEY ISSUES FOR FUTURE INVESTIGATION**

A prospective cohort study of patients with cervical spinal cord injuries due to facet fracture-subluxation injuries treated with or without pre-reduction MRI would provide Class II medical evidence in support of a treatment recommendation on this issue. This could address issues of timing. A randomized controlled trial may provide Class I medical evidence.

No prospective comparative study of closed reduction versus anterior decompression and stabilization for patients with MRI documented herniated discs in association with unreduced cervical fracture-dislocation injuries has been performed. A prospective study of this issue would provide Class II medical evidence in support of a treatment recommendation.

## EVIDENTIARY TABLES

First Author Reference	Description of Study	Results	Data Class	Conclusions
Grant et al. <i>J Neurosurg</i> Spine 90:13-18, 1999	82 pts. Retrospective series All closed C-spine injuries with malalignment included Unilateral and Bilateral locked facets Early rapid closed reduction attempted in all patients MRI scans obtained after reduction ASIA and Frankel Grades determined on admission, 6, 24 hrs Weight up to 80% of patient's body weight	Successful reduction in 97.6% Average time to reduction 2.1 +/- 0.24 hours Overall ASIA scores improved by 24 hours following reduction. 1 patient deteriorated 6 hours post-reduction (probable root lesion). 46% had disc injury on MRI, 22% had herniation. Disc injury on MRI did correlate with cord edema on MRI	III	Closed reduction is effective and safe despite high incidence of MRI demonstrable disc injuries/ herniations
Vital et al, <i>Spine</i> 23:949-955, 1998	168 Patients retrospective series unilateral and bilateral employed manipulation under general anesthesia in minority of cases used relatively light weights (max 8.8 lbs plus 2.2 per level for max ox 40 lbs) All patients operated upon immediately post-reduction or post failure of reduction MRI's not done prereduction (although discs noted in 7 patients?)	43% reduced by traction without anesthesia (time <2 hrs) 30% reduced by manipulation under anesthesia 27% reduced intraoperatively 5 patients did not reduce (delayed referral, surgical error) Authors observed no cases of neurological deterioration	III	Authors promote their protocol as a safe and effective means for reduction and stabilization of fractures.
Lee AS: <i>JBJS</i> 76B: 352-6, 1994	210 pts Rapid traction-reduction 119, manipulation under anesthesia in 91 Retrospective historical cohort study Groups similar except traction group had longer delay to treatment Weights up to 150 lbs used No MRI done pre-reduction	Reduction Successful: MUA: 66/91 (73%) RT: 105/119 (88%) All failures in RT group were due to associated fractures or delayed referral Time to reduction: RT 21 minutes MUA: not reported No loss of Frankel Grade in either group 6 MUA and 1 RT had deterioration on ASIA score	III	RT superior to MUA, both procedures safe and effective, MRI not done.

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Results</b>	<b>Data Class</b>	<b>Conclusions</b>
Cotler, <i>Spine</i> 18:386-390, 1993	24 patients (all awake) prospective study no fractured facets all acute injuries (1 5 day patient) Weights up to 140 lbs used No CT or MRI done	All 24 reduced No incidence of neurological deterioration Manipulation used in addition to weights in 9 patients (when facets perched) Time required ranged from 8-187 minutes	III	Reduction with weights up to 140 lbs is safe and effective in monitored setting with experienced physicians
Mahale, <i>JBJS</i> 75B:403-9, 1993	341 patients treated for traumatic dislocations of cervical spine 15 suffered neurological deterioration variety of treatments used to reduce deformity (4.3%)	Complete injuries: 6 after OR, 1 after manipulation Incomplete Injuries: 1 after OR, 3 after manipulation, 2 after traction, 1 during application of cast Radiculopathy: 1 (occurred when tongs slipped during traction) Deterioration delayed in 11 patients	III	Numbers of patients subject to each treatment arm not given. Purely a descriptive paper. Only conclusion is that neurological deterioration can happen.
Hadley et al. <i>Neurosurg</i> 30:661-666, 1992	68 patients retrospective series Facet fracture dislocations only Unilateral and Bilateral locked facets 66 treated with early attempted closed reduction (2 late referrals) Average weights used for successful reduction were between 9-10 lbs/cranial level	58% of patients had successful reduction Overall, most patients (78%) demonstrated neurological recovery by last followup (not quantified) 7 patients deteriorated during "treatment" (six improved following ORIF, one permanent root deficit following traction) No MRI data reported	III	Early decompression by reduction led to improved outcomes based on fact that patients who did best were reduced early (<5-8 hrs). No comparison possible between CR and ORIF due to small numbers. 1.2% permanent deficit (root) related to traction
Starr et al. <i>Spine</i> 15:1068-1072, 1990	57 patients retrospective series Unilateral and bilateral Early rapid reduction attempted in all patients No MRI done pre-reduction One patient was a delayed transfer Weights up to 160 lbs (began at 10 lbs) Frankel grades recorded at admission and discharge	53/57 (93%) reduced mean time to reduction was 8 hours no patient deteriorated a Frankel grade Two patients lost root function, one transiently 45% improved one Frankel grade by time of d/c, 23% improved less substantially 75% of patients required >50 lbs	III	Closed reduction is safe and effective for decompressing cord and establishing alignment.

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Results</b>	<b>Data Class</b>	<b>Conclusions</b>
Sabiston et al. <i>J Trauma</i> 28:832-835, 1988	39 patients retrospective series unilateral and bilateral injuries up to 70% of body weight used all acute injuries No MRI	35/39 (90%) of patients successfully reduced average weight used 62.5 lbs no neurological deterioration failures due to surgeon unwillingness to use more weight	III	Closed reduction with up to 70% of body weight is safe and effective for reducing locked facets. Authors state that patients seen in delayed fashion (>10 days) are unlikely to reduce (no evidence presented here)
Maiman, DJ: <i>Neurosurg</i> 18:542-547, 1986	28 patients Variety of treatments offered No MRI 18 patients had attempt at closed reduction (max weight 50 lbs)	10/18 reduced with traction No patient treated by authors deteriorated One referred patient had an over distraction injury	III	Mixed group of patients and treatments. In general, traction seemed to be safe.
Kleyn, PJ: <i>Paraplegia</i> 22: 271-281, 1984	101 patients unilateral and bilateral, all with neurological involvement All treated with traction If injury <24 hours, MUA attempted initially, if reduction fails with max 18 kgs weight, MUA performed Pre-MRI	82/101 successfully reduced (4 open reduction, 6 partial reduction accepted, 9 no further attempt due to poor condition of patient) 37/45 incomplete lesions improved 7/56 complete lesions improved no neurological deterioration	III	Traction followed by MUA is safe, usually (80%) effective, and may result in improved neurological function.
Sonntag, <i>J Neurosurg</i> 8:150-152, 1981	15 patients retrospective analysis all bilateral locked facets all acute injuries manual traction, tong traction, and open reduction used weight used ranged from 30-75 lbs No MRI done	Reduction with traction successful in 10 patients 5 failed: 1 with C1 fracture which did not allow traction, 2 with fractured facets, 1 with radicular sx's worsened by traction (transient), 1 with an ascending spinal cord injury (patient died of pulmonary complications 2 weeks later)	III	Stepwise algorithm (traction, manual manipulation, operative reduction is indicated. Closed reduction by weight application is the preferred method for reduction of deformity.

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Results</b>	<b>Data Class</b>	<b>Conclusions</b>
Shrosbee R <i>Paraplegia</i> 17: 212-221, 1979-80	216 patients identified with locked facets* Used traction (no weight specified) followed by manipulation under anesthesia if traction failed. Pre-MRI  *86 died within 3 months, excluded from series	70/95 unilaterals reduced (74%) 77/121 bilaterals reduced (64%) No neurological morbidity reported Patients who were successfully reduced improved more often than patients who were not successfully reduced (41% vs 32% unilateral, 16% vs. 0% bilateral)	III	Discarded patients and lack of statistical analysis preclude firm statements. Highly suggestive paper. Conclusions: Traction followed by manipulation is safe, usually effective, and reduction seems to improve outcome (or, patients who are reducible do better)
Burke <i>DCJBJS</i> 53B:165-182, 1971	41 patients treated by MUA light traction followed by induction of anesthesia and intubation, followed by manipulation under anesthesia if necessary (same as Evans) 32 patients treated with traction alone 3 treated by traction after manipulation failed C7-T1 not attempted Pre-MRI	37/41 successfully reduced by MUA 21/25 reduced with traction prior to anesthetic 7 patients were judged too sick for anesthesia and underwent traction for stabilization, not reduced 2 cases of neurological deterioration: 1 over distraction 1 unrecognized injury	III	MUA and traction both safe if proper diagnosis and careful attention paid to radiographs
Evans DK: <i>JBJS</i> 43B:552-555, 1961	17 patients treated by induction of anesthesia and intubation, sometimes with manipulation under anesthesia Pre-MRI	No neurological deterioration noted All successfully reduced 2 unchanged, 2 died, 13 improved	III	Reduction under anesthesia safe and effective. Small series.

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## **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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## **BLOOD PRESSURE MANAGEMENT FOLLOWING ACUTE SPINAL CORD INJURY**

### **RECOMMENDATIONS**

Standards: There is insufficient evidence to support treatment standards.

Guidelines: There is insufficient evidence to support treatment guidelines.

Options:

- Hypotension (systolic blood pressure < 90 mm Hg) should be avoided if possible or corrected as soon as possible following acute SCI.
- Maintenance of mean arterial blood pressure at 85 – 90 mm Hg for the first seven days following acute SCI to improve spinal cord perfusion is recommended.

### **RATIONALE**

Acute traumatic spinal cord injury is frequently associated with systemic hypotension. Hypotension may be due to associated traumatic injuries with hypovolemia, direct severe spinal cord trauma itself, or a combination. The occurrence of hypotension has been shown to be associated with worse outcomes after traumatic injury, including severe head injury.(1,2,8,16,20,24) While a prospective controlled assessment of the effects of hypotension on acute human SCI has not been performed, laboratory evidence suggests that hypotension contributes to secondary injury after acute SCI by further reducing spinal cord blood flow and perfusion.(1,3,4,8,16,18,19,20-22,24) Hypotension in animal models of spinal cord injury results in worse neurological outcome.(13,14,23,26,28,29) Several clinical series of human patients with acute SCI managed in an aggressive fashion with attention to blood pressure, oxygenation and hemodynamic performance report no deleterious effects of therapy and suggest improved neurological outcome. (13,14,23,26,28,29) Despite these observations, the majority of patients with acute SCI treated in contemporary practice are not routinely monitored nor treated

with blood pressure augmentation following injury. For these reasons the issues of routine blood pressure support and threshold levels of mean arterial pressure maintenance following acute SCI have been raised.

## **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, hypotension and spinal cord blood flow. Approximately 3000 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 the guidelines. We focused on two specific topics concerning human patients with acute spinal cord injuries: hypotension (22 articles reviewed) and spinal cord blood flow (no articles identified). Additional references were culled from the references 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, related general review articles, and relevant studies of hypotension and human traumatic brain injury referenced in the Scientific Foundation are included in the bibliography. These efforts resulted in six manuscripts describing clinical case series (Class III evidence), which form the foundation for this review. They are summarized in Evidentiary Table format.

## SCIENTIFIC FOUNDATION

Ischemia of the spinal cord is felt to be one of the most important contributors to neuronal injury and neurologic deficit after acute spinal cord injury. Both local and systemic vascular alterations can contribute to ischemia after acute SCI by further reducing spinal cord blood flow which can exacerbate and extend the principle spinal cord insult.(1,6,8,16,20,24)

In the normal, non-injured spinal cord, arterial blood supply is diffuse, primarily delivered via a single anterior spinal artery and two posterior spinal arteries. A variable number of anterior and posterior radicular arteries provide segmental contributions over the length of the cord.(24,25) They feed anastomotic arterial channels over the pial surface that supply the outer half of the cord, and penetrating central arteries from the anterior spinal artery, which supply the central portion of the cord. Terminal branches of the central arteries extend rostral and caudal to overlap with adjacent terminal arteries, yet the terminal arterioles that originate from the terminal arteries do not interconnect within the cord. They in turn give rise to an extensive capillary network, which does interconnect within the deep gray and white matter of the cord. Capillaries are much more numerous and extensive in the gray matter than the white matter reflecting the increased metabolic needs of cell bodies compared to axons.(24,25) Perfusion of the spinal cord under normal physiological circumstances is maintained over a wide range of systemic blood pressure by autoregulatory mechanisms that appear identical to those which regulate cerebral blood flow. (1,3-5,7,9,10,15,16,18,19-21,24)

Local vascular alterations after acute SCI are multiple and the precise mechanisms of injury-induced ischemia of the cord have yet to be elucidated. Most investigators cite direct vascular injury at the site of the primary trauma as the earliest component of the ischemic injury process.(1,6,8,20-22) The principal spinal cord injury leads to not only white and gray matter injury at the insult site, but because of sulcal vessels and collateral terminal arteries which pass

through the primary injury site, creates white matter ischemia distal to the direct injury site.(8,19,20,24) In addition, the primary SCI creates intraluminal thrombosis, vasospasm and initiates a variety of secondary injury biochemical phenomena that further reduce blood flow, injure endothelium or increase edema, microvascular compression and contribute to microvascular collapse.(1,8,19,20,22,27) Post-traumatic spinal cord ischemia has been shown to become progressively worse over the first several hours after injury in animals.(1,4,6,7,16,20)

Laboratory models of spinal cord injury have convincingly demonstrated that autoregulation of spinal cord blood flow is lost after injury, exacerbating local spinal cord ischemia and rendering the spinal cord vulnerable to systemic hypotension.(1,3-5,7,8,16,18,20,27) This is analogous to that which often occurs in regional cerebral vasculature after acute traumatic brain injury. (1,4,5,7,8,10,15,16,20,22,24,27)

Systemic hemodynamic alterations after acute SCI have been well documented and include hypotension, cardiac dysrhythmias, reduced peripheral vascular resistance and reduced cardiac output.(1,12,13,14,17,20,26) Patients with the most severe injuries, particularly those with severe cervical spinal cord injuries are at greatest risk for cardiac, hemodynamic and respiratory disturbances in the first week following acute SCI.(11,12,17) These untoward occurrences, which may be episodic in nature, can result in hypotension and hypoxia. If, as many investigators suspect, acute SCI with loss of spinal cord autoregulation is analogous to acute traumatic brain injury, hypotension and hypoxia can worsen the severity of the original insult and can be disastrous for potential neurological recovery.(1,8,20,21) While the relationship between systemic hypotension and outcome following acute SCI has not been directly studied in human patients, inference from studies of patients with traumatic brain injury (TBI) appears appropriate.(2,8,20) Prospectively collected data from the Traumatic Coma Data Bank (Class II evidence) demonstrates that hypotension (systolic blood pressure < 90 mm Hg) or

hypoxia ( $\text{paO}_2 < 60 \text{ mm Hg}$ ) were independently associated with significant increases of morbidity and mortality following severe TBI.(2) A single episode of hypotension was associated with a 150% increase in mortality. It is in this very setting that therapeutic intervention aimed at correcting hypotension and maintaining threshold levels of MAP to improve cerebral or spinal cord perfusion has its greatest potential.

Several reports of case series suggest that treatment of hypotension and resuscitation to maintain mean arterial blood pressure at high-normal levels, 85 to 90 mm Hg, may enhance neurological outcome following acute traumatic SCI. (13,14,23,26,28,29)

Zach et al, utilized a prospective aggressive medical management paradigm in the treatment of 117 consecutive acute SCI patients.(28) All patients were treated in the intensive care unit with central venous pressure monitoring and were treated with volume expansion (Rheomacrodex 40, 500 ml/day) for maintenance of systemic blood pressure for seven 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. 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” with attention to maintenance of acceptable blood pressure appeared to improve neurologic recovery.

Tator and colleagues in 1984 described their experience with 144 patients with acute SCI managed between 1974 and 1979 at a dedicated spinal cord injury unit in Toronto, Canada.(23)

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. 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.

Wolf et al, in 1991 reported their experience with fifty-two patients with acute cervical bilateral facet dislocation injuries managed with 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.(27) 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. The

authors described 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. 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 above 85 mm Hg 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 setting 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.(13) 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. Volume resistant hypotension was 5.5 times more common among patients with complete motor injuries. Forty percent of patients managed by protocol including several with complete injuries improved, 42% remained unchanged and nine patients died (18%). 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.(26) 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, 25 with incomplete cervical injuries, 21 patients with complete thoracic SCI and eight patients with incomplete thoracic level SCI. The average admission MAP for complete cervical SCI patients was 66 mm Hg. Nine of ten complete cervical SCI patients 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) three of ten complete 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 SCI 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.

The collective experience described in these case series (Class III evidence) strongly suggests that maintenance of MAP at 85 to 90 mm Hg improves spinal cord perfusion or impacts neurological outcome.(13,14,23,26,28,29) Prompt treatment of hypotension and resuscitation to MAP levels of 85 to 90 mm Hg is safe, and it suggests that elevation of MAP to threshold levels

may be beneficial to patients with acute SCI. The seven-day duration of treatment and the threshold levels of MAP maintenance appear to have been chosen arbitrarily by the individual clinical investigators.(13,26,28) They are felt to be analogous to initial duration and threshold MAP level recommendations for management of patients following acute traumatic brain injury. None of the authors provides a specific recipe or an algorithm to guide blood pressure augmentation. All of the manuscripts describe acutely injured patients who have arterial lines and central venous or Swan Ganz catheters in place to monitor pressures and volume status.(13,14,23,26,28,29) Initially crystalloid is given intravenously in response to MAP below 85 mmHg. Colloid is administered if the hematocrit is low (blood) or as a volume expander (albumin). If the patient's volume status is optimal but the MAP remains below threshold, the authors describe the use of pressors, typically (although not exclusively) a beta-agonist (Dopamine) before the addition of an alpha-agonist (Neosynephrine), to elevate the MAP. These agents are titrated to the appropriate dose level to achieve the threshold MAP utilizing volume, pressure, and cardiac performance data provided by the invasive monitoring devices.

## **SUMMARY**

Hypotension is common after acute traumatic SCI in humans. Hypotension contributes to spinal cord ischemia after injury in animal models and can worsen the initial insult and reduce the potential for neurological recovery. Although unproven by Class I medical evidence studies, it is likely that this occurs in human SCI patients as well. Since the correction of hypotension and maintenance of homeostasis is a basic principle of ethical medical practice in the treatment of patients with traumatic neurological injuries, depriving acute SCI patients of this treatment would be untenable. For this reason, Class I evidence about the effects of hypotension on outcome following acute human SCI will never be obtained. However, correction of

hypotension has been shown to reduce morbidity and mortality after acute human traumatic brain injury, and is a guideline level recommendation for the management of TBI. While a similar treatment guideline cannot be supported by the existing spinal cord injury literature, correction of hypotension in the setting of acute human SCI is offered as a strong treatment option. Class III evidence from the literature suggests that maintenance of mean arterial pressure at 85 to 90 mm Hg after acute SCI for a duration of seven days is safe and may improve spinal cord perfusion and ultimately, neurological outcome.

### **KEY ISSUES FOR FUTURE RESEARCH**

The issue of whether or not blood pressure augmentation has an impact on outcome following human SCI is important and deserves further study. If augmentation of mean arterial pressure is determined to be of potential benefit, the threshold levels of MAP most appropriate and the length of augmentation therapy need definition. These issues are best analyzed in a multi-institution prospective cohort study or a properly designed multi-institution retrospective case control study.

## EVIDENTIARY TABLE

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
Vale et al, 1997, J Neurosurg	Prospective assessment of 77 ASCI treated in ICU, aggressive Hemodynamic support, MAP > 85 No control group	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 med treatment, MAP > 90	CLASS III	Improved outcome with aggressive hemodynamic support at 6 weeks post-injury.
Levi et al, 1991, Neurosurgery	103 ACSI, 50 incomplete (Group A), 53 complete (Group B), ICU care hemodynamic support, MAP > 85	CLASS III	Improved neurological outcome, no sig. 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..	CLASS III	Closed reduction 61% 52% 1 year follow up. In general, improved neurological outcome with hemodynamic therapy.
Tator, et al, 1984, Canadian J Surg	144 ASCI patients managed per protocol of ICU care, hemodynamic support. Compared to prior cohort	CLASS III	Improved neurological outcome, less mortality with early transfer and ICU care
Zach, et al, 1976 Paraplegia	Prospective assessment of 117 ACSI at Swiss Center, ICU setting Aggressive BP, volume therapy Rheomacrodex x 7d Dexamethasone x 10d No comparison or control group	CLASS III	Improved neurological outcome with aggressive medical treatment and blood pressure management. Better outcome for early referrals.

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## **THERAPY AFTER ACUTE CERVICAL SPINAL CORD INJURY**

### **RECOMMENDATIONS**

#### **Corticosteroids:**

- Standards:** There is insufficient evidence to support treatment standards.
- Guidelines:** There is insufficient evidence to support treatment guidelines.
- Options:** Treatment with There is insufficient evidence to support treatment standards. Methylprednisolone for either 24 or 48 hours is recommended as an option in the treatment of patients with acute spinal cord injuries that should be undertaken only with the knowledge that the evidence suggesting harmful side effects is more consistent than any suggestion of clinical benefit.

#### **GM-1 Ganglioside:**

- Standards:** There is insufficient evidence to support treatment standards.
- Guidelines:** There is insufficient evidence to support treatment guidelines.
- Options:** Treatment of patients with acute spinal cord injuries with GM-1 ganglioside is recommended as an option without demonstrated clinical benefit.

### **RATIONALE**

The hope that administration of a pharmacological agent delivered shortly after acute spinal cord injury (ASCI) might improve neurological function and/or assist neurological recovery has long been held. A variety of promising substances have been tested in animal models of ASCI, but few have had potential application to human spinal cord injury (SCI) patients. Four pharmacological substances have met rigorous criteria in laboratory testing and initial human investigations: two corticosteroids (methylprednisolone and tirilazad mesylate), naloxone, and GM-1 ganglioside. All four pharmacological agents have been evaluated in controlled, randomized, blinded clinical trials of human patients with ASCIs. Two of these substances, tirilazad and naloxone, have been studied less extensively and as yet have unclear efficacy in the management of acute human SCI. The purpose of this medical evidence-based review is to define the usefulness of administration of methylprednisolone with or without GM-1 ganglioside in the contemporary management of ASCI patients.

### **SEARCH CRITERIA**

A computerized search of the National Library of Medicine database of literature published from 1966 to 2001 was undertaken. The following medical subject headings were used in combination with "spinal cord injury" and "neurological deficit": steroids, methylprednisolone, and GM-1 ganglioside. Approximately 2400 citations were acquired. Non-English language citations and nonhuman experimental studies were deleted. Titles and abstracts of 652 manuscripts were reviewed, 639 on the topic of corticosteroids and human SCI and 13 on the topic of GM-1 ganglioside and human SCI. Additional references were culled from the reference lists of the remaining papers. Finally, the 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. Duplications, case reports, pharmacokinetic reports, general reviews, and articles with mention of one agent or another but without scientific assessment were eliminated. Several editorials, critiques, and responses to published reports and studies were included. Forty-six published references on the topic of methylprednisolone in the treatment of patients with ASCI and seven published

references for GM-1 ganglioside provide the basis for this guideline. Thirteen studies on methylprednisolone and two studies on GM-1 ganglioside are summarized in Tables 9.1 and 9.2.

## **SCIENTIFIC FOUNDATION**

### **Methylprednisolone**

Corticosteroids, particularly methylprednisolone, have been studied extensively in animal models of SCI (2,19,47,48,50,51). Although their precise mechanisms of action are not completely known, they have the potential to stabilize membrane structures, maintain the blood-spinal cord barrier potentially reducing vasogenic edema, enhance spinal cord blood flow, alter electrolyte concentrations at the site of injury, inhibit endorphin release, scavenge damaging free radicals, and limit the inflammatory response after injury (2,47,48,50,51). After considerable positive study in the laboratory, methylprednisolone was studied in human SCI patients in a multicenter, randomized, double-blinded clinical trial initiated in 1979. The first National Acute Spinal Cord Injury Study (NASCIS I) (11), reported in 1984, compared the efficacy of administration of a 100-mg bolus of methylprednisolone and then 100 mg daily thereafter for 10 days with administration of a 1000-mg bolus and then 1000 mg daily for 10 days in 330 acute injury patients assessed 6 weeks and 6 months after injury. There was no control group. The study revealed no difference in neurological recovery (motor or sensory function) between the treatment groups at either 6 weeks or 6 months after injury. Motor scores were determined from the examination of seven muscle groups on each side of the body scored on a 6-point scale. Sensory function was assessed using a 3-point scale of dermatomal light touch and pinprick sensation. The authors reported the motor and sensory scores from the right side of the body only. There was no anatomic level injury limit (superior to T12 vertebral level, for example) in the study to include only SCI patients and exclude primary cauda equina injuries or “mixed” central and cauda equina injuries that might occur with a lower fracture injury (e.g., T12-L1 or L1-L2 injuries). The study did not require a minimum motor impairment for inclusion; hence, patients with normal motor examinations and those with minimal neurological deficits were included in the study if the attending physician determined that the patient had an SCI of any severity. In 1985, the same group of investigators reported on the 1-year follow-up of these study patients (15). No differences in motor or sensory outcome were identified between the two treatment groups.

Animal studies of the efficacy of methylprednisolone after experimental SCI suggested that the doses of methylprednisolone used in the NASCIS I investigation were too low to demonstrate a significant in outcome (2,14,19,50,51). A multicenter NASCIS II trial was initiated in 1985 using a much higher dose of methylprednisolone (30 mg/kg as a bolus and then 5.4 mg/kg/h infusion for 23 h). These patients were compared with similarly injured patients who received either naloxone (5.4 mg/kg bolus and then an infusion of 4.0 mg/kg/h for 23 h) or placebo. Patients had to be randomized to one of three treatment arms within 12 hours of ASCI. The results of NASCIS II were reported in 1990 (14). Four hundred eighty-seven patients were entered into the study; 162 received methylprednisolone, 154 were given naloxone, and 171 patients were in the placebo control group. The authors reported that the administration of methylprednisolone within 8 hours of injury was associated with a significant improvement in motor function (neurological change scores, right side of body only,  $P = 0.03$ ), and in sensation (pinprick,  $P = 0.02$ ; light touch,  $P=0.03$ ) at the 6-month follow-up compared with patients receiving methylprednisolone more than 8 hours after injury and patients receiving naloxone or placebo. No similar significant improvements were noted at the 6-week follow-up, either motor or sensory. Motor scores were determined from the examination of seven muscle groups on each side of the body scored on a scale of 0 to 5 points. Sensory function was assessed using a 3-point scale of dermatomal light touch and pinprick sensation. The NASCIS II study reported on the motor scores from the right side of the body only. Bilateral sensory scores were provided. Like the NASCIS I study, there was no anatomic level injury limit in the study (superior to T12 vertebral level, for example) to ensure that only SCI

patients were included for study (11,15). Similarly, NASCIS II did not require a minimum motor impairment for inclusion; hence, patients with normal motor examinations and those with minimal neurological deficits were included. No outcome measures involving patient function were used in this study. In 1992, NASCIS investigators reported on the 1-year follow-up of NASCIS II study patients (13). They reported statistically significant improvement in motor scores on the right side of the body for 62 of 487 study patients ( $P = 0.03$ ). These 62 patients received methylprednisolone within 8 hours of injury. Significant right body motor score improvement was identified in two of three categories of patients, plegic patients with total sensory loss ( $P = 0.019$ ) and paretic patients with variable sensory loss ( $P = 0.024$ ), but not among plegic patients with partial sensory loss ( $P = 0.481$ ). There were no significant improvements in motor change scores described among the remaining 421 patients entered in the study. There were no significant differences in sensory scores for any treatment group or categories of patients despite the differences reported at the 6-month follow-up for patients receiving methylprednisolone within 8 hours of injury. Patients treated more than 8 hours after injury with methylprednisolone or naloxone experienced less recovery of motor function compared with placebo treatment patients. The authors concluded that treatment with the study dose of methylprednisolone administered within 8 hours of injury improves neurological outcome and is therefore indicated in the treatment of patients with ASCI. The use of study dose methylprednisolone in patients was not associated with harmful side effects compared with patients in the other treatment groups, although the authors reported an increased incidence of wound infection and gastrointestinal bleeding among corticosteroid-treated patients. Treatment with methylprednisolone beyond 8 hours after injury was not recommended.

There are several flaws in the NASCIS II study, and criticism has been offered on several methodological, scientific, and statistical issues (18,19,22,31,32,35,37,40-42,44-46,51). The investigators described two a priori hypotheses: that treatment effect would be influenced by how soon the drug was given after injury and by the severity of injury. Patients were considered eligible for inclusion if they were admitted to the study and randomized to treatment within 12 hours of injury. At some point, patient outcome was stratified according to the timing of methylprednisolone administration (<8h, >8h). Some reviewers have requested examination of the raw data to look for time-related diminishing effects of methylprednisolone administration relative to injury rather than assignment of an "all or nothing" time cutoff (18,32,37,40,42,51). Analysis of results of the entire population of patients according to the second a priori hypothesis was not provided by the authors (18,31,37,40,42,51). Analysis using the second hypothesis was accomplished on the group of patients previously stratified according to the first hypothesis. It may be that the two hypotheses are fully independent, yet no justification for this assumption was offered (31,40). The study did not offer a standardized medical treatment regimen for all ASCI patients in this study. The medical management of study patients including monitoring, blood pressure augmentation, respiratory care, deep venous thrombosis prophylaxis, nutritional support, and initiation of rehabilitation activities was neither consistent within centers nor consistent from center to center (18,22,31,37). Similarly, surgical treatment offered to patients in the NASCIS II study was not consistent from center to center (19,31,35,51). There was no description of surgical approaches used for specific pathology or documentation of the timing of surgical intervention for individual patients. There was no consideration given to the independent effect that either aggressive medical management or surgery had, or may have had, on outcome (18,19,22,31,35,37,51).

The most important and significant criticism of the NASCIS II study is the failure to measure patient functional recovery (e.g. functional independence measure [FIM]) to determine animation (change in motor scores) in the methylprednisolone treated patients had meaningful clinical significance (18,32,35,37,44). It is unclear from the change in score data provided whether the improvement had any clinical significance to the injured patients (1,18,32,35,37,44-46). One of the most frequent criticisms of the reported NASCIS II results is the failure to provide scientific data on which statistical comparisons were made (18,19,31,32,37,40-42,46,51). As with the NASCIS I study, only right-sided motor scores were reported in NASCIS II, but bilateral sensory scores were reported. Change in motor score

(improvement) on the right side only of ASCI patients has been cited by the study authors as a significant neurological benefit associated with methylprednisolone administration given at study doses within 8 hours of injury and assessed at 6-month and 1-year follow-up ( $P = 0.03$ ) (13,14). These findings were observed in only a small subset of study patients (18,31,37,41). Was this an a priori hypothesis of the investigators and was the result significant for the whole population of patients? If so, then the finding stands and the post hoc subgroup analysis suggests which subgroup receives the benefit. If, however, the entire result is from a post hoc hypothesis and analysis and is significant only for the subgroup and not for all of the patients analyzed together, then it is a weak suggestive finding. This is not made clear by the authors. Reviewers have argued against the use of right-side only motor scores, and particularly the change of score results in NASCIS II publications (18,22,31,32,40,41). The lack of evidence describing left-sided motor scores and total body motor scores in NASCIS II is confusing (4,8-10,12,50).

Also confusing is the reported difference in change of motor score outcome for patients with incomplete SCI who were in the placebo treatment arm. Patients with incomplete SCIs in the NASCIS II study who received placebo more than 8 hours after injury had significantly better neurological recovery than did patients who received placebo within 8 hours of injury (13,18,32,42). Additionally, the neurological recovery curve generated for patients with incomplete SCIs treated with methylprednisolone within 8 hours of injury is virtually identical to that of patients with incomplete SCIs treated with placebo beyond 8 hours after injury. The benefit of treatment with respect to neurological recovery (motor change score) with methylprednisolone given within 8 hours of injury seems equal to treatment with placebo more than 8 hours after injury (18,37,42).

Statistical criticisms of the NASCIS II results are many (18,19,22,31,32,40-42,45,46,51). They include potential interpretive errors, problematic statistical comparisons, simplification of subgroup analysis from the pre-planned 15 categories to 3 seemingly arbitrarily determined categories, an improper and incomplete presentation of odds ratios, and a post hoc analysis of study data including only 127 patients (62 methylprednisolone, 65 placebo) treated within 8 hours of injury, rather than the entire study population of 487 patients (18,19,22,31,32,40-42,45,46,51). NASCIS II was designed and implemented to be a randomized, controlled, double-blinded clinical study in an attempt to generate Class I evidence on the efficacy of methylprednisolone and naloxone after ASCI in human subjects. The lack of a measure of functional significance, the dependence on post hoc analyses, and the absence of an analysis of surgical treatment diminish the quality and usefulness of the evidence provided by these studies.

In 1993, Galandiuk et al (21) described 32 patients with cervical or upper thoracic ASCIs managed in an urban trauma center. Fourteen patients who received NASCIS II doses of methylprednisolone within 8 hours of injury were compared with 18 ASCI patients with similar injuries managed without corticosteroids. The authors reported no difference in neurological outcome between the two sets of patients but noted that methylprednisolone-treated patients had immune response alterations (lower percentage and density of monocyte Class II antigen expression and lower T-cell helper/suppressor cell ratios), a higher rate of pneumonia (79% versus 50%), and longer hospital stays (44.4 d versus 27.7 d) than similar ASCI patients they managed without administration of corticosteroids. Although the conclusions drawn by the authors are interesting, they have little scientific power. The mix of historical patients with contemporary patients, the lack of a prospective design, and the haphazard assignment and assessment of patients dilute the quality of the evidence provided.

Bracken and Holford (8) described the effect of timing of methylprednisolone on neurological recover in NASCIS II study patients in 1993. They concluded from post hoc analysis of the NASCIS II data that methylprednisolone administered to patients within 8 hours of ASCI improves neurological function below the level of the spinal cord lesion in patients initially diagnosed as having complete or incomplete injuries. The majority of the improvements they reported were among patients with incomplete SCIs at admission. Complete injury patients demonstrated very little recovery below the level

of injury irrespective of treatment. Their post hoc analysis also confirmed that methylprednisolone administered more than 8 hours after injury may be associated with a worse neurological outcome.

This 1993 article (8) refers to and references the 1-year follow-up NASCIS II study data, but only describes patient groups and offers percentages (18,42). It provides neither new evidence nor the numbers of patients on whom Bracken and Holford based their conclusions. Although the result that the authors describe is positive (methylprednisolone administered within 8 h of injury improves spinal cord function in patients with SCI), it was identified in a very small subgroup of patients, which raises questions as to its true weight and validity. The manner in which the data and conclusions were presented is ambiguous and suggests that this was a positive result reflected by analysis of the entire NASCIS II study population ( $n = 487$ ) (18). In fact, it was only a subgroup analysis of the population of patients who received methylprednisolone within 8 hours of injury ( $n = 62$ ), compared with those who received placebo within 8 hours of injury ( $n = 65$ ). Forty-five methylprednisolone-treated patients had complete injuries and demonstrated very little change in function below the level of injury. The same is true for 43 similar (complete) patients who received placebo (no significant difference). The actual differences described by the authors are based on 17 methylprednisolone patients compared with 22 placebo-treated patients, all of whom had incomplete SCI and had therapy initiated within 8 hours of injury (18).

Their report (8) does help to clarify the issue of recovery of function (motor score change) in NASCIS II patients with complete injuries at admission who received methylprednisolone within 8 hours of injury. The NASCIS II results at 1 year cite a significant improvement in motor function for patients who received methylprednisolone at study doses within 8 hours of injury compared with placebo-treated patients ( $P=0.03$ ) (13). For the patients who had complete injuries who met the early treatment criteria ( $n=45$ ), the significance of improvement (change in motor score) was  $P = 0.019$ , compared with similar patients who received placebo. Bracken and Holford's (8) post hoc analysis revealed no significant difference in recovery below the level of the lesion in these patients compared with placebo-treated patients. This suggests that the primary improvements in function identified in the NASCIS II study for patients with complete spinal injuries treated within 8 hours were at the level of injury, likely root recovery, rather than a significant gain in spinal cord function (18). Again, the relationship between any such recovery and an improvement in patient function is unknown, irrespective of the sample size, because the study did not use functional outcome assessments (18,35,37).

In 1994, Duh et al (20) reported on the effect of surgery on outcome among NASCIS II study patients. In all, 298 of 487 study patients underwent 303 operative procedures, 56 via the anterior approach and 247 via the posterior approach. The authors examined the influence of surgery on neurological outcome across all study groups of patients at time periods of less than 25 hours, 26 to 50 hours, 51 to 100 hours, 101 to 200 hours, and more than 200 hours. They found that the most severely injured patients were less likely to be treated surgically. The authors did not identify significant differences in outcome, motor or sensory, with surgical treatment, either early or late. Functional recovery was not measured.

Gerhart et al (29), in 1995, reported a population-based, concurrent cohort comparison study of 363 ASCI survivors treated in Colorado. Two hundred eighteen patients were managed between May 1990 and December 1991, and 145 injury patients were managed 2 years later in 1993. Of 218 patients managed in 1990 to 1991, 100 (46%) were treated according to the NASCIS II protocol. Fifty-one patients (23%) received no methylprednisolone, and 67 patients (31%) received another corticosteroids, were given an incorrect dose, or had insufficient data. In the 1993 study population, 61% of ASCI patients ( $n = 88$ ) received methylprednisolone according to NASCIS II protocol. Thirty-nine patients (27%) received no methylprednisolone and 18 patients (13%) were given another corticosteroid, received an incorrect dose, or had insufficient data. The authors reported no significant differences in outcome as

assessed by the Frankel scale at the time of hospital discharge when 188 patients who received protocol methylprednisolone (appropriate dose and timing) were compared with those ( $n = 90$ ) who did not receive any methylprednisolone during treatment. This was true for the combined population of patients and for both the 1990 to 1991 and the 1993 patient populations. It does not seem, however, that adequate numbers of patients were analyzed by the authors, substantially diluting the statistical power of their findings.

In 1995, George et al (28) reported their experience with ASCI patients at Michigan State University from 1989 through 1992. One hundred forty-five patients were described, 80 of whom were treated with methylprednisolone per the NASCIS II protocol (MP group) and 65 of whom did not receive methylprednisolone (No-MP group). Admission, discharge, and follow-up neurological assessments were accomplished according to the FIM instrument. Fifteen patients were excluded from review, leaving 130 patients (85 MP, 55 No-MP). The MP group was significantly younger than the No-MP group (30 yr versus 38 yr,  $P < 0.05$ ). Although the mean trauma scores were similar between the two groups, the MP patients had a significantly lower injury severity score (ISS) than the No-MP patients ( $P < 0.05$ ). The authors found no differences in mortality or neurological outcome between patients treated with methylprednisolone and those who were not. Despite older age and higher injury severity score, the No-MP group had better mobility at the time of hospital discharge. Admission mobility scores were similar (MP = 5.99 versus No-MP = 5.90), but the mobility scores differed significantly on hospital discharge (MP = 5.16 versus No-MP = 4.67,  $P < 0.05$ ). The authors argued that the MP patient group had a more favorable opportunity for improvement than the No-MP patient group owing to younger age and lower ISS scores; however, neurological improvements in the MP group compared with the No-MP group were not observed. It is unclear from the study why most patients did not receive corticosteroid therapy, and this is the weakness of a nonrandomized study in which patient assignment to treatment may introduce bias. For example, an examination of the data indicates that the worst neurologically injured patients at admission were more likely to have received methylprednisolone. The findings of no difference in neurological examination improvement or functional recovery in this group seem to refute the findings of neurological improvement in NASCIS II patients who received methylprednisolone less than 8 hours after injury compared with those who did not receive the drug.

Gerndt et al (30), in 1997, reported a retrospective review of 231 patients with ASCI for the purpose of examining medical complications. Ninety-one patients were excluded because they received corticosteroids outside the NASCIS II protocol. One hundred forty patients were reviewed, comparing 93 patients who received methylprednisolone per the NASCIS II protocol with a historical control group of 47 patients who received no corticosteroid during treatment. The patient groups were similar with respect to age and injury severity. The authors found significant differences (increases) in the incidence of pneumonia ( $P = 0.02$ , 2.6-fold increase), particularly acute pneumonia ( $P = 0.03$ , 4-fold increase), ventilated days ( $P = 0.04$ ), and ICU length of stay ( $P = 0.045$ ) in methylprednisolone-treated patients compared with those who did not receive corticosteroids during treatment. Non-corticosteroid-treated patients had a higher incidence of urinary tract infections ( $P = 0.01$ ). Methylprednisolone-treated patients had decreased general care floor length of stay ( $P = 0.02$ ) and rehabilitation length of stay ( $P = 0.035$ ). The authors concluded that methylprednisolone may increase the incidence of early infection, particularly pneumonia, in ASCI patients but has no adverse effect on long-term outcome. In 1997, Poynton et al (39) described 71 consecutive ASCI patients managed at the National Spinal Trauma Unit in Dublin, Ireland. They attempted a case-control analysis of ASCI patients treated with methylprednisolone ( $n = 38$ ) compared with patients who did not receive methylprednisolone ( $n = 25$ ) and provided follow-up from 13 months to 57 months after injury. Patients who did not receive methylprednisolone were referred more than 8 hours after injury. The authors concluded that multiple factors influenced outcome after ASCI. They found no difference in neurological outcome when they compared patients who received methylprednisolone with those who did not.

The results of the third NASCIS study (NASCIS III) were published in 1997 (16). NASCIS III was a double-blind randomized clinical trial comparing the efficacy of methylprednisolone administered for 24 hours with that of methylprednisolone administered for 48 hours. There was no placebo group. Entry criteria were similar to those described for NASCIS II study patients. Patients were assessed neurologically according to NASCIS I and II (change in motor and sensory scores) and by change in FIM at 6 weeks and 6 months. Four hundred ninety-nine patients were entered into the study, 166 in the 24-hour methylprednisolone group (24 MP), 167 in the 48-hour tirilazad mesylate group (48 TM), and 166 in the 48-hour methylprednisolone group (48MP). The authors reported that patients in the 48 MP group showed improved motor recovery at 6 weeks ( $P = 0.09$ ) and at 6 months ( $P = 0.07$ ) follow-up compared with 24 MP patients and 48 TM patients. When therapy was initiated between 3 and 8 hours after injury, the effect of the 48 MP regimen on change in motor score was significant at 6 weeks ( $P = 0.04$ ) and at 6 months ( $P = 0.01$ ) follow-up compared with patients in the 24 MP and 48 TM treatment groups. 48MP patients had more improvement in FIM at the 6-month follow-up ( $P = 0.08$ ) compared with patients in the other two treatment groups. 48MP treatment patients also had higher rates of severe sepsis ( $P = 0.07$ ) and severe pneumonia ( $P = 0.02$ ). When treatment was initiated within 3 hours of injury, the same recovery pattern was observed in all three treatment groups. The authors concluded that patients with ASCI who receive methylprednisolone within 3 hours of injury should be maintained on the 24 MP regimen. When methylprednisolone is administered 3 to 8 hours after injury, they recommended the 48 MP regimen.

In 1998, the 1-year follow-up results of the NASCIS III trial were reported (17). The authors reported that for patients treated within 3 hours of injury, recovery rates at 1 year were equal in all three treatment groups. For patients treated between 3 and 8 hours after injury, 24 MP patients had diminished motor recovery and 48 MP patients had increased motor recovery at 1 year ( $P = 0.053$ ). They noted no significant difference in functional outcome as measured by FIM in any treatment group. The authors concluded that if methylprednisolone is administered to patients with ASCI within 3 hours of injury, 24-hour maintenance is recommended. If methylprednisolone is administered 3 to 8 hours after injury, they recommended that a 48-hour maintenance regimen be followed. These final recommendations seem to be based on motor recovery score improvement alone ( $P = 0.053$ ).

Predominant criticisms of the NASCIS III study and the reported results focus on three major issues: determination of optimum timing of therapy, method of motor assessment of SCI patients, and insignificant differences in motor recovery scores and functional outcome measures among study patients (18,19,32,33,37,51). For optimum timing of therapy, time-to-treatment data were not offered or explained. Like the 8-hour time for treatment cutoff “result” that came from the NASCIS II study, the “within 3 hours of injury” versus the “3 to 8 hours after injury” timeframes reported in NASCIS III seem arbitrary (18,32,37). It is not intuitive or likely that the 3-hour treatment time is an “all or nothing” time period supported by physiological evidence. With respect to the method of motor assessment and reporting, like the NASCIS II study, NASCIS III motor scores were reported as change in motor scores from the right side of the body. Left-side motor scores and total body motor scores were not provided. The failure to provide this study’s scientific evidence (particularly in light of the NASCIS I and II criticisms) suggests that the changes in right-side only motor scores are the only findings that approach significance at 1 year ( $P = 0/053$ ) and argue against the meaningful nature of the data as interpreted and provided by the authors (18,32,37). Finally, the clinical significance of the changes in motor scores between groups, in light of the non-significant differences in patient function as determined by FIM scores, is not evident. NASCIS III patients who received 48 MP treatment had a 2-fold higher incidence of severe pneumonia, a 4-fold higher incidence of severe sepsis, and a 6-fold higher incidence of death due to respiratory complications than patients in the 24 MP treatment group (8,32). These differences, although not statistically significant, raise questions about the safety of the 48-hour treatment strategy proposed for patients with ASCI treated within 3 to 8 hours of injury. Additional important criticisms of the NASCIS III trial include those levied against both the NASCIS I and II studies (i.e., lack of standardized medical treatment, lack of a minimum motor impairment for inclusion [hence, normal motor

function patients admitted to the study], no vertebral level of injury cutoff, and unclear statistical methodology, analysis, and data interpretation) (18,32,37). NASCIS III was designed and implemented to be a randomized, double-blind clinical study in an attempt to generate Class I evidence on the efficacy of methylprednisolone, offered in two different treatment regimens, and tirilazad mesylate after ASCI in human subjects. The absence of evidence for functional improvement in any group argues against the clinical relevance of any of these regimens.

Wing, et al (49) examined the effect of methylprednisolone administered per the NASCIS II protocol on avascular necrosis (AVN) of the femoral heads of 91 ASCI patients, 59 who received the corticosteroid, and 32 who did not. The authors found no case of AVN in their study population and estimate the relative risk of AVN with high-dose 24-hour methylprednisolone therapy to be less than 5%.

In 2002, Pointillart et al (38) reported the results of a prospective, randomized clinical trial designed to evaluate the safety and effect of nimodipine, methylprednisolone, or both versus no pharmacological therapy in 106 ASCI patients. Patients were randomly assigned to one of four treatment groups, methylprednisolone per NASCIS II protocol (M), nimodipine (N), both methylprednisolone and nimodipine (MN), and neither medication (P). Blinded neurological assessment was accomplished via the American Spinal Cord Injury Association (ASIA) score at initiation of treatment and at 1-year follow-up. The authors performed early spinal decompression and stabilization as indicated. One hundred patients were available at 1-year follow-up. There was no significant difference in outcome among the four treatment groups for any of the ASIA scores recorded. Patients in all four treatment groups demonstrated significant neurological improvement at the 1-year follow-up compared with admission ( $P < 0.0001$ ). Two-way analysis of variance revealed no interaction between methylprednisolone and nimodipine. There was a significant difference in recovery below the level of injury among patients with complete SCIs compared with those with incomplete injuries ( $P < 0.0001$ ). Improvement among complete injury patients when present, involved the level of the lesion and the two adjacent caudal levels. The greatest neurological improvements were identified in incomplete injury patients. There was no significant difference in neurological outcome for patients who underwent surgery within 8 hours of injury, patients treated surgically between 8 and 24 hours after injury, and those managed without surgery. The incidence of infectious complications was higher among the patients treated with methylprednisolone compared with those who did not receive corticosteroids (66% versus 45%), but this difference was not significant. The authors concluded that pharmacological therapy offered no added benefits to patients with ASCIs. Unfortunately, sample size calculations are not provided by the authors, and therefore the statistical power of the study to show a significant benefit of the treatment(s) is unknown. In addition, indications for surgery and for timing of surgery were not provided, potentially adding bias. The failure to show a difference between groups in this study may be explained by these potential study design flaws.

A number of published critiques of the NASCIS data and their presentation in support of the use of methylprednisolone in the management of patients with ASCI have been offered (1,18,19,22,31-33a,35,37,40-42,44-46,51). A recent medical evidence-based review is provided by Short et al (46). These authors conclude, after review of the medical literature on the use of methylprednisolone for ASCI (animal and human experimental studies, including randomized human clinical trials), that the available evidence does not support the use of methylprednisolone in the treatment of ASCI. A number of reviews that support the use of methylprednisolone after ASCI have also been published, including a Cochrane Database of Systematic Reviews (3-5,6a,7,9,10,50).

In 2001, Matsumoto et al (36) reported their results of a prospective, randomized double-blind clinical trial comparing methylprednisolone with placebo in the treatment of patients with acute cervical SCI. The authors focused on potential medical complications after ASCI. Forty-six patients were included in the study: 23 treated with methylprednisolone per the NASCIS II protocol were compared

with 23 patients in a placebo treatment group. Complications associated with therapy were noted at 2-month follow-up. Patients treated with methylprednisolone had a higher incidence of complications compared with placebo-treated patients (56.5% versus 34.8%). Respiratory complications ( $P = 0.009$ ) and gastrointestinal complications ( $P = 0.036$ ) were the most significant between the two treatment populations. The authors concluded that patients with ASCI treated with methylprednisolone (particularly older patients) are at increased risk for pulmonary and gastrointestinal complications and deserve special care. This incidence of medical complications using methylprednisolone for 24 hours seems clinically important. The NASCIS III study demonstrated that these complications are even higher for 48-hour methylprednisolone administration as described above (17). This calls into question the use of corticosteroids for any timeframe, but especially for the 48-hour duration.

Finally, a review of the data in a large number of patients in the most recent GM-1 ganglioside trial who had methylprednisolone alone according to NASCIS II and III protocols did not confirm the findings of the NASCIS II and III trials (23). This is described in detail in the section below on the GM-1 ganglioside trials.

In summary, the available medical evidence does not support a significant clinical benefit from the administration of methylprednisolone in the treatment of patients after ASCI for either 24 or 48 hours duration. Three North American, multicenter randomized clinical trials have been completed and several other studies have been accomplished addressing this issue (11,13-17,21,28,29,38,39). The neurological recovery benefit of methylprednisolone when administered within 8 hours of ASCI has been suggested but not convincingly proven. The administration of methylprednisolone for 24 hours has been associated with a significant increase in severe medical complications. This is even more striking for methylprednisolone administered for 48 hours. In light of the failure of clinical trials to convincingly demonstrate a significant clinic benefit of administration of methylprednisolone, in conjunction with the increased risks of medical complications associated with its use, methylprednisolone in the treatment of acute human SCI is recommended as an option that should only be undertaken with the knowledge that the evidence suggesting harmful side effects is more consistent than the suggestion of clinical benefit.

## **GM-1 Ganglioside**

GM-1 ganglioside has been evaluated in both animal and human studies of ASCI (2,26,27,47,48). In 1991, Geisler et al (25) described the results of a prospective, randomized placebo-controlled, double-blind trial of GM-1 ganglioside in the treatment of human patients with ASCI. Of 37 patients entered into the study, 34 were available for 1-year follow-up (16 GM-1 patients, 18 placebo). All patients received a 250-mg bolus of methylprednisolone and then 125 mg every 6 hours for 72 hours. GM-1 patients were administered 100 mg of GM-1 ganglioside per day for 18 to 32 days, with the first dose provided within 72 hours of injury. Neurological evaluation was accomplished with Frankel scale and ASIA motor score assessments. The authors reported that GM-1 ganglioside treated patients had significant improvements in the distribution of Frankel grades from baseline to 1-year follow-up ( $P = 0.034$ ) and significantly improved ASIA motor scores compared with placebo-treated patients ( $P = 0.047$ ) (26,27). The recovery of motor function in GM-1 ganglioside-treated patients was thought to be caused by recovery of strength in paralyzed muscles rather than strengthening of paretic muscles. There were no adverse effects attributed to the administration of the study drug. The authors concluded that GM-1 ganglioside enhances neurological recovery in human patients after SCI and deserves further study.

In 1992, a multicenter GM-1 ganglioside ASCI study was initiated. It was a prospective, double-blind randomized and stratified trial that enrolled 797 patients by study end in early 1997 (23). All patients received methylprednisolone per the NASCIS II protocol. Patients were randomized into three initial study groups: placebo, low-dose GM-1 (300-mg loading dose and then 100 mg/d for 56 d), and

high-dose GM-1 (600-mg loading dose and then 200 mg/d for 56 d). Placebo or GM-1 was administered at the conclusion of the 23-hour methylprednisolone infusion. Patients were assessed using the modified Benzel Classification and the ASIA motor and sensory examinations a 4, 8, 16, 26, and 52 weeks after injury. Aggressive medical and surgical management paradigms were used. Patients had to have an acute, nonpenetrating SCI (anatomic vertebral level C2 through T11) of at least moderate severity (no neurologically normal or nearly normal patients). The primary efficacy assessment was the proportion of patients who improved at least two grades from baseline examination (defined as “marked recovery”), at Week 26 of the study. Secondary efficacy assessments included the time course of marked recovery, the ASIA motor score, and ASIA sensory evaluations, relative and absolute sensory levels of impairment, and assessments of bladder and bowel function. A planned interim analysis of the first 180 patients resulted in the addition of stratification by patient age and discontinuation of the high-dose GM-1 treatment strategy because of an early trend for higher mortality. At the study conclusion, 37 patients were judged ineligible, leaving 760 patients for primary efficacy analysis. The authors found no significant difference in mortality between treatment groups (23). The authors did not identify a higher proportion of patients with marked recovery in motor function at 26 weeks when they compared GM-1 treated patients to the placebo treatment group in their primary efficacy analysis. The time course of recovery indicated earlier attainment of marked recovery in GM-1-treated patients. The authors concluded that, despite the lack of statistical significance in the primary analysis, numerous positive secondary analyses indicate that GM-1 ganglioside is a useful drug in the management of ASCI (23). The placebo group within this study of GM-1 represents a group of 322 patients who received methylprednisolone within 8 hours of injury. Of interest, these 322 patients (measured in a similar, albeit, more detailed manner as NASCIS II patients) did not demonstrate the previously published neurological examination improvement found in 62 NASCIS II patients treated within the same timeframe (13,14). Similarly, 218 of these patients received 24 hours methylprednisolone treatment within 3 hours of injury, as suggested in NASCIS III, and did not show the same neurological examination motor improvement as the 75 NASCIS III patients who received the same regimen (16,17). The authors could not confirm the NASCIS findings that timing of methylprednisolone therapy had an impact on spinal cord recovery. This further brings into question the conclusions of the NASCIS II and III methylprednisolone trials.

In summary, the available medical evidence does not support a significant clinical benefit from the administration of GM-1 ganglioside in the treatment of patients after ASCI. Two North American multicenter, randomized clinical trials have been completed addressing this issue (23,29). The neurological recovery benefit of GM-1 ganglioside when administered for 56 days after the administration of methylprednisolone within 8 hours of ASCI has been suggested but not convincingly proven. At present, GM-1 ganglioside (a 300-mg loading dose and then 100 mg/d for 56 d), when initiated after the administration of methylprednisolone given within 8 hours of injury (NASCIS II protocol), is recommended as an option in the treatment of adult patients with ASCI.

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

Given the problems associated with the many trials attempting to answer the questions surrounding the use of pharmacological agents in acute spinal cord-injured patients, it is clear that more research is required. Issues such as adequate numbers of patients to achieve statistical power, a placebo group as one of the treatment arms, standardized medical and surgical protocols to diminish bias, careful collection of relevant outcome data, especially functional outcomes, and appropriate statistical analyses need to be further addressed a priori. Research into all potentially promising pharmacological agents, including, but not limited to, tirilazad mesylate, naloxone, methylprednisolone, and GM-1 should be undertaken.

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**TABLE 9.1 Summary of Reports on Treatment with Methylprednisolone after Acute Cervical Spinal Cord Injury\***

Series (Ref No)	Description of Study	Evidence Class	Conclusions
Bracken et al, 1984 (11)	Multicenter, double-blind randomized trial comparing MP (2000 mg/d versus 100 mg/d for 11 d) in treatment of 330 ASCI patients (NASCIS I study).	III (study design, data presentation, interpretation and analysis flaws)	No treatment effect at 6 wk and 6 mo post-injury. No control group.
Bracken et al, 1985 (15)	1-yr follow-up of NASCIS I study.	III (study design, data presentation, interpretation and analysis flaws)	No significant difference in neurological recovery of motor or sensory function 1-yr post-injury.
Bracken et al, 1990 (14)	Multicenter, randomized, double-blind, placebo-controlled trial comparing MP with naloxone and placebo in treatment of 487 ASCI patients (NASCIS II study).	III (study design, data presentation, interpretation and analysis flaws)	Significant improvement in motor change scores ( $P = 0.03$ ), and sensation change scores ( $P = 0.02$ ) at 6 mo post-injury for patients treated with MP within 8 h of injury.
Bracken et al, 1992 (13)	1-yr follow-up of NASCIS II study.	III (study design, data presentation, interpretation and analysis flaws)	Significant improvement in motor changes scores 1 year post-injury for patients treated with MP within 8 h of injury ( $P = 0.03$ ). Administration of MP detrimental if given more than 8 h after injury.
Galandiuk et al, 1993 (21)	Prospective assessment of 15 patients from 1990 to 1993 with retrospective review of 17 patients from 1987 to 1990 to assess differences in treatment outcome with MP compared with treatment without corticosteroids.	III	No difference in neurological outcome between two sets of patients. MP patients had immune response alterations, higher rate of pneumonia, and longer hospital stays than patients who did not receive corticosteroids.
Gerhart et al, 1995 (29)	Concurrent cohort comparison study (population-based) of 363 ASCI patients managed from 1990 to 1991 and 1993. 188 patients managed with NASCIS II MP compared with 90 patients with no MP.	III (Inadequate statistical power)	No differences in neurological outcome using Frankel classification between MP and No-MP patients. However, may be insufficient numbers of patients to show significant differences.
George et al, 1995 (28)	Retrospective review of 145 ASCI patients, 80 treated with MP compared with 65 who did not receive MP.	III	No difference in mortality or neurological outcome between groups despite younger age, less severe injury in MP-treated patients.
Gerndt et al, 1997 (30)	Retrospective review with historical control of 231 ASCI patients, 91 excluded. Comparison of medical complications among 93 MP patients compared with 47 who received no corticosteroid.	III	MP-treated patients had significant increases in pneumonia ( $P = 0.02$ ), acute pneumonia ( $P = 0.03$ ), ventilated days ( $P = 0.04$ ), and ICU stay ( $P = 0.45$ ), but no adverse effect on long-term outcome.
Poynton et al, 1997 (39)	Case-control analysis of 71 consecutive ASCI admissions. 63 available for 13 mo to 57 mo follow-up. 38 patients treated with MP compared with 25 referred > 8 hr after injury who received no MP.	III	Multiple factors influence recovery after SCI. No effect of MP or surgery on outcome.

Series (Ref No)	Description of Study	Evidence Class	Conclusions
Bracken et al, 1997 (16)	Multicenter, randomized double-blind trial comparing MP administered for 24 hr to MP administered 48 hr and TM in the treatment of 499 ASCI patients (NASCIS III study).	III (study design, data presentation, interpretation and analysis flaws)	48 MP patients had improved motor recovery at 6 wk and at 6 mo compared with 24 MP and 48 TM groups NS. When treatment initiated between 3 h and 8 h after injury, 48 MP had significant improvement of motor scores at 6 wk (P = 0.04) and 6 mo (P = 0.01). 48 MP was associated with high rates of sepsis and pneumonia. No control group.
Bracken et al, 1997 (17)	1-yr follow-up of NASCIS III study.	III (study design, data presentation, interpretation and analysis flaws)	Recovery rates equal in all 3 groups when treatment initiated within 3 h of injury. When treatment initiated between 3 h and 8 h, 24 MP patients had diminished recovery, 48 MP patients had increased motor recovery (P = 0.053).
Pointillart et al, 2000 (38)	Multicenter, prospective, randomized clinical trial of 106 ASCI patients treated with MP, nimodipine, neither, or both.	III (Inadequate statistical power)	No significant difference in neurological outcome at 1-yr follow-up between groups. Incomplete ASCI had significant improvement below level of injury compared to complete patients (P < 0.0001). Higher incidence of infectious complications among patients receiving corticosteroids (NS).
Matsumoto et al, 2001 (36)	Prospective randomized, double-blind study comparing incidence of medical complications among 46 ASCI patients, 23 treated with MP, 23 with placebo.	I	MP patients had higher incidence of complications (56.5% versus 34.8%). Respiratory complications (P = 0.009) and gastrointestinal bleed (P = 0.036) were most significant between groups. No data on neurological improvement.

\*ASCI, acute spinal cord injury; NASCIS National Acute Spinal Cord Injury Study; MP, methylprednisolone; ICU, intensive care unit; SCI, spinal cord injury; TM tirilazad mesylate; NS, not significant.

**Table 9.2 Summary of Reports on Treatment with GM-1 Ganglioside after Acute Spinal Cord Injury.**

Series (Ref No)	Description of Study	Evidence Class	Conclusions
Geisler et al, 1991 (25)	Prospective, randomized, double-blind trial of GM-1 ganglioside in 37 human ASCI patients. All received 250-mg MP bolus followed by 125 mg ever 6 h x 72 h before randomization (placebo group)	I	GM-1 ganglioside enhances recovery of neurological function, significant difference in recovery compared with MP group (P = 0.047). Insufficient numbers of patients to draw meaningful conclusions. No true placebo group.
Geisler et al, 2001 (23)	Prospective, randomized, double-blind stratified multicenter trial of GM-1 ganglioside in 760 ASCI patients. All received MP per NASCIS II protocol (placebo group).	I	No significant differences in neurological recovery identified between GM-1 treated patients and MP-treated patients at 26-wk follow-up. Trend for earlier recovery in GM-1-treated patients. No true placebo group.

\*ASCI, acute spinal cord injury; NASCIS National Acute Spinal Cord Injury Study; MP, methylprednisolone

## DEEP VEIN THROMBOSIS AND THROMBOEMBOLISM IN PATIENTS WITH CERVICAL SPINAL CORD INJURIES

### RECOMMENDATIONS

Standards: • Prophylactic treatment of thromboembolism in patients with severe motor deficits due to spinal cord injury is recommended.

• The use of low molecular weight heparins, rotating beds, adjusted dose heparin, or a combination of modalities is recommended as a prophylactic treatment strategy.

• Low dose heparin in combination with pneumatic compression stockings or electrical stimulation is recommended as a prophylactic treatment strategy.

Guidelines: • Low dose heparin therapy alone is not recommended as a prophylactic treatment strategy.

• Oral anticoagulation alone is not recommended as a prophylactic treatment strategy.

Options: • Duplex Doppler ultrasound, impedance plethysmography, and venography are recommended for use as diagnostic tests for DVT in the spinal cord injured patient population.

• A three-month duration of prophylactic treatment for DVT and PE is recommended.

• Vena cava filters are recommended for patients who fail anticoagulation or who are not candidates for anticoagulation and/or mechanical devices.

## **RATIONALE**

Deep venous thrombosis (DVT) and pulmonary embolism (PE) are problems frequently encountered in patients who have sustained cervical spinal cord injuries. Several means of prophylaxis and treatment are available including anticoagulation, pneumatic compression devices, and vena cava filters. The purpose of this evidence-based medicine review is to evaluate the literature on the methods of prevention and identification of DVT and PE complications in patients following acute cervical spinal cord injury.

## **SEARCH CRITERIA**

A National Library of Medicine computerized literature search from 1966 through 2001 was performed using Medical Subject Headings in combination with “spinal cord injury”: “deep venous thrombosis” “pulmonary embolism” and “thromboembolism.” The search was limited to human studies in the English language. This resulted in 129 citations. Duplicate references, reviews, letters, and tangential reports were discarded. Thirty-seven papers dealing with the prophylaxis or treatment of thromboembolic disease in adult spinal cord injured patients make up the basis for this guideline and are summarized in Evidentiary Table format. Supporting references included four evidence-based reviews published by various organizations concerned with thromboembolism prophylaxis and treatment in a variety of patient populations. Finally, several series dealing with thromboembolism in general trauma patients with results germane to a discussion of spinal cord injured patients are included in the bibliography as supporting documents.

## **SCIENTIFIC FOUNDATION**

The incidence of thromboembolic complications in the untreated spinal cord injury (SCI) patient population is high. Depending upon injury severity, patient age, and the methods used to diagnose a thromboembolism, the incidence of thromboembolic events ranges from 7% to 100% in reported series of patients receiving either no prophylaxis or inadequate prophylaxis. (3,8,10,13,16,22,23,26,27,29,30,33,36)) Substantial morbidity and mortality has been associated with the occurrence of DVT and PE events in the SCI patient population. (6,11)

### **Prophylaxis**

Prophylactic therapy has been shown to be effective for the prevention of DVT and PE. In a small randomized study, Becker et al demonstrated that the use of rotating beds during the first 10 days following SCI decreased the incidence of DVT. Four of five control patients were diagnosed with DVT (by fibrinogen screening) compared to one of ten treated patients. (2) The use of low dose heparin (5000 units given via subcutaneous injection twice or three times daily) has been described by several authors. (4,8,16,17,22,30,37) Hachen published the results of a retrospective historical comparison of low dose heparin versus oral anticoagulation in a group of 120 SCI patients. He found a lower incidence of thromboembolic events in the low dose heparin group compared to the oral anticoagulation group.(17) In 1977, Casas et al reported the results of a prospective assessment of low dose heparin in SCI patients. They administered heparin for a mean period of 66 days in 18 SCI patients and noted no thromboembolic events as detected by clinical examination.(4) Watson reported a lower incidence of thromboembolic events with the use of low dose heparin when compared to no prophylaxis in a retrospective historical cohort study.(37) Frisbie and Sasahara however, found that low dose heparin did not affect the incidence of DVT in a prospective study of 32 SCI patients compared to treatment with twice

daily physical therapy alone. These authors felt that the lack of effect was due to the very low incidence of DVT in their control group compared to other series because of the aggressive physical therapy paradigm employed in their patients. Although they performed venous occlusion plethysmography screening (VOP) with confirmatory venography weekly, the incidence of DVT was only 7% in both groups. (8) An identical observed frequency of DVT in both treatment groups cannot be explained by anything other than that the treatments were equivalent in this study. This incidence is substantially lower than that reported by two separate groups of investigators a decade later. In 1992, Kulkarni et al reported a much higher incidence of DVT (26%) and of PE (9%) in a group of 100 SCI patients prospectively treated with low-dose heparin.(22) In 1993, Gunduz et al reported a 53% incidence of DVT confirmed by venography in 31 patients they managed with SCI treated with low dose heparin.(16) In a study published in 1999, Powell et al noted that the incidence of DVT in 189 SCI patients receiving prophylaxis was significantly lower than that identified in SCI patients who did not receive prophylaxis, 4.1% vs. 16.4%. They found, in addition, that DVT in the prophylaxis group occurred in patients who received low dose heparin alone.(30)

Several studies have demonstrated that better prophylactic therapies than low dose heparin exist.(13,25,26) Green et al published a randomized controlled study comparing low dose versus adjusted dose heparin (dose adjusted to APTT 1.5 times normal) in SCI patients.(13) They found that patients treated with adjusted dose heparin had fewer thromboembolic events (7% versus 31%) during the course of their ten-week study, but had a higher incidence of bleeding complications. Merli et al in 1988 reported their findings of the additive protective effects of electrical stimulation in combination with low dose heparin, heparin alone, and placebo in 48 SCI patients treated for four weeks duration. In this Class I prospective,

randomized trial, they found that the heparin therapy alone group had a similar incidence of DVT compared to the placebo group. The combination of low dose heparin and electrical stimulation significantly decreased the incidence of DVT (one of fifteen patients compared to the other two treatment groups (eight of 16 low dose heparin alone and eight of 17 placebo,  $p < 0.05$ ).(26) In 1992, this same group reported that heparin in combination with pneumatic stockings was equal to the effectiveness of heparin plus electrical stimulation. The heparin in combination with electrical stimulation group and the placebo group for this comparison were a historical cohort. This constitutes Class III evidence because the comparison group is a historical one. (25) Winemiller et al studied a large series of 428 SCI patients with a multivariate analysis and found that the use of pneumatic compression devices for six weeks duration was associated with a significant decrease in thromboembolic events (odds ratio of 0.5 (95% CI 0.28-0.90)).(40) Low dose heparin treatment seemed to have a protective effect as well, however the effect of heparin alone was not statistically significant.

Recently, low molecular weight heparins (LMWH) have been studied as prophylactic therapy for thromboembolism in SCI patients. Green et al treated a series of SCI patients with eight weeks of LMWH (tinzaparin) and compared the results with a historical cohort of patients treated with low dose or adjusted dose heparin.(12) They found that the use of LMWH compared favorably with the use of either heparin dosing regimen in terms of fewer thromboembolic events (16 of 79 in heparin group versus 7 of 68 in LMWH group,  $p = 0.15$ ) and a significant decrease in bleeding complications (nine of 79 in heparin group versus one of 68 in LMWH group,  $p = 0.04$ ).(12) More recently, Harris et al performed a retrospective study of LMWH (enoxaparin) administration in a series of 105 patients with spinal injuries. Forty of their 105 patients suffered neurologically complete injuries. No patient exhibited clinical or

ultrasound evidence of DVT and no patient suffered a PE treated with LMWH.(18) Roussi et al reported a 9% incidence of DVT in a study involving 69 SCI patients receiving LMWH, testimony to the fact that no prophylactic therapy is 100% effective.(32)

The use of inferior vena cava (IVC) filters as prophylactic devices for thromboembolism has been advocated.(19,20,31,39) Wilson et al placed caval filters in 15 SCI patients who were concurrently treated with either low dose heparin or pneumatic stockings. None suffered a PE during a one-year follow-up period. The one-year patency rate of the IVC was 81%.(39) These authors reported that their results are superior to those from a historical cohort of 111 patients treated without IVC filters. Seven of the cohort patients suffered a PE, however six of the seven were not receiving any prophylaxis at the time of their PE. The patient who was receiving DVT prophylaxis suffered a gunshot wound to the spine.(39) Khansarina and colleagues described a historical cohort study of 108 general trauma patients treated with prophylactic IVC filter placement. None of these patients suffered a PE. They compared this group to a historical cohort of 216 patients treated (apparently) with either low dose heparin or pneumatic compression devices prior to the use of IVC filters. Thirteen of these 216 suffered PE, nine were fatal. (20) The overall mortality of the filter group was lower than the control group, but this difference was not significant (16% vs. 22%).(20) Tola and colleagues have shown that percutaneous IVC filter placement in the ICU setting is as safe and is less costly than IVC filter placement in the operating room or the invasive radiology suite.(35) These authors suggest that IVC interruption is an effective means to prevent PE. Placement of filters is not without complications. Balshi et al, Kinney et al, and others have described distal migration, intraperitoneal erosion, and symptomatic IVC occlusion in patients with SCI treated with IVC filters.(1,15,21) Balshi et al have hypothesized that quadriplegic patients are at higher risk for

complications from IVC filter placement due to loss of abdominal muscle tone, as well as the use of the “quad cough” maneuver.(1) There has been no study performed to date comparing the use of prophylactic IVC filters to the use of modern methods of pharmacologic PE and DVT prophylaxis.

### **Duration of Prophylaxis**

The vast majority of thromboembolic events appear to occur within the first 2-3 months following injury. Naso described his experience with four patients with PE in a group of 43 SCI patients. All four PE events were documented within three months injury. (28) Perkash et al reported an 18% incidence of thromboembolism in a series of 48 patients with acute spinal cord injury and two patients with transverse myelitis. Only one patient had a new onset PE three months after injury; two other patients had recurrent PE three months after injury due to existing DVT. (29) Lamb et al described a series of 287 SCI patients. The overall risk of thromboembolic events in their patient population was 10%. The vast majority of events occurred within the first six months following injury. Twenty-two of 28 events they documented occurred within the first three months of injury.(23) El Masri and colleagues reported 21 documented events of PE in a series of 102 spinal injured patients. Twenty of twenty-one events occurred within the first three months following SCI. A pulmonary embolism occurred in a patient with a history of PE whose therapeutic anticoagulation was discontinued for gallbladder surgery.(7) DeVivo et al found a 500-fold risk of dying from PE in the first month following SCI compared to age- and gender-matched non-injured patients. This risk decreased with time, however remained approximately 20 times greater than that for normative controls six months following injury.(6) McKinley et al studied chronic spinal injured patients in a rehabilitation center setting and found an incidence of DVT of 2.1% in the first year following injury. This

incidence dropped to between 0.5% and 1% per year thereafter.(24) Based upon these data, it is apparent that the great majority of thromboembolic events (DVT and PE) occur within three months of acute spinal injury. Although late thromboembolic events can occur, the risk of these events must be balanced against the cost and risks of indefinite anticoagulation. Prolonged prophylactic anticoagulation therapy is not without risk, and is associated with bleeding complications.(12,13) The vast majority of studies addressing prophylactic treatment for DVT and PE have utilized treatment courses of eight to 12 weeks duration with success. For these reasons, it is recommended that prophylactic treatment be continued for eight to 12 weeks in spinal cord injury patients without other major risk factors for DVT and PE (previous thromboembolic events, obesity, advanced age). Prophylactic treatment may be discontinued earlier in patients with useful motor function in the lower extremities, as these patients appear to be at less risk for DVT and PE. (11,27)

## **Diagnosis**

The diagnosis of DVT in various studies has been made based on clinical criteria, Doppler ultrasound examination, venous occlusion plethysmography (VOP), venography, fibrinogen scanning or by D-Dimer measurement. (2-4,7-10,13,14,16,17,22,26-30,32,34,36-38,41) Although venography may be considered a “gold standard” examination for DVT, venography is not possible in all patients, is invasive, and expensive.(10) Gunduz and colleagues report a 10% incidence of adverse effects from venography including post-venographic phlebitis and allergic reactions.(16) Doppler ultrasound examination and VOP are both less invasive, less expensive, and more broadly applicable.(10,30) The sensitivity and specificity of these examinations when compared with venography has been generally reported

to range from 80% to 100%.<sup>(5)</sup> Chu et al compared Doppler ultrasound and VOP with the clinical examination and found all three to agree 100% of the time in a small series of 21 patients. <sup>(5)</sup> Perkash and colleagues studied a series of 48 SCI patients with daily physical examinations and weekly VOP. They found that the sensitivity of the clinical examination compared to VOP was 89%. The specificity was 88%, the negative predictive value was 97%, and the positive predictive value was 62% in their study.<sup>(29)</sup> Other authors have described the increased sensitivity of fibrinogen scanning and the use of D-Dimer measurements for the diagnosis of DVT.<sup>(32,34)</sup> Increased sensitivity is associated with decreased specificity. For example, Roussi et al reported 100% sensitivity and 100% negative predictive value with D-Dimer determinations compared to Doppler ultrasound and the clinical examination. However, the specificity of D-Dimer was only 34%, and the positive predictive value was only 13%.<sup>(32)</sup> Similarly, Todd et al found that fibrinogen scanning was positive in all 20 patients studied in a prospective fashion, yet the diagnosis of DVT was confirmed by another test in only half of the cases.<sup>(34)</sup> Overall, no single test is completely applicable, accurate, and sensitive for the detection of DVT in the SCI patient population. Furthermore, a substantial number of patients who suffer from PE are found to have negative lower extremity venograms.<sup>(7,10)</sup> The Consortium for Spinal Cord Medicine has recommended the use of ultrasound for the study of patients with suspected DVT, and venography when clinical suspicion is strong and the ultrasound examination is negative.<sup>(11)</sup> Based upon the medical evidence available, these recommendations appear to be sound.

## **SUMMARY**

Thromboembolic disease is a common occurrence in patients who have sustained a cervical spinal cord injury and is associated with significant morbidity. Class I medical evidence exists demonstrating the efficacy of several means of prophylaxis for the prevention of thromboembolic events. Therefore, patients with SCI should be treated with a regimen aimed at prophylaxis.

Although low dose heparin therapy has been reported to be effective as prophylaxis for thromboembolism in several Class III studies, other Class I, Class II, and Class III medical evidence indicates that better alternatives than low dose heparin therapy exist. These alternatives include the use of low molecular weight heparin, adjusted dose heparin, or anticoagulation in conjunction with pneumatic compression devices or electrical stimulation. Oral anticoagulation alone does not appear to be as effective as these other measures used for prophylaxis.

The incidence of thromboembolic events appears to decrease over time and the prolonged use of anticoagulant therapy is associated with a definite incidence of bleeding complications. There are multiple reports of the beneficial effects of the prophylaxis therapy for six to twelve weeks following spinal cord injury. Very few thromboembolic events occur beyond three months following injury. For these reasons, it is recommended that prophylactic therapy be discontinued after three months unless the patient is at high risk (previous thromboembolic events, obesity, advanced age). It is reasonable to discontinue therapy earlier in patients with retained lower extremity motor function after spinal cord injury, as the incidence of thromboembolic events in these patients is substantially lower than those patients with motor complete injuries.

Caval filters appear to be efficacious for the prevention of PE in SCI patients. The relative efficacy of caval filters versus prophylactic combination therapy with LMWH and pneumatic compression stockings has not been studied. Caval filters are associated with long-term complications in SCI patients, although these complications are relatively rare. Caval filters are recommended for SCI patients who have suffered thromboembolic events despite anticoagulation and for SCI patients with contraindications to anticoagulation and/or the use of pneumatic compression devices.

There are several methods available for the diagnosis of DVT. Venography is considered the “gold standard,” but is invasive, not applicable to all patients, and associated with intrinsic morbidity. Duplex Doppler ultrasound and venous occlusion plethysmography have been reported to have sensitivities of approximately 90% and are non-invasive. It is reasonable to use these non-invasive tests for the diagnosis of DVT and to reserve venography for the rare situation when clinical suspicion is high and the results of VOP and ultrasound testing are negative.

## **KEY ISSUES FOR FUTURE INVESTIGATION**

Although thromboembolic events in the SCI patient are associated with significant morbidity, no study has demonstrated improved outcomes in SCI patients as a result of surveillance testing for them. A prospective study evaluating six-month outcomes in patients treated with prophylaxis with or without surveillance ultrasound imaging would be a substantial and potentially cost-saving contribution to the literature.

Caval filters appear to be effective in preventing PE and many institutions are using these devices as first-tier preventive therapy without trying other preventive measures. Caval filters

have not been compared to LMWH or combination therapy with anticoagulants and pneumatic compression devices for efficacy in the SCI patient population. As filters do appear to be associated with long-term morbidity in a fraction of SCI patients, a prospective study needs to be performed to establish whether the potential increase in protection against PE offsets the risks for long-term complications. A study comparing the use of vena caval filters prophylactically versus other modes of prevention with the use of filters placed only after failure of alternative methods should be instituted, including cost-effectiveness outcomes of each mode of prevention currently employed in spinal cord injured patients.

## EVIDENTIARY TABLE

First Author Reference	Description of Study	Data Class	Conclusions
Chen et al, Arch Phys Med and Rehab, 1999	Large population of SCI pats (1649) studied from admission to rehab (mean 19 days) to discharge (mean 50 days). Incidence of DVT + PE declining over time but remains 6.1% despite prophylaxis.	Class III	DVT/PE still problems despite prophylaxis. (See McKinley for follow-up)
McKinley et al, Arch Phys Med Rehabil, 1999	Chronic SCI population studied. Incidence of DVT highest during first year (2.1%) but then drops off to 0.5-1% per year thereafter.	Class III	Risk of DVT/PE highest during first year following injury and then risk drops significantly.
Powell et al, Arch Phys Med Rehab, 1999	Incidence of DVT in SCI population (n=189) on transfer to rehab (dx with ultrasound) was 4.1% in group who received prophylaxis vs. 16.4% in group without prophylaxis. In prophylaxis group, DVTs only occurred in pts receiving heparin alone.	Class II	Prophylaxis decreases incidence of DVT in SCI population. Heparin alone was the least effective measure.
Roussi et al, Spinal Cord, 1999	6/67 (9%) of SCI patients developed DVT despite prophylaxis with LMWH. D-Dimer had 100% negative predictive value compared to duplex Doppler. (However, specificity only 34% and PPV 13%)	Class I for diagnostic test, class III otherwise	Incidence of DVT despite prophylaxis with LMWH still 9%. D-Dimer is sensitive but not specific test for DVT
Winemiller et al, Journal of Spinal Cord Medicine, 1999	Retrospective study of 428 SCI patients. TE occurred in 19.6%. Compression stockings and sequential compression devices lowered risk of TE. Effects of low dose heparin were seen in first 14 days but were not significant. TEs all occurred in first 150 days.	Class III	SCD and stockings reduce risk of thromboembolism. Low dose heparin may be effective in first 14 days following injury.
Tomaio et al, Journal of Spinal Cord Medicine, 1998	Enoxaprin (LMWH) vs heparin use for initial DVT treatment in group of 6 SCI patients.	Class III	Enoxaprin was cost effective alternative to IV heparin for initial treatment of DVT
Harris et al, Am J of Phys Med and Rehab, 1996	Retrospective study of enoxaparin (LMWH) in 105 SCI pts. (1/3 intact, 40 complete). No clinical DVT/PE in 105, no ultrasound evidence in 60.	Class III	Enoxaparin is safe and effective for DVT prophylaxis in the SCI patient.
Khansarina et al, Journal of Vascular Surgery, 1995	Retrospective historical cohort comparison of prophylactic PGF in 324 general trauma patients. PGF group had fewer PE than control group.	Class III	Greenfield filter safe and effective for PE prophylaxis in general trauma population
Geerts et al, New England Journal of Medicine, 1994	Prospective evaluation of 716 trauma patients (no prophylaxis) with VOP and venography. Incidence of DVT in SCI population (N=66) was 62%	Class III	DVT is very common in SCI patients if no prophylaxis used
Wilson et al, Neurosurgery, 1994	Inserted Caval filters in 15 SCI patients. None had DVT or PE in 1 year. Claims this result superior to historical controls (No evidence presented to support this claim). One-year patency rate was 81%.	Class III	Insertion of caval filters appears to be safe in SCI patients.

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
Green et al, Archives of Physical Medicine and Rehabilitation, 1994	Historical cohort comparison of LMWH and standard and adjusted dose heparin prophylaxis. Trauma patients treated with 8 week course of LMWH had fewer bleeding episodes ( $p<0.05$ ) and thromboembolic complications ( $p=0.15$ ) than those treated with heparin.	Class III	LMWH may be safer and more effective for prophylaxis than mini dose or adjusted dose heparin
Gunduz et al, Paraplegia, 1993	31 SCI patients on low dose heparin therapy underwent venography. Incidence of DVT was 53.3%	Class III	Incidence of DVT high in SCI patients despite low dose heparin (therapy started on rehab unit)
Burns et al, Journal of Trauma, 1993	Prospective assessment of DVT in high risk trauma patients with US. Found incidence of 21% (12/57) despite low dose heparin or pneumatic boots in 85%.	Class III	DVT is common despite use of low dose heparin or pneumatic boots.
Lamb et al, J Am Paraplegia Soc, 1993	287 chronically injured SCI patients followed. Overall incidence of thromboembolic events was 10%, vast majority of events in first 6 months	Class III	Prophylactic therapy not necessary beyond 6 months in SCI population
Kulkarni et al, Paraplegia, 1992	100 SCI patients prospectively treated with low dose heparin. 26% incidence of clinically detected DVT (9% PE) noted	Class III	DVT and PE incidence significant despite low dose sq heparin
Merli et al, Paraplegia, 1992	Heparin plus pneumatic stockings equal to historical controls of heparin plus stimulation and better than historical controls of heparin or placebo in SCI patients.	Class II	Low dose heparin plus pneumatic hose safe effective as DVT prophylaxis in SCI patients.
Waring and Karunas, Paraplegia, 1991	Large database (1419) of SCI patients. Incidence of DVT was 14.5%, PE 4.6%. Severity of injury was a predictor of DVT and age was a predictor of PE. No mention made of prophylactic measures.	Class III	DVT and PE are significant problems in SCI population. Age and injury severity need to be addressed in studies comparing treatment modalities.
Yelnik et al, Paraplegia, 1991	Prospective study of 127 SCI patients with phlebography. 29/127 had DVT on admission to rehab unit. Of 87 patients with initially negative studies, 12 developed DVT despite prophylaxis for up to 80 days.	Class III	Incidence of DVT in SCI population is high and high risk period extends to end of third month. Authors recommend periodic screening with phlebography.
Balshi et al, Journal of Vascular Surgery, 1989	Case series of 13 quadriplegic patients who had vena caval filters placed for DVT or PE. Abnormalities of the filter were detected in 5/11 patients who had follow-up X-rays. Two patients required laparotomy to remove filters, four had distal migration, and two had narrowing of diameter associated with caval occlusion. Nine of these 11 patients were treated with the "quad cough" technique.	Class III	Filter placement may be associated with significant long-term morbidity in the SCI population, particularly those requiring aggressive pulmonary toilet.

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
DeVivo et al, Arch Intern Med, 1989	Epidemiological study of causes of death for SCI patients. Highest ratios of actual to expected causes of death were for pneumonia, PE, and septicemia. The risk ratio for TE dropped significantly after the first month post injury but remained elevated at 6 months post injury.	Class III	TE is a significant problem for patients who survive SCI. Biggest period of risk is in first few months following injury, but risk continues even after 6 months.
Green et al, JAMA, 1988	RCT of Low dose vs. adjusted dose heparin in SCI patients. Rate of TE lower in adjusted dose group (7% vs 31%) (intent to treat p=ns), but also had higher rate of bleeding complications (7 of 29).	Class I	Adjusted dose heparin more effective than low dose heparin, bleeding more common in adjusted dose group.
Merli et al, Arch Phys Med Rehabil, 1988	Prospective randomized trial of placebo vs. mini dose heparin vs. heparin plus electrical stimulation in group of 48 SCI patients. Heparin group=placebo group at 50%, stim group significantly fewer DVT	Class I	Low dose heparin no better than placebo, heparin plus electrical stimulation much better for DVT prophylaxis in SCI patients.
Weingarden et al, Paraplegia, 1988	Retrospective review of 148 SCI patients. Ten had documented DVT or PE. Of six patients who had adequate records, all 6 had fever as a presenting sign, 4 had no other clinical signs recorded. All episodes occurred in first 12 weeks.	Class III	Fever may indicate thromboembolic disease in SCI patients.
Becker et al, Neurosurgery, 1987	Randomized trial of rotating versus non-rotating beds in the acute setting following SCI (10 days) N=15 Plethysmography and fibrinogen leg scans used	Class I	Rotating beds reduce the incidence of DVT during the first 10 days following SCI
Tator, Canadian Journal of Neurological Sciences, 1987	17% incidence of DVT in series of 208 SCI patients. Incidence was higher in operated patients (23%) compared to non operated (10%). Use of prophylaxis is not mentioned.	Class III	Patients requiring surgery may have higher incidence of DVT.
Chu et al, Archives of Physical Medicine and Rehabilitation, 1985	Comparison between doppler US, Venous occlusion plethysmography and clinical exam in SCI patients. All had sensitivity and specificity of 100% in small (n=21) series. Overall incidence 19%. (Class III because no gold standard used)	Class III	Doppler US, VOP, and clinical examination all good for diagnosis of DVT
Myllynen et al, Journal of Trauma, 1985	Compared incidence of DVT in immobilized spinal injured patients with and without paralysis. Those with paralysis had a 100% DVT incidence (fibrinogen scan) vs. 0% for patients immobilized following spinal fracture without paralysis.	Class III	Incidence of DVT is very high in SCI patients and is not totally dependent on immobilization.
El Masri and Silver, Paraplegia, 1981	Retrospective review of 102 patients with SCI. There were 21 episodes of PE in 19 patients. No patient with PE was adequately anticoagulated at the time of the PE (oral phenindione). Only 8/19 patients had evidence of DVT by exam or VOP.	Class III	The authors recommend prolonged treatment (up to 6 months) in patients with obesity or prior history of DVT.

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
Frisbie and Sasahara, Paraplegia, 1981	Small prospective controlled study of Low dose (5000 $\mu$ BID) heparin vs. Control group. No difference in incidence of DVT noted (only 7% in each group). Authors suggest protective effect of frequent physiotherapy.	Class II	No difference between low dose heparin and control groups in SCI patients receiving twice daily physiotherapy.
Perkash et al, Paraplegia, 1980	Treatment of 8 patients with thromboembolism discussed. Authors used heparin followed by coumadin with reasonable results	Class III	Anticoagulation is effective treatment for SCI patients with thromboembolism
Perkash et al, Paraplegia, 1978-9	Incidence of thromboembolism in 48 SCI patients was 18% Clinical exam sensitivity 89%, specificity 88%, NPV 97%, PPV 62% 1/3 of thromboembolic events occurred >12 weeks following injury	Class I for diagnostic tests, Class III otherwise	Clinical examination appears to be quite good for detection of DVT in subacute setting. Period of risk may extend beyond 12 weeks.
Watson, Paraplegia, 1978	Retrospective historical cohort study looking at low dose heparin vs. no prophylaxis.	Class III	Heparin group had fewer TE complications. No TE events after 3 months despite prophylaxis cessation at 3 months.
Casas et al, Paraplegia, 1977	Prospective assessment of low dose heparin in 18/21 patients with SCI (mean duration 66 days). No patient treated had symptomatic DVT or PE. No use of US/PG/venography	Class III	Low dose heparin may be useful for prevention of symptomatic DVT
Todd et al, Paraplegia, 1976	Used VOP, Fibrinogen scan and venography to study 20 SCI patients for 60 days. Fibrinogen scan was positive in all patients but was confirmed by another test in only half of the cases.	Class III	DVT is common in SCI population.
Hachen, Paraplegia, 1974	Cohort controlled trial of low-dose heparin (5000 $\mu$ t.i.d.) vs. oral warfarin in SCI patients. Heparin group had significantly fewer TE events.	Class II	Low dose SQ heparin better than oral warfarin for prophylaxis following acute SCI.
Naso, Arch Phys Med Rehab, 1974	PE occurred in 4/26 patients with acute (<3 months) SCI but none occurred in 17 patients with chronic (>3 months) SCI	Class III	SCI patients primarily at risk during first 3 months following injury.
Watson, Paraplegia, 1968	Incidence of thromboembolic complications per year ranges from 8-40% in same unit (no prophylaxis)	Class III	Thromboembolic complications are a significant problem and there is variability year to year despite identical treatment strategies.

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## NUTRITIONAL SUPPORT AFTER SPINAL CORD INJURY

### RECOMMENDATIONS

Standards: There is insufficient evidence to support treatment standards.

Guidelines: There is insufficient evidence to support treatment guidelines.

Options: Nutritional support of SCI patients is recommended. Energy expenditure is best determined by indirect calorimetry in these patients as equation estimates of energy expenditure and subsequent caloric need tend to be inaccurate.

### RATIONALE

Hypermetabolism, an accelerated catabolic rate and rampant nitrogen losses are consistent sequelae to major trauma, particularly acute traumatic brain injury and acute spinal cord injury. (7,9-11,13,18,20,23). A well-documented hypermetabolic, catabolic injury cascade is initiated immediately after central nervous system injury which results in depletion of whole body energy stores, loss of lean muscle mass, reduced protein synthesis, and ultimately in loss of gastrointestinal mucosal integrity and compromise of immune competence. (5,9,10,12,18,20,23). Severely injured brain and spinal cord injury patients, therefore, are at risk for prolonged nitrogen losses and advanced malnutrition within two to three weeks following injury with resultant increased susceptibility for infection, impaired wound healing and difficulty weaning from mechanical ventilation. (6,9,13,18,20,23). These factors added to the inherent immobility, denervation and muscle atrophy associated with spinal cord injury provide the rationale for nutritional support of spinal cord injured patients following trauma.

## **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”: nutrition and nutritional support. Approximately 105 citations were acquired. Non-English language citations were deleted. Titles and abstracts of the remaining publications were reviewed. We focused on the specific issue of nutrition and human patients with acute spinal cord injuries and identified 18 articles. Relevant manuscripts and reviews describing nutritional support of head injured patients and several reports describing the nutritional status of chronic SCI patients are included in the bibliography. These efforts identified one Class II study and four Class III studies which describe metabolism, nitrogen wasting and the effect of feeding on nitrogen balance and serum biochemistries in patients after acute SCI. They are summarized in Evidentiary Table format. There were no studies that examined the effects of nutritional support on outcome following acute SCI.

## **SCIENTIFIC FOUNDATION**

Hypermetabolism, catabolism and accelerated nitrogen losses are well-recognized complications that follow traumatic injury. (9,11,20,23). They have been identified and studied in human patients who have sustained traumatic brain and spinal cord injuries. A number of publications have described the increased energy requirements and nitrogen losses of patients following acute head injury. (7-9,11,12,17,18,22,23) Fewer studies have focused on hypermetabolism, catabolism and nitrogen losses following acute SCI. (5,13,14,19,23). While there are metabolic similarities between isolated traumatic brain injury and severe isolated SCI, it

appears there may be important biological differences between the two CNS injury types that have bearing on supplemental nutritional therapy. (5,13,14,18,19,20,23)

Severe head injury is associated with a resting energy expenditure (REE) of approximately 140% of predicted normal basal energy expenditure (BEE). (8,9,11,17,18,22,23). Indirect calorimetry is the most widely used reliable means to determine individual energy requirements in hospitalized patients after traumatic injury. (9,18,20,23). It requires the use of a portable metabolic cart and employs a technique that measures respiratory gas exchange and the rate of oxygen utilization in a given patient. It provides an estimate of energy expenditure by the patient by determining the known caloric yield from one liter of oxygen based on differences in oxygen consumption and carbon dioxide production. It is performed at the bedside in the intensive care unit in severely injured patients. Metabolic expenditure is expressed as a percent of normal BEE at rest (predicted). Indirect calorimetry is typically performed once daily for the first several days post-injury and periodically thereafter. (9,18,20,23) The Harris-Benedict equation, with activity and stress of injury variables, has been shown to predict energy expenditure after TBI with reasonable accuracy without indirect calorimetry. (9,14,19,20,23)

Nutritional support of head injured patients is typically begun within days of admission and is guided by the metabolic information provided by indirect calorimetry and by predicted energy expenditure (PEE) values derived by equation. Hypermetabolism, accelerated catabolism and excessive nitrogen losses continue for at least two weeks after injury. (8-11,18,20,23). The exact duration of this response to injury is unknown, may vary among similar patients and can be affected by other traumatic injuries, pancreatitis, infection or sepsis. (2,9,18,20,23). Nutritional support in this setting is designed to provide nitrogen rich, high-energy supplemental fuel to blunt excess catabolism and preserve energy stores, muscle mass, gastrointestinal integrity and

immune competence. (5,9,18,20,23) Nitrogen balance is difficult, often impossible to achieve, particularly within the first week of injury.(7,11,13,14,19) Matching nutritional replacement with caloric needs, therefore, has become the primary goal of nutritional therapy.

The extent of neuronal connectivity and the neurogenic stimuli (muscle tone) to the musculoskeletal system appears important to the level of metabolic expenditure after CNS injury. (1,3,4,13-16,18-21,23). Agitated, combative head injured patients, for example, can have REEs as high as 200% of expected BEE levels.(9,11,18,23). Conversely, pharmacological paralysis of head injured patients has been associated with reductions in resting energy expenditure by 20% to 30%.(9,11,18,23) Patients who have sustained isolated acute SCI often have increased metabolic expenditure compared to normative energy expenditure levels. (13,14,18-20,23). However, because of the paralysis and flaccidity associated with acute SCI, measured resting energy expenditure (REE) values in these patients are considerably lower than those predicted by the Harris-Benedict equation based on age, sex, body surface area, activity and injury severity.(14,19,20,22) Patients with the greatest neurological deficits and the least muscle tone after SCI (high cervical level quadriplegic patients) have lower measured REE values than those found in patients with incomplete spinal injuries or lower spinal cord injuries (thoracic level paraplegic patients).(13,14,19,20,23) Kaufman et. al., in 1985, described their experience with eight acute SCI patients managed at the University of Texas.(13) They noted accelerated nitrogen losses and ongoing negative nitrogen balance greater than expected. Differences in initial and follow-up nutritional assessments revealed deterioration in nutritional status during the two-week period of observation, partly due to inadequate supply of protein and calories. Infective complications and prolonged respiratory support were common. The authors concluded that muscle atrophy might play an important role in the accelerated nitrogen losses

they identified in patients with paralysis due to complete spinal cord injury and that improved nutritional support might reduce medical complications following acute SCI.

Young, Ott and Rapp reported four quadriplegic acute SCI patients they assessed with indirect calorimetry.(23) They found that indirect calorimetry provided more accurate REE values for their patients compared to Harris-Benedict equation estimates, even Harris-Benedict equation estimates without incorporating injury and activity factors. They too noted marked daily nitrogen losses and negative nitrogen balance. They concluded that equation estimates of REE of SCI patients overestimate metabolic expenditure and emphasized the importance of indirect calorimetry in predicting energy expenditure following acute SCI.

Kearns and associates prospectively assessed and provided nutritional support to ten acute SCI patients they managed and monitored for four weeks.(14) Their 1992 report documents the use of indirect calorimetry to determine REE and provide matched caloric supplementation. All patients had isolated SCI without associated head injury or other organ system trauma. Initial measured resting energy expenditures were 10% below predicted BEE levels. All patients experienced exaggerated nitrogen and three-methylhistidine losses indicating excessive lean body mass and muscle loss. A 10% decrease in body weight accompanied these losses despite caloric replacement matched to or exceeding measured REE values for each patient. The specifics of nutrition administration (mix and route of delivery) were not presented. The authors noted an increase in REE over time in part due to reductions in body weight and in part due to return of muscle tone. The authors concluded that acute isolated SCI is associated with lower REE values compared to predicted. Acute SCI patients have exaggerated nitrogen and three-methylhistidine losses due to atrophy of denervated muscle. They attributed the reduced metabolic activity seen in these patients to the flaccidity of denervated musculature after

severe SCI, and noted that as muscle loss and weight reductions progress, REE increases, particularly if recovery of motor function and/or return of muscle tone occurs.

Rodriguez et. al., studied the metabolic response to SCI in 12 acute trauma patients. (19) Assessment and nutritional support were instituted immediately after injury and continued for four weeks post-injury. Harris-Benedict estimations of energy expenditure were compared to values obtained from indirect calorimetry in each patient. All patients had accelerated nitrogen losses and negative nitrogen balance. Eleven of 12 patients had negative nitrogen balance for the entire four weeks of therapy despite matched caloric replacement. The single patient in whom nitrogen balance was realized had an incomplete SCI. The Harris-Benedict equation with activity factor of 1.2 and a stress/injury factor of 1.6 consistently overestimated energy expenditure in these 12 patients and would have resulted in excessive feeding. The authors concluded that large nitrogen losses after severe SCI are “obligatory” as a result of atrophy and wasting of denervated musculature below the level of injury. Patients with complete traumatic myelopathy had greater obligatory nitrogen losses than patients with incomplete spinal cord injuries. They recommended that indirect calorimetry be used as the energy expenditure assessment method after SCI, particularly in the early post-injury period. If the Harris-Benedict equation is used in these patients in this setting, they recommend that the activity factor should be eliminated and the stress/injury factor of the equation should be reduced.

Cruse and associates examined the neurological, immune, endocrine and nutritional status of 15 male SCI patients and compared them to 16 healthy age-matched control subjects.(6) The timing of assessment in relation to SCI for each patient was not specified. Their report described decreased natural and adaptive immune responses in the SCI patient population beginning within two weeks of injury that reached a nadir three months after injury. They noted increased ACTH

and plasma cortisol levels, decreased zinc, albumin and prealbumin serum levels, surface marker changes in both lymphocytes and granulocytes and decreased adhesion molecule binding ability after SCI compared to healthy control patients. They concluded that patients with severe acute SCI have decreased immune function, impaired nutritional status and a decreased number of adhesion molecules, all of which occurs within weeks after acute injury. The authors note that these hormonal alterations, nutritional deficiencies and changes in immune function may increase susceptibility to infection and may contribute to delayed wound healing.

The change in energy expenditure identified in patients following acute SCI appears to persist long after the initial injury and recovery phase.(1,3,4,15,16,20,21) Several investigators have noted long-standing reductions in REE in spinal cord injury patients, reductions that correlate to the degree of neurological injury and the extent of lean body mass loss after paralysis.(1,3,4,15,16,20,21) Cox et al measured energy expenditure in stable non-acute SCI patients in the rehabilitation setting.(4) They reported that quadriplegic patients required 22.7 kcal/kg/day compared to 27.9 kcal/kg/day for paraplegic patients they studied. Most investigators conclude that equation methods to estimate energy expenditure in SCI patients are inaccurate, both in the acute and chronic settings.(15,16,19,21,23)

There has been no report assessing the efficacy of the route of feeding (parenteral or enteral) for SCI patients in the acute setting. The literature on nutritional support for head injury patients supports using the enteral route for nutritional supplementation if the gut is functional. (8,9,11,18-20,23) This general policy appears to have been followed by investigators of nutritional support for acute SCI patients.(13,14,19,23) The potential benefits of enteral feeding over parenteral delivery include maintenance of gut integrity and function, reduced expense, lower risk of infection and avoidance of intravenous catheter related complications.

(8,9,11,18,20,23) Nasoduodenal or nasojejunal feeding tubes usually allow full caloric, high-nitrogen, high volume feeding within days of injury. In patients with bowel injury, mechanical bowel obstruction or prolonged ileus it is recommended that parenteral nutrition be initiated until the bowel recovers and conversion to enteral nutrition can be accomplished.(8,9,18,20,23)

There has been no report assessing the mix or composition of nutritional supplementation for SCI patients. The literature on nutritional support for head injury patients suggests beginning with a high nitrogen enteral or parenteral solution containing at least 15% of calories as protein, no greater than 15% glucose/dextrose, a minimum of 4% of total energy needs as essential fatty acids and the addition of vitamins, essential elements and trace minerals. (9,12,17,18,20,22,23)

There has been no study published that has examined the effect of nutritional support on neurological outcome following acute SCI.

## **SUMMARY:**

Alterations in metabolism occur after acute SCI, but the marked hypermetabolic response seen after acute traumatic brain injury appears to be blunted in SCI patients, by the flaccidity of denervated musculature after spinal cord transection/injury. As a result, resting energy expenditure (REE) is lower than predicted after acute SCI. Equation estimates of REE in these patients have proven to be inaccurate, therefore indirect calorimetry is the recommended technique to assess energy expenditure in both the acute and chronic settings.

Protein catabolism does occur after acute, severe SCI, and marked losses in lean body mass due to muscle atrophy result in huge nitrogen losses, prolonged negative nitrogen balance and rapid weight loss. Nutritional support of the SCI patient to meet caloric and nitrogen needs,

not to achieve nitrogen balance, is safe and may reduce the deleterious effects of the catabolic, nitrogen wasting process which occurs after acute spinal cord injury.

**KEY ISSUES FOR FUTURE INVESTIGATION:**

An assessment of the timing, route of administration and the composition of nutritional therapy on outcome, both neurological and medical should be performed. This could be best accomplished with a multi-center case control study.

## EVIDENTIARY TABLES

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
Cruse JM et al, 2000, <i>J Spinal Cord Medicine</i>	Comparison of nutritional, immune, endocrine status in 15 acute SCI patients vs. 16 matched controls.	Class II	SCI patients have hormonal changes, poor nutritional status, and decreased immune function compared to controls.
Rodriguez DJ et al, 1997, <i>Spinal Cord</i>	Prospective assessment and treatment of 12 acute SCI patients.	Class III	REE less than predicted, marked "obligatory" nitrogen losses due to flaccidity and atrophy of denervated muscle after SCI.
Kearns PJ et al, 1992, <i>J Parenteral Enteral Nutrition</i>	Prospective assessment of 10 acute SCI patients over 4 week period of observation.	Class III	Exaggerated nitrogen and 3MeH excretion marked weight loss. Lower REE than predicted after SCI.
Young B et al, 1987, <i>Critical Care Clinics</i>	Four acute SCI patients assessed via indirect calorimetry.	Class III	Indirect calorimetry best means to determine energy expenditure after acute SCI.
Kaufman HH et al 1985, <i>Neurosurgery</i>	Assessment of nutritional status of 8 SCI patients over 2 week period of observation.	Class III	Deterioration in nutritional status despite attempted treatment. Marked nitrogen losses. Increased infectious and respiratory complications.

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## MANAGEMENT OF PEDIATRIC CERVICAL SPINE AND SPINAL CORD INJURIES

### RECOMMENDATIONS

#### Diagnostic:

Standards: There is insufficient evidence to support diagnostic standards.

Guidelines:

- In children who have experienced trauma and are alert, have no neurological deficit, no midline cervical tenderness, no painful distracting injury, and are not intoxicated, cervical spine radiographs are unnecessary to exclude cervical spine injury and are not recommended.

- In children who have experienced trauma and who are either not alert or have neurological deficit, midline cervical tenderness, painful distracting injury, or are intoxicated, it is recommended that cervical spine radiographs be obtained.

Options:

- In children less than nine years of age who have experienced trauma, and who are non-convulsant or have an altered mental status, a neurological deficit, neck pain, a painful distracting injury, are intoxicated, or have unexplained hypotension, it is recommended that AP and lateral cervical spine radiographs be obtained.

- In children nine years of age or older who have experienced trauma, and who are non-convulsant or have an altered mental status, a neurological deficit, neck pain, a painful distracting injury, are intoxicated, or have unexplained hypotension it is recommended that AP, lateral, and open-mouth cervical spine radiographs be obtained.

- CT scan with attention to the suspected level of neurological injury to exclude occult fractures, or to evaluate regions not seen adequately on plain radiographs is recommended.
- Flexion and extension cervical radiographs or fluoroscopy may be considered to exclude gross ligamentous instability when there remains a suspicion of cervical spine instability following static radiographs.
- MRI of the cervical spine may be considered to exclude cord or nerve root compression, evaluate ligamentous integrity, or provide information regarding neurological prognosis.

## **Treatment**

**Standards:** There is insufficient evidence to support treatment standards.

**Guidelines:** There is insufficient evidence to support treatment guidelines.

- Options:**
- Thoracic elevation or an occipital recess to prevent flexion of the head and neck when restrained supine on an otherwise flat backboard may allow for better neutral alignment and immobilization of the cervical spine in children less than eight years of age and is recommended.
  - Closed reduction and halo immobilization for injuries of the C2 synchondrosis between the body and odontoid is recommended in children less than seven years of age.
  - Consideration of primary operative therapy is recommended for isolated ligamentous injuries of the cervical spine with associated deformity.

## **RATIONALE**

There are distinct, unique aspects of the management of children with potential injuries of the cervical spinal column and cervical spinal cord compared to adult patients that warrant specific recommendations. The methods of pre-hospital immobilization necessary to approximate “neutral” cervical spinal alignment in a young child differ from those methods commonly employed for adults. The spinal injury patterns among young children differ from those that occur in adults. The diagnostic studies and images necessary to exclude a cervical spine injury in a child may be different than in the adult as well. The interpretation of pediatric radiographic studies must be made with knowledge of age-related development of the osseous and ligamentous anatomy. Methods of reduction, stabilization, and subsequent treatment, surgical and non-surgical, must be customized to each child taking into account the child’s degree of physical maturation and his/her specific injury. The purpose of this review is to address the unique aspects of children with real or potential cervical spinal injuries and provide recommendations regarding their management.

## **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 injuries” and “child” yielded 1022 citations. These citations were reviewed in combination with “cervical vertebra”, “spinal injuries” and “child” which yielded 152 citations. Non-English language citations were deleted. The remaining abstracts were reviewed for those that described children who had sustained or were being evaluated for a cervical spinal cord or cervical spinal column injury. Articles describing the clinical aspects and management of children were used to

generate these guidelines. Case reports were excluded. Of the 57 articles meeting selection criteria, none were Class I studies. There was one Class II study addressing diagnostic imaging in children. All remaining articles were case series representing Class III evidence. Summaries of these 57 articles are provided in Evidentiary Table format.

## **SCIENTIFIC FOUNDATION**

### ***Pre-hospital Immobilization***

The primary goal of pre-hospital management of pediatric patients with potential cervical spine or spinal cord injury is to prevent further injury. Along with assuring an adequate airway, ventilation, and perfusion, spinal immobilization likely plays an important role in preventing further injury to the vertebral column and spinal cord. Immobilization of the child's cervical spine in the neutral position is desired. To achieve neutral alignment of the cervical spine in children less than eight years of age, allowances must be made for the relatively large head compared to the torso, that forces the neck into a position of flexion when the head and torso are supine on a flat surface (39). Nypaver and Treloar prospectively evaluated 40 children less than eight years of age seen in an emergency room for reasons other than head and neck trauma and assessed them with respect to neutral positioning upon a backboard (39). They found that all 40 children required elevation of the torso to eliminate positional neck flexion and achieve neutral alignment as determined by two independent observers. The mean amount of elevation required was 25 millimeters. Children less than four years of age required greater elevation than those four years of age or older ( $P < 0.05$ ). Because of these findings it was recommended that when immobilizing children less than eight years of age that either the torso be elevated or an occipital recess be created to achieve a more neutral position for immobilization of the cervical spine. In a

separate report, Treloar and Nypaver similarly found that semi-rigid cervical collars placed on children less than eight years of age did not prevent this positional forced flexion when placed supine on standard, rigid spinal boards (60).

Herzenberg, et al, studied ten children less than seven years of age with cervical spine injuries who were positioned on a backboard. All had anterior angulations or translation at the injured segment that was reduced by allowing neck extension into a more neutral position (26). They suggested that alignment of the patient's external auditory meatus with his/her shoulders would help to achieve neutral cervical spine positioning.

Curran, et al, however, found no correlation with age regarding degree of cervical kyphosis identified in children transported on backboards (8). They did note however, that 30% of children had greater than ten degrees of kyphosis as determined by Cobb angle measurements between C2 and C6. No specific technique or device allowed superior neutral positioning of the cervical spine in patients they studied. None of their patients were immobilized on boards with an occipital recess or thoracic padding.

Huerta, et al, evaluated a variety of immobilization devices on children, infants and child-sized mannequins (27). They concluded that no collar provided "acceptable immobilization" when used alone. They found that the combination of a modified half-spine board, rigid cervical collar, and tape was the most effective means of immobilization of the cervical spine for transport in children.

Shafermeyer, et al, however, cautioned that immobilization techniques that employ taping across the torso to secure the child to the spine board may have deleterious effects on respiratory function (55). They studied 51 healthy children, ages six to 15 years by measuring forced vital capacity (FVC). FVC dropped when going from the upright to supine position.

Taping across the torso to secure the volunteer to the spine board caused further reductions in FVC of 41% to 96% (mean 80%), compared to the supine FVC without tape. The authors cautioned that this restriction of FVC might be enough to create respiratory insufficiency in some trauma patients.

In summary when spinal immobilization is indicated for children for transportation, the type of immobilization should take into account the child's age and physical maturity. It should allow for the relatively larger head with respect to the torso in younger children. While ideal spinal immobilization of pediatric trauma victims appears to be provided by a combination of a spinal board, rigid collar, and tape, these immobilization techniques may influence negatively the child's respiratory function.

### ***Imaging***

Following immobilization and transport to an acute care facility, initial clinical evaluation and medical/hemodynamic support, the need for and type of radiographic assessment must be decided and undertaken. Several authors have evaluated the indications for radiographic assessment of children with a potential cervical spinal injury (4,32). Laham, et al, investigated the role of cervical spine x-ray evaluation of 268 children with apparent isolated head injuries (32). They retrospectively divided the children into high (n=133) and low-risk (n=135) groups. High-risk characteristics were children incapable of verbal communication either because of age (less than two years of age) or head injury, and those children with neck pain. They employed the "three-view approach" of A-P, lateral, and open-mouth radiographs. They discovered no cervical spine injuries in the low-risk group but discovered ten in the high-risk group (7.5%). The authors concluded that cervical spine radiographs are not necessary in children with isolated

head injuries who can communicate and have no neck pain or neurological deficit. Bohn, et al, emphasized that unexplained hypotension or absent vital signs in childhood trauma victims are likely to be from a severe cervical cord injury (4). Therefore, they advocate suspicion for a cervical spinal cord injury in children with either multisystem trauma, or an isolated head injury presenting with hypotension or cardiopulmonary arrest.

Viccellio, et al, evaluated the cervical spines in children less than 18 years of age utilizing the National Emergency X-Radiography Utilization Study (NEXUS) decision instrument in a Class II prospective multicenter study (62). They employed five low-risk criteria. These criteria were the absence of: 1) midline cervical tenderness, 2) evidence of intoxication, 3) altered level of alertness, 4) focal neurological deficit, and 5) a painful distracting injury. Radiographs were obtained at the discretion of the treating physician. When radiographs were obtained a minimum of three-views was obtained. Only those patients who obtained radiographs were included in the study. If all five criteria were met the child was considered low-risk. If any one of the five criteria were present the child was considered high-risk. Three thousand and sixty-five children were evaluated. Of these 603 fulfilled the low-risk criteria. None of these 603 children defined as low-risk had a documented cervical spine injury by radiographic evaluation. Thirty injuries (0.98%) were documented in children not fulfilling the low-risk criteria. They concluded that applying the NEXUS criteria to children would reduce cervical spine radiograph use by 20% and not result in missed injuries. They cautioned that they had relatively small numbers of young children less than two years of age (n=88). Statistically this created large confidence intervals for the sensitivity of their instrument when applied to younger children. From this Class II study, they “cautiously” endorsed the application of NEXUS criteria in children, particularly those from birth to nine years of age. Their conclusions

are consistent with the Class III evidence previously described by Laham, et al, on this topic (32).

The need for and utility of open-mouth odontoid views in pediatric trauma victims has been questioned (6,59). Swischuk, et al, surveyed 984 pediatric radiologists to determine how many injuries were missed on lateral cervical spine radiographs, yet detected on an open-mouth view (59). There were 432 responses. One hundred and sixty-one respondents did not routinely use open-mouth views. Of the 271 that obtained open-mouth views in young children, 191 (70%) would not persist beyond a single attempt. Seventy-one radiologists (26%) would make up to five attempts to obtain an adequate image. Twenty-eight of the 432 respondents (7%) reported missing a total of 46 fractures on the lateral view that were detected on the open-mouth view. The types of injuries were not classified (i.e. odontoid versus C1 injury). The authors calculated a missed fracture rate of 0.007 per year per radiologist in their study. They concluded that the open-mouth view x-ray might not be needed routinely in children less than five years of age. Buhs, et al, also investigated the utility of open mouth views in children (6). They performed a multi-institutional retrospective review of a large metropolitan population of patients less than 16 years of age who were assessed for cervical spine trauma over a ten-year period. Fifty-one children with cervical spinal injuries were identified. The lateral cervical spine radiograph made the diagnosis in 13 of 15 children less than nine years of age. In none of the 15 younger patients did the open-mouth view provide the diagnosis. In only one of 36 patients in the nine to 16 years of age group was the open mouth view the diagnostic study (a type III odontoid injury). The authors concluded that the open mouth view radiograph is not necessary for clearing the cervical spine in children less than nine years of age.

Lui, et al, in their review of 22 children with C1-C2 injuries commented that flexion and extension radiographs were required to “identify the instability” of traumatic injuries to the dens in four of 12 children with odontoid fractures, and in six of nine children with purely ligamentous injuries resulting in atlantoaxial dislocation (33). The authors did not state whether an abnormality on the static radiograph led to the dynamic studies, or whether the initial static studies were normal. Because they did not describe flexion and extension x-rays as part of their routine for the assessment of children with potential cervical spine injuries it is likely that some imaging or clinical finding prompted the decision to obtain dynamic films in these children.

The experience of Ruge, et al, highlighted the propensity for upper cervical injuries in children under the age of nine years (49). They reported no injuries below C3. Evans and Bethem described 24 children with cervical spine injuries (14). In half of the patients, the injury was at C3 or higher (14). Givens, et al, however, described the occurrence of important injuries occurring at all levels of the cervical spine in young children (20). They described 34 children with cervical spine injuries. There was no correlation of level of injury with age. Two of the children they managed had injuries at C7-T1. Hence, it would be dangerous to assume that lower cervical spine injuries do not occur in young children and irresponsible to discount the need for adequate imaging of the lower cervical spine and cervical-thoracic junction in these young patients.

Scarrow, et al, attempted to define a protocol to evaluate the cervical spine in obtunded children following trauma (50). They utilized somatosensory-evoked responses during flexion and extension fluoroscopy. Of the 15 children evaluated with this protocol, none showed pathological motion during flexion and extension fluoroscopy. Three children were thought to have a change in the evoked responses during flexion and extension. Only one of the three

children with an abnormal evoked response underwent magnetic resonance imaging (MRI) that was normal. Their investigation failed to demonstrate any utility for evoked responses, flexion and extension fluoroscopy, or MRI of the cervical spine in the evaluation of the cervical spine in children with altered mental status following trauma. Larger numbers of children investigated in this manner might define a role for one or more of these diagnostic maneuvers but as yet there is no evidence to support their use.

Ralston, et al, retrospectively analyzed the cervical spine radiographs of 129 children who had flexion and extension x-rays performed after an initial static radiograph (45). They found that if the static radiograph was normal or depicted only loss of lordosis the flexion and extension views would reveal no abnormality. The authors concluded that the value of the dynamic radiographs was confirmation of cervical spinal stability when there was a questionable finding on the static, lateral radiograph (45).

The interpretation of cervical spine x-rays must account for the age and anatomical maturation of the patient. Common normal findings on cervical spine radiographs obtained on young children are pseudosubluxation of C2 on C3, overriding of the anterior atlas in relation to the odontoid on extension, exaggerated atlanto-dens intervals, and the radiolucent synchondrosis between the odontoid and C2 body. These normal findings can be mistaken for acute traumatic injuries in children following trauma. Cattell and Filtzer obtained lateral cervical radiographs in neutral, flexion, and extension in 160 randomly selected children who had no history of trauma or head and neck problems (7). The subjects' ages ranged from one to 16 years with ten children for each year of age. They found a 24% incidence of moderate to marked C2 on C3 subluxation in children between one and seven years of age. Thirty-two of 70 children (46%) less than eight years of age had three millimeters or more of anterior-posterior motion of C2 on C3 on flexion

and extension radiographs. Fourteen percent of all children had radiographic pseudosubluxation of C3 on C4. Twenty percent of children from one to seven years of age had an atlanto-dens interval of three millimeters or greater. Overriding of the anterior arch of the atlas on the odontoid was present in 20% of children less than eight years-old. The synchondrosis between the odontoid and axis body was noted as a lucency in all children imaged up to the age of four years. The synchondrosis remained visible in half the children up to eleven years of age. The authors also described an absence of the normal cervical lordosis in 14% of subjects, most commonly in the eight to 16 year-old age groups. Shaw, et al, in a retrospective review of cervical spine x-rays in 138 children less than 16 years of age who were evaluated following trauma, found a 22% incidence of radiographic pseudosubluxation of C2 on C3 (56). The only factor that correlated with the presence of pseudosubluxation in their study was patient age. The pseudosubluxation group had a median age of 6.5 years versus nine years in the group without this finding. It was identified however, in children as old as 14 years of age. Intubation status, injury severity score, and gender had no correlation with pseudosubluxation of C2 on C3. To differentiate between physiological and traumatic subluxations they recommend a method that involves drawing a line through the posterior arches of C1 and C3. In the circumstance of pseudosubluxation of C2 on C3, the C1-C3 line should pass through, touch, or lie up to one millimeter anterior to the anterior cortex of the posterior arch of C2. If the anterior cortex of the posterior arch of C2 is two millimeters or more behind the line, then a true dislocation (rather than pseudosubluxation) should be assumed.

Keiper, et al, reviewed their experience of employing MRI in the evaluation of children with clinical evidence of cervical spine trauma who had no evidence of fracture by plain radiographs or CT, but who had persistent or delayed symptoms, or instability (29). There were

16 abnormal MRI examinations in 52 children. Posterior soft tissue and ligamentous changes were described as the most common abnormalities. Only one child had a bulging disc. Four of these 52 children underwent surgical treatment. In each of the four surgical cases, the MRI findings led the surgeon to stabilize more levels than otherwise would have been undertaken without the MRI information. Davis, et al, described the use of MRI in evaluating pediatric spinal cord injury and found it did not reveal any lesion that would warrant surgical decompression (9). They did note however that MRI findings did correlate with neurological outcome. Evidence of hematomyelia was associated with permanent neurological deficit. While little information is available on this subject, it appears that preoperative MRI of children with unstable cervical spinal injuries, who require surgical stabilization, may affect the specifics of the surgical management.

There are no studies that have systematically reviewed the role of CT in the evaluation of the cervical spines of pediatric patients following trauma. In children less than ten years of age with cervical spinal injuries, the majority of patients will have ligamentous injuries without fracture (10,13,24,25,41). In older children with cervical spinal injuries, the incidence of a fracture is much greater than ligamentous injury without fracture, 80% vs. 20% respectively (14, 62). Therefore, normal osseous anatomy as depicted on an axial CT image should not be used alone to exclude injury to the pediatric cervical spine. Schleeauf, et al, in 1989, concluded that CT should not be relied upon to exclude ligamentous injuries in a series of pediatric and adult trauma patients (51). They reported two false negative CT studies in patients with C1-C2 ligamentous injuries in their study of the merits of CT to evaluate the cervical spine in high-risk trauma patients. The authors favored CT for the evaluation of regions that could not be viewed

adequately with plain radiographs (e.g. C7-T1), and for the investigation of the osseous integrity of specific vertebra suspicious for fracture on plain radiographs (51).

In a series consisting of almost entirely adults, the role of helical CT in the evaluation of the cervical spine in “high-risk” patients following severe, blunt, multisystem trauma has been prospectively studied (3). The plain spine radiographs and CT images were reviewed by a radiologist blinded to the patients and their history. The investigators found 20 cervical spine injuries (12 stable, eight unstable) in 58 patients (34%). Eight of these injuries (five stable, three unstable) were not detected on plain radiographs. The authors concluded that helical cervical spinal CT should be utilized to assess the cervical spine in high risk trauma patients. In young children in whom the entire cervical spine is often easily and accurately visualized on plain x-ray studies, the need for cervical spinal helical CT is likely not as great. In older high-risk children who have spinal biomechanics and injury patterns more consistent with those of adult trauma patients, helical CT of the cervical spine may be fruitful.

In summary, to “clear” a child’s cervical spine Class II and Class III evidence supports obtaining lateral and A-P cervical spine radiographs in children who have experienced trauma and cannot communicate because of age or head injury, have a neurological deficit, have neck pain, have a painful distracting injury, or are intoxicated. In children who are alert, have no neurological deficit, no midline cervical tenderness, no painful distracting injury, and are not intoxicated cervical spine radiographs are not necessary to exclude cervical spine injury (32,62). Unexplained hypotension should raise the suspicion of a spinal cord injury. Open-mouth views of the odontoid do not appear to be useful in children less than nine years of age. Open-mouth views should be attempted in children nine years of age and older. Flexion and extension studies (fluoroscopy or radiographs) are likely to be unrevealing in children with static radiographs

proven to be normal. Dynamic studies could be considered however, when the static radiographs or the child's clinical findings suggest but do not definitively demonstrate cervical spinal instability. CT studies of the cervical spine should be employed judiciously to define bony anatomy at specific levels but are not recommended as a means to "clear" the entire cervical spine in children. MRI may provide important information about ligamentous injury that may influence surgical management, and may provide prognostic information regarding neurological outcome.

### ***Injury Management***

Injury patterns that have a strong predilection for or are unique to children merit discussion because of the specialized management paradigms employed to treat them. Spinal cord injury without radiographic abnormality (SCIWORA) and atlanto-occipital dislocation injuries have been addressed in other sections (see SCIWORA guideline, see Atlanto-occipital dislocation guideline). Spinal cord injuries secondary to birth-related trauma and epiphysiolysis of the axis are injuries unique to children. Common but not unique to children are C1-C2 rotary subluxation injuries. These entities will be discussed below in light of the available literature. It should be noted that there is no information provided in the literature describing the medical management of pediatric patients with spinal cord injuries. The issue of steroid administration following acute pediatric spinal cord injury, for example, has not been addressed. While prospective, randomized clinical trials such as NASCIS II and NASCIS III have evaluated pharmacological therapy following acute spinal cord injury, children younger than 13 years of age were excluded from study (5).

Neonatal spinal cord injury. Birth injuries of the spinal cord occur approximately one per 60,000 births (63). The most common level of injury is upper cervical followed by cervicothoracic (34). Mackinnon, et al, described 22 neonates with birth-related spinal cord injuries (34). The diagnosis was defined by the following criteria: clinical findings of acute cord injury for at least one day and evidence of spinal cord or spinal column injury by imaging or electrophysiological studies. Fourteen neonates had upper cervical injuries, six had cervicothoracic injuries, and two had thoracolumbar injuries. All upper cervical cord injuries were associated with cephalic presentation and the use of forceps for rotational maneuvers. Cervicothoracic injuries were associated with the breech presentation. All infants had signs of “spinal shock”, defined as flaccidity, no spontaneous motion and no deep tendon reflexes. Of the nine infants with upper cervical injuries surviving longer than three months, seven were alive at last follow-up. Six of these seven are dependent upon mechanical ventilation. The two neonates with upper cervical injuries who had breathing movements on day one of life were the only two thought to have satisfactory outcomes. All survivors with upper cervical cord injuries whose first respiratory effort was beyond the first 24 hours of life have remained ventilator dependent. Only two children of six who sustained cervicothoracic spinal cord injuries lived and remained paraplegic. One requires mechanical ventilation. Hypoxic and ischemic encephalopathy was noted in nine of 14 newborns with upper cervical cord injuries, and in one of six with a cervicothoracic cord injury. The authors did not describe any treatment provided for the underlying spinal column or cord injury, or whether survivors experienced progression of any spinal deformities.

Menticoglou, et al, drawing partly from the same patient data as Mackinnon, et al, reported 15 neonates with birth-related upper cervical spinal cord injuries (37). All were

associated with cephalic deliveries requiring rotational maneuvers with forceps. All but one child was apneic at birth with quadriplegia. There is no description of post-injury spinal column or spinal cord management, medical or surgical, in their report.

Rossitch and Oakes described five neonates with birth-related spinal cord injuries (48). They reported that incorrect diagnoses were made in four. They consisted of Werdnig-Hoffmann syndrome, occult myelodysplasia, and birth asphyxia. Only one neonate had an abnormal plain radiograph (atlanto-occipital dislocation). They provided no description of the management of the spinal cord or column injuries in these five neonates.

Fotter, et al, reported the use of bedside ultrasound to diagnose neonatal spinal cord injury. They found excellent correlation with MRI studies with respect to the extent of cord injury in their two cases (17).

Pang and Hanley provide the only description of an external immobilization device for neonates (42). They described a thermoplastic molded device that is contoured to the occiput, neck, and thorax. Velcro straps cross the forehead and torso, securing the infant and immobilizing the spinal column.

In summary cervical instability following birth-related spinal cord injury is not addressed in the literature. The extremely high mortality rate associated with birth-related spinal cord injury may have generated therapeutic nihilism for this entity, hence the lack of aggressive management. The literature suggests that the presentation of apnea with flaccid quadriplegia following cephalic presentation with forceps manipulation is the hallmark of upper cervical spinal cord injury. Absence of respiratory effort within the first 24 hours of life is associated with dependence upon long-term mechanical ventilation. It appears reasonable to treat these

neonates with spinal immobilization for a presumed cervical spinal injury. The method and length of immobilization is at present arbitrary.

Odontoid Epiphysiolysis. The neurocentral synchondrosis of C2 that may not fuse completely until age seven years represents a vulnerable site of injury in young children (22). The lateral cervical spine radiograph is the diagnostic imaging modality of choice to depict this injury. It will often reveal the odontoid process to be angulated anteriorly, and rarely posteriorly (57). While injuries to the neurocentral or subdental synchondrosis may be seen in children up to seven years of age, it most commonly occurs in pre-school aged children (35). Mandabach, et al, described 13 children with odontoid injuries ranging in age from nine months to seven years (35). They reported that eight of ten children who were initially managed with halo immobilization alone achieved stable fusion. The average time to fusion was 13 weeks with a range of ten to 18 weeks. Because the injury occurs through the epiphysis, it has a high likelihood of healing if closed reduction and immobilization are employed. Mandabach, et al, in their review cited several other reports describing the successful treatment of young children with odontoid injuries who were managed with a variety of external immobilization devices. Sherk, et al, reported 11 children with odontoid injuries and reviewed an additional 24 from the literature. Only one of these 35 children required surgical fusion (57). While the literature describes the use of Minerva jackets, soft collars, hard collars, and the halo vest as means of external immobilization to achieve successful fusion in young children with odontoid injuries, the halo is the most widely employed immobilization device in the contemporary literature for these injuries (35,40,57).

To obtain injury reduction in these children Mandabach, et al, advocate the application of the halo device under ketamine anesthesia followed by realignment of the dens utilizing C-arm

fluoroscopy (35). Other reports describe using traction to obtain alignment, before immobilizing the child in an external orthosis (22). Compared to halo application and immediate reduction and immobilization, traction requires a period of bed rest and is associated with the potential risk of over-distraction (35).

The literature is scant regarding the operative treatment of C2 epiphysiolysis. Most reports describe employing operative internal fixation and fusion only if external immobilization has failed to maintain reduction or achieve stability. Reinges, et al, noted that only three “young” children have been reported in the literature who have had odontoid injuries primarily treated with surgical stabilization (47). This underscores the near universal application of external immobilization as the primary means of treating odontoid injuries in young children. Odent, et al, reported that of the 15 young children with odontoid injuries they managed, three that were treated with surgical stabilization and fusion experienced complications. The other 12 children with similar injuries nonoperatively managed did well (40). Wang, et al, described using anterior odontoid screw fixation as the primary treatment option in a three-year-old child with C2 epiphysiolysis (64). A hard cervical collar was used postoperatively. Halo immobilization was not used either preoperatively or postoperatively. They successfully employed anterior odontoid screw fixation as the primary treatment in two older children (ages ten and 14 years) followed by hard collar immobilization. It is likely that these two children had true type II odontoid fractures and not C2 epiphysiolysis. Likewise, Godard et al performed anterior odontoid screw fixation in a two-year-old child with a severe head injury. They used skeletal traction to align the fracture pre-operatively (21). The rationale for proceeding to operative stabilization without an attempt at treatment with external immobilization was to avoid the halo orthosis, and to allow for more aggressive physiotherapy in this severely injured child. They believe that anterior odontoid

screw fixation is advantageous because no motion segments are fused, normal motion is preserved, and the need for halo immobilization is obviated.

For management of injuries of the C2 neurocentral synchondrosis the literature supports the use of closed reduction and external immobilization for approximately ten weeks. This strategy is associated with an 80% fusion success rate. While primary surgical stabilization of this injury has been reported, the experience in the literature is limited. Surgical stabilization appears to play a role when external immobilization is unable to maintain alignment of the odontoid atop the C2 body. While both anterior and posterior surgical approaches have been successfully employed in this setting, there are more reports describing posterior C1-2 techniques than reports describing anterior operative techniques.

Atlanto-axial rotary subluxation. Fixed rotary subluxation of the atlanto-axial complex is not unique to children but is more common during childhood. They can present following minor trauma, in association with an upper respiratory infection, or without an identifiable inciting event. The head is rotated to one side with the head tilted to the other side causing the so-called “cock-robin” appearance. The child is unable to turn his/her head past the midline. Attempts to move the neck are often painful. The neurological status is almost always normal (12,31,44,58).

It can be difficult to differentiate atlanto-axial rotary subluxation from other causes of head rotation on clinical grounds alone. Several reports describe the radiographic characterization and diagnosis of this entity. Fieldings and Hawkins described 17 children and adults with atlanto-axial rotary subluxation, and classified their dislocations into four types based on radiographic features (15). Type I was the most common type identified in eight of the 17 patients. It was described as unilateral anterior rotation of the atlas pivoting around the dens with a competent transverse ligament. Type II was identified in five patients. It was described as

unilateral anterior subluxation of the atlas with the pivot being the contralateral C1-C2 facet. The atlanto-dens interval is increased to no more than five millimeters. Type III is described as anterior subluxation of both C1 facets with an incompetent transverse ligament. Type IV is posterior displacement of C1 relative to C2 with an absent or hypoplastic odontoid process.

Kawabe, et al, reviewed the radiographs of a series of 17 children with C1-C2 rotary subluxation and classified them according to Fieldings and Hawkins (28). There were ten Type I, five Type II, two Type III, and no Type IV subluxations in their experience. CT has been employed to help define the C1-C2 complex in cases of suspected rotary subluxation. Kowalski, et al, demonstrated the superiority of dynamic CT studies compared to information obtained with static CT studies (31). They compared the CT scans of eight patients with C1-C2 pathology to CT studies of six normal subjects. The CT scans obtained with normal subjects maximally rotating their heads could not be differentiated from the CT scans of those with known C1-C2 rotary subluxation. When the CT scans were performed with the head rotated as far as possible to the contralateral side, CT studies of normal subjects could be easily differentiated from those performed on patients with rotary subluxation.

Type I and Type II subluxation account for the vast majority of rotary atlanto-axial subluxations in reports describing these injuries. Groggaard, et al, (23) and Subach, et al, (58) have published retrospective reviews on the success of conservative therapies in children presenting early following C1-C2 rotary subluxation. Groggaard, et al, described eight children who presented within five days of subluxation, and one child who presented eight weeks after injury (23). All were successfully treated with closed reduction and immobilization. The child presenting late required one week of skeletal traction to achieve reduction and was ultimately treated with halo immobilization for ten weeks. The children who presented early had their

injuries reduced with manual manipulation. They were treated in a hard collar for four to six weeks. Two patients had recurrent subluxation. Both were reduced and treated successfully without surgical intervention. Subach et al reported 20 children with C1-C2 rotary subluxation, in whom four injuries reduced spontaneously (58). Injury reduction was accomplished in fifteen of 16 patients treated with traction for a mean duration of four days. Six children required fusion because of recurrent subluxation (n=5) or irreducible subluxation (n=1). No child experienced recurrent subluxation if reduced within 21 days of symptom onset.

El-Khoury, et al, reported three children who presented within 24 hours of traumatic rotary subluxation (12). All three were successfully treated with traction or manual reduction within 24 hours of presentation. One child experienced recurrent subluxation the next day that was successfully reduced manually. External orthoses were used from ten weeks to four months. Phillips, et al, reviewed 23 children with C1-C2 rotary subluxation (44). Sixteen children were seen within one month of subluxation onset and experienced either spontaneous reduction or were reduced with traction. Of seven children presenting with a duration of symptoms greater than one month, one subluxation was irreducible, and four recurred after initial reduction. Schwarz described four children who presented greater than three months after the onset of C1-C2 rotary subluxation (53). Two children had irreducible subluxations. One child had recurrent subluxation despite the use of a Minerva cast. Only one child had successful treatment with closed reduction and a Minerva cast immobilization for eight weeks. These experiences highlight the ease and success of non-surgical management for these injuries when the subluxation is treated early rather than late. If the subluxation is easily reducible and treated early, four weeks in a rigid collar appears to be sufficient for healing. Because C1-C2 rotary subluxation can reduce spontaneously in the first week, traction or manipulation can be reserved

for those subluxations that do not reduce spontaneously in the first few days. The use of more restrictive external immobilization devices (e.g. halo vest, Minerva cast) for longer periods of treatment up to four months has been described in those children presenting late, or those who have recurrent subluxations (44).

Operative treatment for C1-C2 rotary subluxations has been reserved for recurrent subluxations or those that cannot be reduced by closed means. Subach, et al, operated on six of the 20 children they reported with rotary subluxation using these indications. They employed a posterior approach and accomplished atlantoaxial fusion (58). They had no complications and all fusions were successful.

In summary the diagnosis of atlantoaxial rotary subluxation is suggested when findings of a “cock-robin” appearance are present, the patient is unable to turn the head past midline to the contralateral side, and experiences spasm of the ipsilateral (the side to which the chin is turned) sternocleidomastoid muscle (44). Plain cervical spine radiographs may reveal the lateral mass of C1 rotated anterior to the odontoid on a lateral view. The A-P radiograph may demonstrate rotation of the spinous processes toward the ipsilateral side in a compensatory motion to restore alignment. If the diagnosis of C1-C2 rotary subluxation is not certain after clinical examination and plain radiographic study, a dynamic CT study should be considered. It appears that the longer a C1-C2 rotary subluxation is present before attempted treatment, the less likely reduction can be accomplished. If reduction is accomplished in these older injuries it is less likely to be maintained. Therefore, rotary subluxations that do not reduce spontaneously should undergo attempted reduction with manipulation or traction. The subsequent period of immobilization should be proportional to the length of time that the subluxation was present before treatment.

Surgical arthrodesis can be considered for those with irreducible subluxations, recurrent subluxations, or subluxations present for greater than three weeks duration.

Other injuries. Lui et al, described nine children with ligamentous injuries resulting in atlantoaxial dislocation (33). Unlike children with traumatic injuries to the dens who can be managed with closed reduction and immobilization, these children with atlantoaxial dislocation required surgical stabilization and fusion. The authors attempted to treat two children with halo immobilization for three months duration; both failed to achieve stability. All nine children with atlanto-axial instability required operative stabilization and fusion.

Finally, Rathbone, et al, described a series of 12 children who sustained a “spinal cord concussion” while participating in athletic events. They found that four of these children had plain spine radiographs consistent with cervical spinal stenosis (46). The authors raised the concern that children with congenital cervical stenosis may be more susceptible to spinal cord injury in contact sports.

Therapeutic Cervical Spine Immobilization. Once an injury to the pediatric cervical spine has been diagnosed some form of external immobilization is usually necessary to allow for either application of traction to restore alignment, or to immobilize the spine to allow for healing of the injury. This section will discuss the literature available concerning methods of skeletal traction in children, and various external orthoses used to immobilize the pediatric cervical spine.

Traction for the purpose of restoring alignment or reducing neural compression in children is rarely addressed in the literature. Unique concerns of cervical traction in children exist because of the relatively thinner skull with a higher likelihood of inner skull table penetration, lighter body weight which provides less counter force to traction, more elastic ligaments and less well-developed musculature increasing the potential for over-distraction. The

placement of bilateral pairs of parietal burr holes and passing 22 gauge wire through them to provide a point of fixation for traction has been described for infants with cervical spinal injuries. Gaufin and Goodman reported a series of three infants with cervical injuries, two of whom had injuries reduced in this fashion (19). Up to nine pounds was used in a ten week-old infant and a 16 month-old boy. They experienced no complications with 14 and 41 days of traction, respectively. Other techniques of cervical traction application in children are not described in the literature.

Mubarak, et al, described halo application in infants for the purpose of immobilization but not halo-ring traction (38). They described three infants ages seven months, 16 months, and 24 months. Ten pins were used in each child. The pins in the youngest child were “inserted to finger tightness only”, while the older children had two inch/pounds of torque applied. The children were maintained in the halo devices for two to three and a half months. Only the youngest child had a minor complication of frontal pin site infection necessitating removal of two anterior pins.

Marks, et al, described eight children ages three months to 12 years who were immobilized in halo vests for six weeks to 12 months with a mean duration of two months (36). Only three of these children had cervical spinal instability. Five had thoracic spinal disorders. The only complication they reported was the need to remove and replace the vest when a foreign body became lodged under the vest. Dormans, et al, reported on 37 children ages three to 12 years they managed in halo immobilization devices (11). They had a 68% complication rate. Pin-site infections were most common. They arbitrarily divided their patient population into those less than ten years of age and those ten years or older. Purulent pin site infections occurred more commonly in the older group. Loosening of pins occurred more commonly in the younger

group. Both loosening and infection occurred more often at the anterior pin sites. They also reported one incident of dural penetration and one transient supraorbital nerve injury. Baum, et al, compared halo use complications in children and adults (1). The complication rates in their series were eight per cent for adults and 39 per cent among children. The complications reported for the children were one skull penetration and four pin site infections. While the halo device appears to provide adequate immobilization of the cervical spine in children, there is a higher rate of minor complications compared to halo use with adults.

Gaskill and Marlin described six children ages two years to four years who had cervical spinal instability managed with a thermoplastic Minerva orthosis as an alternative to a halo immobilization device (18). Two of the children they described had halo devices removed because of complications before being placed in Minerva orthoses. The authors described no problems with eating or with activities of daily living in these children. Only one child had a minor complication from Minerva use, a site of skin breakdown. The authors concluded that immobilization with a thermoplastic Minerva orthosis offered a reliable and satisfactory alternative to halo immobilization in young children.

Benzel, et al, analyzed cervical motion during spinal immobilization in adults serially treated with halo and Minerva devices (2). They found that the Minerva offered superior immobilization at all intersegmental levels of the cervical spine with the exception of C1-C2. While this study was carried out in adults with cervical spine instability, it underscores the utility of the Minerva device as a cervical immobilization device. Because a great proportion of pediatric cervical spine injuries occur between the occiput and C2, the Minerva device may not be ideal for many pediatric cervical spine injuries.

In summary the physical properties of young skin, skull thickness, and small body size likely contribute to the higher complication rate among children who require traction or long-term cervical spinal immobilization compared to adults. The literature includes descriptions of options available for reduction and immobilization of cervical spine injuries in children, but does not provide evidence for a single best method.

*Surgical Treatment.* There are no reports in the literature that address the topic of early versus late surgical decompression following acute pediatric cervical spinal cord injury. Pediatric spinal injuries account for only 5% of all vertebral column injuries. Recent reports that describe the management of pediatric spinal injuries have been offered by Turgut et al, Finch and Barnes, and Elaraky, et al (13,16,61). These authors managed pediatric spinal injuries operatively in 17%, 25%, and 30% of patients, respectively. The report by Elaraky, et al, in 2000 suggests that operative treatment of pediatric cervical spine injuries is being utilized more frequently than in the past. Specific details of the operative management including timing of intervention, the approach (anterior versus posterior), and the method of internal fixation as an adjunct to fusion are scarce in the literature. Finch and Barnes employed primary operative stabilization in most children they managed with ligamentous injuries of the cervical spine (16). They stated that while external immobilization may have resulted in ligamentous healing, they elected to internally fixate and fuse such injuries. They based their approach on two cases of ligamentous injuries of the cervical spine that they managed with external immobilization, which failed to heal, that later required operative fusion. Shaked, et al, described six children ages three years to 14 years who had cervical spine injuries that they treated surgically via an anterior approach (54). They reported successful fusion with good alignment and normal cervical spine growth in follow-up for all six children. The procedure varied (i.e. total or partial corpectomy

versus discectomy only) depending on the pathology. All underwent autograft fusion without instrumentation. The authors described severe hyperflexion injury with fracture and avulsion of the vertebral body, fracture-dislocation with disruption of the posterior elements and disc, and major anatomic deformity of the cervical spine with cord compression as indications for an anterior approach.

Pennecot et al described 16 children with ligamentous injuries of the cervical spine (43). They managed minor ligamentous injuries (atlanto-dens interval of five to seven mm, or interspinous widening without dislocation or neurological deficit) with reduction and immobilization. Of eleven children with injuries below C2, eight required operative treatment with fusion via a posterior approach. They used interspinous wiring techniques in younger children (preschool aged) and posterior plates and screws in older children as adjuncts to fusion. All had successful fusion at last follow-up. All children were immobilized in a plaster or halo cast postoperatively. Similarly, Koop, et al, described 13 children with acute cervical spine injuries who required posterior arthrodesis and halo immobilization (30). They reported successful fusion in 12 patients. The single failure was associated with allograft fusion substrate. All other children had autologous grafts. Internal fixation with wire was employed in only two children. Halo immobilization was utilized for an average of 150 days. They reduced the length of post-operative halo immobilization to 100 days in their most recent cases. They commented that careful technique allowed successful posterior fusion in children with minimal complications. Schwarz, et al, described ten children with traumatic cervical kyphosis (52). Two children who underwent anterior reconstruction with fusion had successful deformity reduction. All others managed with either external immobilization with or without traction (n=7)

or posterior fusion (n=1) had either progression of the post-traumatic deformity or a stable unreduced kyphotic angulation.

In summary, pediatric spinal injuries are relatively infrequent. The vast majority are managed non-operatively. Selection criteria for operative intervention in children with cervical spine injuries are difficult to glean from the literature. Anatomic reduction of deformity, stabilization of unstable injuries and decompression of the spinal cord, and isolated ligamentous injuries associated with deformity are indications for surgical treatment cited by various authors (16,30,33,43,52,54,64). These multiple reports provide Class III evidence.

## **SUMMARY**

The available medical evidence does not allow the generation of diagnostic or treatment standards for the management of pediatric patients with cervical spine or cervical spinal cord injuries. Only diagnostic guidelines and options, and treatment options are supported by this evidence. The literature suggests that obtaining neutral cervical spine alignment in a child may be difficult when standard backboards are used. The determination that a child does not have a cervical spine injury can be made on clinical grounds alone is supported by Class II and Class III evidence. When the child is alert and communicative and is without neurological deficit, neck tenderness, painful distracting injury, or intoxication, cervical radiographs are not necessary to exclude cervical spinal injury. When cervical spine radiographs are utilized to verify or rule out a cervical spinal injury in children less than nine years of age, only lateral and A-P cervical spine views need be obtained. The traditional three-view x-ray assessment may increase the sensitivity of plain spine radiographs in children nine years of age and older. The vast majority of pediatric cervical spine injuries can be effectively treated non-operatively. The most effective immobilization appears to be accomplished with either halo devices and Minerva jackets. Halo

immobilization is associated with acceptable but considerable minor morbidity in children, typically pin site infection and pin loosening. The only specific pediatric cervical spine injury for which medical evidence supports a particular treatment paradigm is an odontoid injury in children less than seven years of age. These children are effectively treated with closed reduction and immobilization. Primarily ligamentous injuries of the cervical spine in children may heal with external immobilization alone, but are associated with a relatively high rate of progressive deformity when treated non-operatively. Pharmacological therapy and intensive care unit management schemes for children with spinal cord injury have not been described in the literature.

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

Prospective epidemiological data may be the best source of information that could lead to methods of prevention by identifying the more common mechanisms of spinal injury in children. Future studies involving pediatric cervical spine injury patients should be multi-institutional because of the infrequency of these injuries treated at any single institution. Further defining the indications and methods for cervical spine clearance in young children (less than nine years of age) with prospective gathering of data would be a valuable addition to the literature. The role of flexion and extension radiographs is poorly defined in the literature and a prospective evaluation of their sensitivity and specificity for spinal column injury in specific clinical scenarios would be a valuable addition to the literature. The incidence and clinical significance of complications of cervical spine injuries in children such as syringomyelia and vertebral artery injury are unknown and could be studied by prospectively gathering data in a multi-institutional setting.

More common injuries, such as odontoid injuries could be studied prospectively in a randomized fashion (e.g. closed reduction and immobilization vs. anterior screw fixation), although it would be difficult from technical and feasibility standpoints. Prospectively collected data could also provide the basis for case-control or other comparative studies to generate Class II evidence.

## Evidentiary Tables – Diagnostic

Authors & Year	Description of Study	Class of Data	Conclusions
Viccellio, et al, Pediatrics, 2001	Prospective multicenter evaluation of cervical spine radiographs obtained in 3065 children incurring trauma. Low-risk criteria of absence of: neck tenderness, painful distracting injury, altered alertness, neurological deficit, or intoxication	II	No child fulfilling all five low-risk criteria had a cervical spine injury. Radiographs may not be necessary to clear the cervical spine in children fulfilling all five criteria.
Ralston et al, Academ Emerg Med, 2001.	Blinded review of 129 children with blunt cervical trauma who had flexion and extension radiographs.	III	Flexion and extension views with normal cervical spine radiographs or with only loss of cervical lordosis did not unmask any new abnormalities.
Buhs C et al, J Ped Surg, 2000.	Multi-institutional review of pediatric cervical spine injuries and the radiographs needed to achieve a diagnosis.	III	Lateral cervical radiograph was diagnostic in 13 of 15 children less than 9-years. In no child less than nine years-old was the open mouth view the diagnostic study. Only one of 36 children older than 9-years was the open-mouth view the diagnostic study.
Swischuk LE et al, Pediatr Radiol, 2000.	Survey of pediatric radiologists regarding use of open mouth view of the odontoid.	III	Less than 50% response. Approximately 40% of respondents did not employ open mouth views in children.
Scarrow AM et al, Pediatr Neurosurg, 1999.	Performed flexion extension cervical fluoroscopy with SSEP monitoring in 15 comatose pediatric patients.	III	None had radiographic abnormalities. three children had changes in the SSEP's. One of these three children was studied with MR and it was normal.
Shaw et al, Clin Radiol, 1999	Retrospective review of the cervical radiographs 138 trauma patients under 16 years-old	III	22% incidence of pseudosubluxation of C2 on C3. Median age of pseudosubluxation group was 6.5 years versus nine years for those without pseudosubluxation.
Berne JD et al, J Trauma, 1999.	58 patients with severe blunt trauma underwent helical CT of entire cervical spine.	III	20 had cervical spine injuries. Plain radiographs missed eight injuries. CT missed two injuries.
Keiper et al, Neurorad, 1998.	Retrospective review of evaluating 52 children by MR with suspected cervical spine trauma or instability without fracture	III	There were 16 abnormal studies. The most common abnormality was posterior ligamentous injury. Four children underwent surgical stabilization. The MR findings caused the surgeon to extend his length of stabilization in all four cases.
Davis PC et al, AJNR, 1993	Retrospective review of 15 children with spinal cord injury underwent MR 12 hours to two months after injury. seven with SCIWORA	III	MR correlated with prognosis. Hemorrhagic cord contusions and cord "infarction" were associated with permanent deficits. No compressive lesions in SCIWORA cases. Normal MR was associated with no myelopathy
Schleehauf K et al, Ann Emerg Med, 1989.	104 "high-risk" patients underwent CT as screening tool for cervical spine injury.	III	Sensitivity overall was 0.78. Sensitivity was 1.0 for unstable injuries not able to be seen by plain radiographs. Two upper cervical subluxations without fracture were missed.

<b>Authors &amp; Year</b>	<b>Description of Study</b>	<b>Class of Data</b>	<b>Conclusions</b>
Kowalski et al, AJR, 1987.	Eight patients with occipitalatlantoaxial problems and six normal subjects were studied with CT.	III	CT looked similar for those with C1-2 rotary subluxation to normal subjects with their heads maximally turned. CT with the head turned to the contralateral side differentiated rotary subluxation from normals and spasmodic torticollis.
Cattell & Filtzer, J Bone Joint Surg, 1965.	Lateral upright cervical radiographs in neutral, flexion, and extension in 160 randomly selected children ages one to 16 years.	III	C2-3 subluxation was moderate to marked in 24% predominantly in children less than eight years of age. The atlanto-dens interval was three millimeters or more during flexion in 20% of children less than eight years of age.

## EVIDENTIARY TABLES – Treatment

Authors & Year	Description of Study	Class of Data	Conclusions
Eleraky MA et al, J Neurosurg (Spine) 2000	Retrospective review of 102 children with cervical spinal injuries.	III	30 children (30%) were treated surgically.
Odent T et al, J Ped Ortho, 1999	Review of 15 young children with odontoid injuries	III	6 with neurological deficits had cervicothoracic cord injuries. External immobilization was a successful primary therapy. Three children who were operated upon as their primary therapy experienced complications.
Schwarz, Arch Orthop Trauma Surg, 1998	A review of four children presenting at least three months after the onset of C1-2 rotary subluxation.	III	Two children had irreducible subluxations. One child had recurrent subluxation in a Minerva cast. One child was successfully treated with closed reduction and eight weeks in a Minerva cast.
Subach et al, Spine, 1998	A review of 20 children with C1-2 rotary subluxation.	III	Four reduced spontaneously. 15 of 16 treated with traction reduced in a mean of four days. Six children required fusion because of recurrent subluxation or irreducible subluxation. No child experienced recurrent subluxation if reduced within 21 days of symptom onset.
Finch GD, Barnes MJ, J Ped Ortho, 1998	Retrospective review of 32 children with major cervical spine injuries.	III	Eight children (25%) were treated surgically. All achieved union or radiological stability. No neurological deterioration from surgery or closed reduction. Operated on ligamentous injuries.
Reinges MHT et al, Child Nerv Sys, 1998	Report of primary C1-2 fusion in a young child with an odontoid injury and lower cervical cord injury	III	No neurological improvement. Successful fusion.
Treloar and Nypaver, Ped Emer Care, 1997.	They measured cervical spine flexion in children with semi-rigid collars on spinal boards.	III	Semi-rigid collars did not prevent the cervical spine from being forced into flexion in children less than eight-years old when on a spinal board.
Lui TN et al, J Trauma, 1996	Retrospective review of C1-2 injuries in 22 children. 12 children had odontoid injuries (OI). nine children had ligamentous injuries (atlanto-axial dislocations) only.	III	Flexion/extension radiographs needed to diagnose four OI and six atlanto-axial dislocations (AAD). 9/12 OI reduced easily. 5/7 OI treated successfully with halo. two OI operated immediately. two OI failed external immobilization. five AAD initially treated with surgical fusion. two AAD initially treated with halo required surgical stabilization.
Givens TG et al, J Trauma, 1996	Review of 34 children with cervical spine injuries over a three-year-period	III	18 injuries occurred below C3. The level of injury did not correlate with age. Young age is not associated with exclusively upper cervical spine injuries.
Turgut M et al, Eur Spine J, 1996	Retrospective review of 82 children with spinal cord or column injuries	III	14 children (17%) were treated surgically.

<b>Authors &amp; Year</b>	<b>Description of Study</b>	<b>Class of Data</b>	<b>Conclusions</b>
Dormans JP et al, J Bone Joint Surg, 1995	A review of 37 children with halo rings and vests ages three to 16 years. Arbitrarily divided into those less than ten years-old, and older.	III	Overall 68% complication rate. Pin-site infection was the most common complication. Purulent infections occurred more frequently in the older group. Both loosening and infection occurred more frequently in the anterior pin sites.
Menticoglou SM et al, Obstet Gyn, 1995.	Retrospective case series of 15 neonates with birth-related high cervical cord injuries.	III	All 15 were cephalic presentations in which forceps and attempted rotation were employed. All but one were apneic at birth.
Curran et al, J Trauma, 1995	Prospective study of 118 children who arrived immobilized to a single emergency room. The cervical spine alignment was measured and compared to age and type of immobilization.	III	No correlation with degree of kyphosis or lordosis was found with age. 30% had a kyphosis of greater than ten degrees. No single immobilization technique was superior.
Schwarz et al, Injury, 1994.	Review of ten children with vertebral fractures and kyphotic angulation.	III	The kyphotic angulation remained unchanged or worsened when external immobilization alone (n=7) or dorsal fusion (n=1) was employed. Only those undergoing a ventral fusion (n=2) had a stable reduction of the kyphotic deformity.
Nypaver M, Treloar D, Ann Emer Med, 1994	40 children were placed on spine boards and observers judged whether the cervical spine was in the "neutral" position.	III	Children less than eight years of age required torso elevation to achieve neutral alignment Children four years of age or younger required the greatest amount of elevation.
Laham JL et al, Pediatr Neurosurg, 1994.	Divided head-injured children into high (less than two years of age, non-communicative, or with neck pain) and low risk groups for cervical spine injury.	III	No cervical spine injuries detected in the low-risk group. Ten injuries (7.5%) were detected in the high-risk group.
Fotter R et al, Ped Radiol, 1994.	Report of birth-related spinal cord injuries imaged with ultrasound and MRI	III	A neonate with complete injury had normal plain radiographs with spinal ultrasound showing inhomogeneous echogenicity and disrupted cord surface. A neonate with an incomplete injury had intact cord surface with increased cord echogenicity. MRI corroborated these findings.
Marks DS et al, Arch Orthop Trauma Surg, 1993	Review of eight children, ages three months to 12 years, immobilized in a halo jacket for six weeks to 12 months (mean two months).	III	The only complication was a jacket change was required for a foreign body (coin). Only three of these children had cervical instability.
Shacked I et al, Clin Orthop, 1993	Retrospective review of six children (3 to 14 years-old) with cervical spine injuries treated via an anterior approach.	III	Autograft without instrumentation following corpectomy was used. They were stabilized postoperatively with hard collar or Minerva cast. All with solid fusions, good alignment, and normal cervical growth. Follow-up three to eight years.

Authors & Year	Description of Study	Class of Data	Conclusions
Grogaard et al, Arch Orthop Trauma Surg, 1993.	Atlanto-axial rotary subluxation described in nine children. Eight diagnosed within five days, one diagnosed after eight weeks.	III	Eight children were treated successfully with "mild" traction and then a collar for four to six weeks. The one child presenting late required one week of traction for reduction. There were two redislocations. All eventually healed in alignment without surgery.
Mandabach M et al, Pediatr Neurosurg, 1993	Thirteen children with axis injuries were reviewed. Ten were treated primarily with closed reduction and halo immobilization	III	Eight of the ten treated primarily with closed reduction and halo immobilization fused. Two required surgical stabilization and fusion.
MacKinnon JA et al, J Pediatr, 1993.	Retrospective case series of 22 neonates with birth-related spinal cord injuries. They excluded neonates with SCIWORA.	III	All 14 with high cervical injuries had cephalic presentations with attempted forceps rotation. All six with cervicothoracic injuries had breech presentations. Both neonates with thoracolumbar injuries were premature.
Rossitch E & Oakes WJ, Pediatr Neurosurg 1992	Retrospective review of five neonates with perinatal spinal cord injury. No flexion/extension views reported.	III	4 of the five had no abnormality on static spinal radiographs. Respiratory insufficiency and hypotonia were common signs. Myelograms were unrevealing. All three with high cervical injuries died by age three years.
Osenbach RK & Menezes AH, Neurosurgery 1992	Retrospective review of 179 children with spinal injuries	III	59 (33%) underwent surgical treatment for irreducible unstable injuries. 83% of those treated surgically were nine years of age or older. No child with complete or severe partial myelopathy regained useful function.
Rathbone D et al, J Ped Orthop, 1992	Retrospective review of 12 children with presumed spinal cord concussion during athletics were investigated for the presence of cervical stenosis.	III	3 had a Torg ratio < 0.8 and four had a canal AP diameter < 13.4 mm. MRI was not used to evaluate for stenosis.
Hamilton MG & Myles ST, J Neurosurg, 1992	Retrospective review of all pediatric spinal injuries over 14 year period. 73 children had cervical injuries.	III	Surgery was performed in 26% of children. 13% of children with fracture and no subluxation, 50% with subluxation alone, and 57% with fracture and subluxation were treated surgically. Of 39 children with complete myelopathy, four improved one or two Frankel grades.
Schafermeyer RW et al, Ann Emer Med, 1991.	Forced vital capacity (FVC) was studied in healthy children when upright, supine, and supine taped to a spinal board.	III	Taping the child to the spinal board caused FVC to drop to 41% to 96% (mean 80%) of supine FVC.
Bohn D et al, J Trauma, 1990.	16 of 19 children presenting with absent vital signs or severe hypotension unexplained by blood loss underwent postmortem examination	III	13 of 16 had cord laceration or transection. Two of these children had a normal cervical radiograph.

<b>Authors &amp; Year</b>	<b>Description of Study</b>	<b>Class of Data</b>	<b>Conclusions</b>
Gaskill S & Marlin A, <i>Pediatr Neurosurg</i> , 1990	Six children ages two to four years were placed in Minerva jackets for cervical spine instability.	III	One child had skin breakdown of the chin. Eating and other daily activities were not impaired. Two were placed in Minerva jackets after complications of halo ring and vest immobilization.
Phillips et al, <i>J Bone Joint Surg</i> , 1989	A review of 23 children with C1-2 rotary subluxation.	III	16 children seen within one month of onset had either spontaneous reduction or reduced with traction. Of the seven children presenting with greater than one month of symptoms, one subluxation was irreducible, and four had recurrent subluxations.
Kawabe et al, <i>J Pediatr Orthop</i> , 1989.	Review of the radiology of 17 children with C1-2 rotary subluxation	III	Classified according to Fielding & Hawkins as ten type I, five type II, two type III, and no type IV.
Benzel et al, <i>J Neurosurg</i> , 1989.	A comparison of cervical motion of injured patients (only one child) immobilized in halo and Minerva jackets.	III	The Minerva jacket allowed less motion than the halo jacket at every level except C1-2.
Baum, et al, <i>Spine</i> , 1989.	A review comparing the halo complications 13 children and 80 adults.	III	39% complication rate in children versus 8% in adults. The children had four pin-site infections and one inner table skull pin penetration.
Mubarak SJ et al, <i>J Ped Ortho</i> , 1989	Review of three children less than two years-old who were placed in halo rings for two to three ½months.	III	Ten pins tightened “finger-tight” in a seven month-old, and two in/lb in a 16 and 24 month-old. Two of three developed minor pin site infections necessitating pin removal.
Herzenberg JE et al, <i>J Bone Joint Surg</i> , 1989	Reported ten children less than seven years of age with cervical spine injuries positioned on a flat backboard.	III	The injuries were anteriorly angulated or translated when on a flat backboard because the head was in forced into flexion. Elevating the torso allowed for more neutral alignment and reduction of the injured segment.
Evans & Bethem, <i>J Ped Ortho</i> , 1989	Review of 24 consecutive cervical spine injuries in children 18 years-old or less.	III	Half of the children had injuries at C3 or above. One child was treated with laminectomy and two with fusion. Fractures healed in 21 of 22 with nonoperative therapy.
Birney TJ and Hanley EN, <i>Spine</i> , 1988	Retrospective review of 61 children with cervical spine injuries. 23 of these injuries were C1-2 rotary subluxation.	III	Rotary subluxation unassociated with neurological deficit. The deformity resolved with halter traction (n=10) or cervical bracing . One child had a recurrence. A child with transverse ligament disruption was treated successfully with a soft collar only.
Hadley MN et al, <i>J Neurosurg</i> , 1988	Retrospective review of 122 children with spinal injuries. There were 97 cervical injuries.	III	Only 12 cervical injuries were treated surgically.
Huerta C et al, <i>Ann Emer Med</i> , 1987.	They evaluated the immobilization of commercially available infant and pediatric cervical collars.	III	No collar used alone provided acceptable immobilization. The use of a modified half-spine board, rigid collar, and tape provided the best immobilization.

<b>Authors &amp; Year</b>	<b>Description of Study</b>	<b>Class of Data</b>	<b>Conclusions</b>
Pennecot GF et al, J Ped Ortho, 1984.	Review of 16 children with ligamentous injuries of the cervical spine. Five with C1-2 injuries.	III	Of the 11 children with injuries below C2, eight underwent surgical stabilization. They recommended a three-month trial of external immobilization in children with ligamentous injuries but no neurological deficit or dislocation.
El-Khoury et al, J Bone Joint Surg, 1984	A review of three children with C1-2 rotary subluxation.	III	All three were treated successfully with traction or manual reduction within 24 hours of presentation. One child had recurrent subluxation the next day and was treated successfully with manual reduction. External orthoses were used for ten weeks, three, and four months, respectively.
Koop SE et al, J Bone Joint Surg, 1984	Retrospective review of 13 children with cervical instability treated with posterior arthrodesis and halo immobilization. Only three had traumatic lesions.	III	One failed fusion when bank-bone was used. Others successfully fused with autogenous iliac crest or rib. Internal wiring used in two children. Average halo immobilization was 150 days.
Sherk HH et al, J Bone Joint Surg (Am),1978.	Report of 11 children with odontoid injuries, and review of 24 from the literature.	III	Majority of injured odontoids are angled anteriorly. All but one child was treated successfully with external immobilization.
Fielding & Hawkins, J Bone Joint Surg, 1977.	The radiographic findings of seventeen patients with atlanto-axial rotary fixation are described and classified into four types.	III	Four classes of C1-2 rotary subluxation were described, types I-IV. Type I: odontoid acts as pivot with competent transverse ligament; type II: one lateral articular process acts as pivot with up to five mm of anterior displacement; type III: both C1 inferior facets are subluxed anteriorly with greater than five mm of anterior displacement which suggests an incompetent transverse ligament; type IV: posterior displacement with absent or incompetent odontoid.
Gaufin & Goodman, J Neurosurg, 1975.	A review of three children less than 20 months-old with cervical spine injuries. Two of these children were treated with traction delivered via 22 gauge wire placed through bilateral parietal burr holes.	III	Successful traction applied to the ten week-old and 16 month old child. Up to nine pounds was used in the 10-week-old infant. No complications were encountered with the traction in place for 14 and 41 days, respectively.

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## **SPINAL CORD INJURY WITHOUT RADIOGRAPHIC ABNORMALITY (SCIWORA)**

### **RECOMMENDATIONS**

#### **Diagnosis:**

Standards: There is insufficient evidence to support diagnostic standards.

Guidelines: There is insufficient evidence to support diagnostic guidelines.

Options:

- Plain spinal radiographs of the region of injury and CT scan with attention to the suspected level of neurological injury to exclude occult fractures are recommended.
- MR of the region of suspected neurological injury may provide useful diagnostic information.
- Plain radiographs of the entire spinal column may be considered.
- Neither spinal angiography nor myelography is recommended in the evaluation of patients with SCIWORA.

#### **Treatment:**

Standards: There is insufficient evidence to support treatment standards.

Guidelines: There is insufficient evidence to support treatment guidelines.

Options:

- External immobilization is recommended until spinal stability is confirmed flexion and extension radiographs.
- External immobilization of the spinal segment of injury for up to 12 weeks may be considered.
- Avoidance of “high-risk” activities for up to six months following SCIWORA may be considered.

**Prognosis:**

Standards: There is insufficient evidence to support prognostic standards.

Guidelines: There is insufficient evidence to support prognostic guidelines.

Options: MRI of the region of neurological injury may provide useful prognostic information about neurological outcome following SCIWORA.

**RATIONALE**Diagnosis:

Pang and Wilberger defined the term SCIWORA (Spinal Cord Injury Without Radiographic Abnormality) in 1982 as “objective signs of myelopathy as a result of trauma” with no evidence of fracture or ligamentous instability on plain spine radiographs and tomography.(11) In their original manuscript they cautioned, “that if the early warning signs of transient symptoms could be recognized and promptly acted upon before the onset of neurological signs, the tragic fate of some of these children might be duly averted”.(11) Hamilton and Myles, Osenbach and Menezes, and Pang and Wilberger, (8,9,11) have documented the delayed onset of SCIWORA in children as late as four days following injury. Therefore, a concern is whether a child with a normal neurological examination, but with a history of transient neurological symptoms or persisting subjective neurological symptoms referable to traumatic myelopathy should be assigned the diagnosis of SCIWORA and managed accordingly, despite the absence of “objective signs of myelopathy.”

Pang and Pollack have recommended obtaining a CT scan focused at the neurological level of injury to exclude an occult fracture in a child with a neurological deficit referable to the spinal cord without abnormalities on plain radiographs of the spine.(12) In addition, dynamic flexion and extension radiographs or fluoroscopy have been advocated to exclude pathological

intersegmental motion consistent with ligamentous injury without fracture. If paraspinous muscle spasm, pain, or uncooperation prevents dynamic studies, they recommended external immobilization until the child can flex and extend the spine for dynamic x-ray assessment. The finding of fracture, subluxation, or abnormal intersegmental motion at the level of neurological injury excludes SCIWORA as a diagnosis. In the initial report by Pang and Wilberger, one of 24 children showed pathological motion on initial dynamic radiographs.(11) By their own definition of SCIWORA, this one child would not be diagnosed with SCIWORA because the initial flexion and extension radiographs were abnormal. While concern exists for the development of pathological intersegmental motion in children with SCIWORA following normal flexion and extension studies, there has not been documentation of such instability ever developing.

Magnetic resonance (MR) imaging findings in children with SCIWORA have spanned the spectrum from normal to complete cord disruption, along with evidence of ligamentous and disc injury in some.(3,6) Possible roles for MR of children with SCIWORA include: 1) exclude compressive lesions of the cord or roots or ligamentous disruption that might warrant surgical intervention, 2) guide treatment regarding length of external immobilization, and/or 3) determine when to allow patients to return to full activity.

#### Treatment:

Because there exists no subluxation or malalignment in SCIWORA the mainstay of treatment has been immobilization and avoidance of activity that may lead to either exacerbation of the present injury or increase the potential for recurrent injury. Medical management issues such as blood pressure support and pharmacological therapy are of concern to this population as well, and have been addressed in other guidelines. (Of note, the often-cited prospective studies

of pharmacological therapy in the treatment of acute spinal cord injury did not include children younger than 13 years of age). (1)

Pang and Pollack have recommended 12 weeks of external immobilization to allow adequate time for the healing of the presumed ligamentous strain/injury and to prevent exacerbation of the myelopathy.(12) It is unclear however, what role immobilization plays in this population once dynamic radiographs have displayed no instability. The length of and even the need for immobilization remain debatable given the current literature. If the incidence of delayed pathological intersegmental motion in children with SCIWORA who have been proven to have normal dynamic radiographs approaches zero, then the role of spinal immobilization for SCIWORA patients needs to be considered in light of the available literature. If physiological motion (normal) of the spinal column can potentiate spinal cord injury (SCIWORA), in these patients when there is no malalignment, subluxation, or lesion causing cord compression, then immobilization is warranted in these patients.

#### Prognosis:

SCIWORA has been shown to be associated with a high incidence of complete neurological injuries, particularly in children less than nine years of age. Hadley, et al, reported four complete injuries in six children less than ten-years-old with SCIWORA.(7) The regions of complete injury tend to be cervical and upper thoracic. Pang found the presenting neurological examination to relate strongly to outcome.(11) There is some data to suggest that MR abnormalities (or lack of abnormalities) of the cord may be more predictive of outcome than presenting neurological status.(3,6) Because no child has been documented to develop spinal instability following the diagnosis of SCIWORA, and has by definition, normal flexion and extension radiographs, there has been little impetus to define predictors of instability. On the

other hand, children have been documented to suffer recurrent SCIWORA (13), and predictors of a “high-risk” sub-group of children with SCIWORA for recurrent injury may exist.

## **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”: “pediatric” and “SCIWORA.” Approximately 145 citations were acquired. Non-English language citations were deleted. Articles written in English were reviewed for those that identified children that incurred a spinal cord injury without radiographic abnormality (SCIWORA). Those articles that described the clinical aspects and management of children with SCIWORA were used to generate these guidelines. Case reports were excluded from review. Of the fifteen articles meeting selection criteria, none were Class I or Class II studies. All were case series representing Class III data. They are summarized in Evidentiary Table format.

## **SCIENTIFIC FOUNDATION**

One concern is whether the child with a normal neurological examination and either a history of transient neurological deficit (i.e. paraparesis or quadriparesis), or persisting subjective symptoms (i.e. numbness or dysesthesias) would be a candidate for the diagnosis of SCIWORA. Pang and Wilberger described 13 of their 24 children to have a “latent” period from 30 minutes to four days (mean 1.2 days) before the onset of objective sensorimotor deficits.(11) All 13 of these children had transient subjective complaints at the time of their initial trauma that cleared within one hour prior to their neurological decline. Those who developed mild neurological deficits often improved to normal, while those that developed severe neurological deficits were

often left with permanent neurological dysfunction. Hamilton and Myles, Osenbach and Menezes, and Pang and Pollack as well reported a 22, 23, and 27% incidence of delayed onset of myelopathy within their series of children with SCIWORA, respectively.(8,9,12) Dickman, et al, Eleraky and associates, and Hadley, et al, described no child having a latent period of neurological normalcy following injury.(4,5,7) The observations of delayed deterioration by different investigators however, raises the concern that any child presenting with a history of transient neurological deficit or symptoms following an appropriate mechanism of injury may be considered for the diagnosis of SCIWORA, despite the absence of objective evidence of myelopathy upon initial neurological examination.

Pang and Wilberger had one child out of 24 demonstrate what was considered to be pathological intersegmental motion on flexion and extension radiographs one week after injury, following resolution of neck pain and paraspinous muscle spasm.(11) By definition this child would not be considered to have had SCIWORA, because the initial flexion and extension radiographs were abnormal. This child was treated successfully with external immobilization alone for eight weeks. No child with SCIWORA has been documented in the literature to have had normal dynamic radiographs and then subsequently develop intersegmental instability.

In 1994 a series of seven children with SCIWORA were demonstrated to have ligamentous, disc, and intramedullary abnormalities identified on MRI imaging.(6) Soft tissue findings consisted of anterior longitudinal ligament disruption in association with a hyperextension injury, posterior longitudinal ligament disruption and a noncompressive C2-3 disc herniation associated with lateral flexion, and one case of C6-7 disc abnormality associated with hyperflexion. Intramedullary findings reported included cord transection and rostral stump hemorrhage, severe hematomyelia, a minor intramedullary hemorrhage, and edema without

hemorrhage. Davis, et al, described seven children with SCIWORA who were imaged with MRI.(3) They described no abnormalities of muscles, ligaments, or discs, but did correlate the presence of intramedullary hemorrhage or cord “infarction” with permanent neurological deficit. The lack of intramedullary findings correlated with a normal neurological outcome. Dickman, et al, commented on seven children with SCIWORA who were imaged with MR. Five of the seven studies revealed no abnormality and two studies documented intramedullary signal changes.(4) Osenbach and Menezes commented in their series of childhood SCIWORA that MRI and CT-myelography performed on their patients did not demonstrate a single compressive lesion.(9) In addition, they performed spinal arteriograms in four of five children with thoracic SCIWORA and found no angiographic abnormalities. Rossitch and Oakes performed myelograms on neonates with SCIWORA from birth injury and found no abnormalities that changed their management.(16) Hadley et al, obtained MR imaging prior to 1988 on five children with SCIWORA and identified no abnormalities.(7) These results need to be viewed in the context of the technology available at the time of study.

There has been no report of any situation in which the care of a child with SCIWORA has been altered by the results of MR and/or myelography imaging studies. No child with MR documented ligamentous injury and SCIWORA has developed spinal instability, early or delayed. There has been no correlation between the ligamentous findings on MRI in SCIWORA patients and subsequent spinal instability to date. The appearance of the spinal cord on MR does provide prognostic information regarding ultimate neurological outcome.

Hadley, et al, noted a 16% incidence of multiple non-contiguous injuries of the spine or spinal cord in children with any type of spinal column or spinal cord injury.(7) Ruge, et al, had a similar incidence (17%) of multiple levels of spinal injury in children.(17) While neither of

these two studies dealt with an isolated population of children with SCIWORA, they do provide consistent observations that one in six children with spinal trauma will have multiple levels of injury. Pang and Wilberger reported one of 24 children with a second level injury (L2 Chance fracture) who had a T6 neural injury (SCIWORA), but they did not obtain complete spine radiographs on every child.(11) Because of these observations one should consider radiographs of the entire spinal column when any traumatic spinal injury, SCIWORA or otherwise, is identified in a child.

In the initial series of children with SCIWORA reported by Pang and Wilberger, treatment routinely consisted of four weeks of external immobilization with a “cervical collar” for cervical injuries.(11) In cases of thoracic injury, if repeat plain radiographs showed no abnormality following one week of bed rest, the child was mobilized without a brace. In a later report in 1989, Pang and Pollack recommended 12 weeks of external immobilization for SCIWORA patients to allow for healing of the presumed ligamentous strain/injury, and to prevent exacerbation of the myelopathy.(12) They also advocated external immobilization for this time frame to prevent recurrent injury during the healing phase. They reported seven children who sustained recurrent SCIWORA of greater severity with lesser degrees of force when external immobilization was removed before 12 weeks time, or they were allowed to participate in activities against physician instructions within six months of the initial injury. For these reasons, they recommend 12 weeks of external immobilization and 12 additional weeks of activity restriction following SCIWORA.

Dickman et al, Eleraky et al, and Hadley and colleagues reported no neurological deterioration in any patient with SCIWORA following admission or discharge.(4,5,7) None of these three reports described the length of time children with SCIWORA were immobilized. It

has not been routine among treating physicians to prescribe 12 weeks of immobilization for children with SCIWORA.(2) While the single report by Pollack and Pang describes recurrent SCIWORA within 12 weeks of the original injury, this has not been validated by other observations.(13) Because MR evaluation was not available for those with recurrent injury, it is not known whether certain MR characteristics (i.e. ligamentous disruption) could predict an “at risk” group for recurrent SCIWORA.

While Pang and Wilberger reported that in their series, neurological outcome correlated with the presenting neurological status, the MR appearance of the spinal cord has been shown to be predictive of neurological outcome in children with SCIWORA.(3,6,11) Absence of signal change within the cord is associated with an excellent outcome. Signal change consistent with edema or micro-hemorrhages, but not frank hematomyelia, is associated with significant improvement of neurological function over time. The presence of frank hematomyelia or cord disruption is associated with a severe, permanent neurological injury.(3,6) The correlation of neurological outcome with spinal cord MR findings in SCIWORA remain consistent with the findings in much larger numbers of patients with spinal cord injury (non-SCIWORA) who have been studied with MR.(15)

## **SUMMARY**

Children presenting with a history of transient neurological signs or symptoms referable to traumatic myelopathy despite the absence of objective evidence of myelopathy and normal radiographs may develop SCIWORA in a delayed fashion.

No child with SCIWORA has developed pathological intersegmental motion with instability after demonstrating normal flexion and extension radiographs.

MR has not identified any lesion in a child with SCIWORA where the management scheme would be changed by the results of the MR. Similarly, no child with MRI documented ligamentous injury and SCIWORA has developed evidence of spinal instability.

Hard collar immobilization for patients with cervical level SCIWORA for 12 weeks and avoidance of activities that encourage flexion and extension of the neck for an additional 12 weeks has not been associated with recurrent injury.

The spinal cord findings on MRI imaging provide prognostic information regarding long-term neurological outcome in patients with SCIWORA.

Myelography and angiography have no defined role in the evaluation of children with SCIWORA.

## **KEY ISSUES FOR FUTURE INVESTIGATION**

The treatment endpoints of spinal immobilization and activity restriction for patients with SCIWORA have been arbitrarily chosen. MR may be helpful to guide the length of time a child is immobilized and activities restricted. The absence of ligamentous injury by MR may indicate that there is no need for external immobilization or activity restriction. It has been observed that recurrent SCIWORA can occur despite no evidence of spinal instability. An investigation that obtained MR imaging on all children with SCIWORA and followed their clinical status longitudinally, may highlight the utility of MR in the management of children who go on to develop recurrent SCIWORA.

The literature provides little guidance as to the likelihood for subsequent catastrophic injury in children presenting with SCIWORA of any severity who are found to have a pre-existing spinal or neurological abnormality such as congenital cervical stenosis or a Chiari

malformation.(14) Longitudinal clinical follow-up of SCIWORA patients of this type may provide information to appropriately counsel these children.

There are no data to elucidate the role of age in the success or failure of various treatments for this condition. This could be undertaken in a longitudinal study of a patient population of reasonable size.

Serious attempts to address the topics above cannot be forthcoming from a single institution or investigator because of the relatively low numbers of children who sustain SCIWORA annually (10,18). A multiple-institution protocol directed study of SCIWORA patients may provide answers to some of the questions which accompany this unique spinal cord injury subtype.

## EVIDENTIARY TABLES

Authors & Year	Description of Study	Class of Data	Results
Eleraky MA et al, <i>J Neurosurg (Spine)</i> 2000	Retrospective review of 102 children with cervical spinal injuries. Young (0-9 years) compared to older children. MR performed in 12 of 18 children with SCIWORA.	III	SCIWORA in 18%. MR findings did not alter management (external immobilization).
Turgut M et al, <i>Eur Spine J</i> , 1996	Retrospective review of 11 of 82 children with spinal injuries with SCIWORA	III	SCIWORA represented 13% of spinal injuries in children.
Grabb PA & Pang D, <i>Neurosurgery</i> 1994	Retrospective review of seven children with SCIWORA underwent MR. Neurological status at presentation and follow-up was correlated to MR findings.	III	No compressive lesions found. Prognosis correlated with MR findings. Hematomyelia involving greater than 50% of cord diameter was associated with permanent severe deficits. Lesser degrees of hematomyelia and edema only were associated with incomplete recovery, and normal MR predicted full recovery.
Davis PC et al, <i>AJNR</i> , 1993	Retrospective review of 15 children with spinal cord injury underwent MR 12 hours to two months after injury. seven with SCIWORA	III	MR correlated with prognosis. Hemorrhagic cord contusions and cord “infarction” were associated with permanent deficits. No compressive lesions in SCIWORA cases. Normal MR was associated with no myelopathy
Hamilton MG & Myles ST, <i>J Neurosurg</i> , 1992	Retrospective review of 174 pediatric spinal injuries over 14 year period	III	SCIWORA represented 13% of spinal injuries. Of children aged 0-9 years with spinal injuries 42% had SCIWORA, whereas children aged 10-14 years only 14% had SCIWORA.
Osenbach RK & Menezes AH, <i>Neurosurgery</i> 1992	Retrospective review of 34 of 179 children with spinal injuries with SCIWORA	III	SCIWORA represented 19% of spinal injuries in children. Younger children (<9 years-old) had higher incidence of SCIWORA.
Rathbone D et al, <i>J Ped Orthop</i> , 1992	Retrospective review of 12 children with presumed spinal cord concussion during athletics was investigated for the presence of cervical stenosis.	III	3 had a Torg ratio < 0.8 and four had a canal AP diameter <13.4 mm. MRI was not used to evaluate for stenosis.
Rossitch E & Oakes WJ, <i>Pediatr Neurosurg</i> 1992	Retrospective review of five neonates with perinatal spinal cord injury. four of the five had no abnormality on static spinal radiographs. No flexion/extension views reported. Myelograms were unrevealing.	III	Perinatal spinal cord injury often has normal radiographs. The neonates are often initially misdiagnosed. Respiratory insufficiency and hypotonia are common signs.
Dickman CA et al, <i>J Spinal Disorders</i> , 1991	Retrospective review of 26 children with SCIWORA over 19-year period. Clinical and epidemiological features were analyzed.	III	SCIWORA 16% of spinal injuries in children. MVA was most common mechanism. Seven children had MR. five were normal studies, two showed cord signal abnormalities. Younger children tended to have more severe injuries.

Authors & Year	Description of Study	Class of Data	Results
Osenbach RK & Menezes AH, <i>Pediatr Neurosci</i> , 1989	Retrospective review of 31 children with SCIWORA	III	26 cervical and five thoracic injuries. Complete cord injury in 12. Delayed onset of deficits in 7. No surgical lesions found by MR or CT-myelography. Spinal angiograms done in four thoracic cases were normal. No delayed instability at follow-up.
Pang D & Pollack IF, <i>J Trauma</i> , 1989	Retrospective review of 55 children with SCIWORA (43 cervical, 12 thoracic). Clinical profiles reported to illustrate syndrome	III	22 "severe" injuries 33 "mild" injuries <8 yo associated with more severe injuries 8 cases of recurrent injury from three days to ten weeks after initial injury No recurrent injuries with 12 weeks of Guilford Brace
Hadley MN et al, <i>J Neurosurg</i> , 1988	Retrospective review of 122 children with spinal injuries. Young (0-9 years) compared to older children.	III	17% with SCIWORA. Higher incidence of SCIWORA in 0-9 year-olds versus 10-16 year-olds. five studied with MRI, no abnormalities detected.
Pollack IF et al, <i>J Neurosurg</i> , 1988	Retrospective review of eight children with recurrent SCIWORA compared to 12 children treated with longer immobilization	III	Recurrent SCIWORA occurred from three days to ten weeks after initial injury. Recurrent injuries were more severe. No recurrent injuries with 12 weeks of Guilford Brace.
Ruge JR et al, <i>J Neurosurg</i> , 1988	Retrospective review comparing 0-3 year-olds to 4-12 year-olds with spinal injury	III	N=47, 21% with SCIWORA
Pang D & Wilberger JE, <i>J Neurosurg</i> , 1982	Retrospective review of 24 children with SCIWORA	III	One child with instability on flex/ext at one week

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## DIAGNOSIS AND MANAGEMENT OF TRAUMATIC ATLANTO-OCCIPITAL DISLOCATION INJURIES

### RECOMMENDATIONS

#### **Diagnostic:**

Standards: There is insufficient evidence to support diagnostic standards.

Guidelines: There is insufficient evidence to support diagnostic guidelines.

Options:

- A lateral cervical radiograph is recommended for the diagnosis of AOD. If a radiological method for measurement is used, the basion-axial interval-basion dental interval (BAI-BDI) method is recommended.
- The presence of upper cervical prevertebral soft tissue swelling on an otherwise non-diagnostic plain radiograph should prompt additional imaging.
- If there is clinical suspicion of AOD, and plain radiographs are non-diagnostic, CT or MR imaging is recommended, particularly for the diagnosis of non-Type II dislocations.

#### **Treatment:**

Standards: There is insufficient evidence to support treatment standards.

Guidelines: There is insufficient evidence to support treatment guidelines.

Options: Treatment with internal fixation and arthrodesis using one of a variety of methods is recommended. Traction may be used in the management of patients with AOD, but is associated with a ten percent risk of neurological deterioration.

### RATIONALE

Although traumatic atlanto-occipital dislocation (AOD) was perceived to be an uncommon injury resulting in frequent death, improvements in emergency management of the

patient in the field, rapid transport, and better recognition have resulted in more survivors of AOD in the past two decades. Infrequent observation of patients with AOD and missed diagnoses may impair outcomes of patients with this unusual injury.(44) An assimilation of the reported experiences of clinicians evaluating and managing AOD may facilitate development of diagnostic and treatment options for this traumatic disorder. Specific questions that were evaluated include the sensitivity of plain radiographs, CT, and MR imaging in the diagnosis of AOD, as well as the safety and efficacy of various treatment modalities for AOD, including no treatment, traction, external immobilization, and internal fixation with fusion.

## **SEARCH CRITERIA**

A National Library of Medicine computerized literature search of publications from 1966 to 2001 was performed using the following headings: “atlanto-occipital joint” and “dislocation”. An exploded search of these headings led to 690 and 86,205 citations, respectively. A subset of 233 citations contained both headings. The references of the identified articles were reviewed to identify additional case reports. Since fewer than 100 cases of survivors of AOD were identified, even single case reports were considered, provided that basic inclusion criteria were met. The articles were reviewed using the following criteria for inclusion in diagnosis: human survivors, type of traumatic atlanto-occipital dislocation, and plain radiographic findings. The articles were also reviewed using the following criteria for inclusion in treatment: human survivors, type of traumatic AOD, management, and outcome. The observations from the reports were combined because the usual methods for analysis were precluded by the infrequent observation of this injury. The type of dislocation was classified according to Traynelis et al (51) into Type I (anterior), Type II (longitudinal), and Type III (posterior) dislocations. Lateral, rotational and multi-directional dislocations that could not be classified into one of these three

types were considered separately and are notated as “Other Type”. The duration of follow-up ranged from several weeks to four years. Of the articles meeting the diagnostic selection criteria reported, 48 articles with 79 patients provided data on 29 Type I, 32 Type II, four Type III, and 14 other types of AOD. Two of these articles (10,44) included one patient each from two previously published individual case reports.(41,42) Of the articles meeting the treatment selection criteria, 43 articles with 62 patients provided data on 24 Type I, 23 Type II, three Type III, and 12 other types of AOD. Two of these articles (10,44) included one patient each from two previously published individual case reports.(41,42) All articles contained Class III medical evidence consisting of either single case studies or small case series with no report containing more than six patients. The information provided by these reports was compiled and scrutinized and make up the basis for this guideline. Summaries of these reports are provided in Evidentiary Table format.

## **SCIENTIFIC FOUNDATION**

### **Diagnosis:**

A variety of radiographic measurements has been proposed for the diagnosis of AOD on a lateral cervical radiograph (Figure 1). A displacement of more than ten mm between the basion and dens is considered abnormal by Wholey et al.(53) A ratio of the basion-posterior atlas arch distance divided by the opisthion-anterior atlas arch distance greater than one is considered abnormal by Powers et al.(43) A distance of more than 13 mm between the posterior mandible and anterior atlas or 20 mm between the posterior mandible and dens is considered abnormal by Dublin et al.(12) Failure of a line from the basion to the axis spinolaminar junction to intersect C2, or a line from the opisthion to the posterior inferior corner of the body of the axis

to intersect C1, are considered abnormal by Lee et al.(32) Finally, a displacement of more than 12mm or less than minus four mm between the basion and posterior C2 line, or a displacement of more than 12 mm from the basion to dens (two mm more than the Wholey recommendation) is considered abnormal by Harris et al.(24,25) A comparative study by Lee found a 50% sensitivity of the Wholey method, 33% sensitivity of the Power's ratio, and a 25% sensitivity of the Dublin method. The authors applied their X-line method with a 75% sensitivity.(32) Although neither the Power's ratio nor X-line method could be applied in nearly half their patients, a comparative study by Harris et al found a 60% sensitivity of the Power's ratio, a 20% sensitivity of the Lee method, and 100% sensitivity of the BAI-BDI method among those in whom the required landmarks could be identified.(25) Przybylski et al (44) reported failure to diagnose AOD in two of five patients with the Power's ratio, one of five patients with the X-line method, and in two of five with the BAI-BDI method. No radiographic method reviewed has complete sensitivity. The BAI-BDI method proposed by Harris et al (which incorporates the basion-dens distance described by Wholey) is at present the most reliable means to diagnose AOD on a lateral cervical spine radiograph.

Many of the case reports and case series in the literature do not describe the method(s) used for diagnosis of AOD. Since the most sensitive method was proposed by Harris et al (25) in 1994, this method was probably not used for many of the evaluations. Although retrospectively a diagnosis was possible on the first lateral radiograph in 60 of 79 patients, (sensitivity = 0.76) the diagnosis was actually made in only 45 of the 79 patients (sensitivity = 0.57) on the first lateral radiograph. Of the fifteen in whom the diagnosis could have been made on the first lateral radiograph, three were not stratified by type, whereas eleven of the remaining twelve were not Type II dislocations. A second lateral radiograph (nine cases), tomography (one

case), fluoroscopy (two cases), CT (two cases), and MRI (five cases) were required for diagnosis in 19 of 79 patients. The sensitivities stratified by type of dislocation are: Type I, 0.83 (24 of 29); Type II, 0.72 (23 of 32); Type III, 0.75 (three of four), Other Type, 0.71 (ten of 14). Since these data were obtained from case reports and small case series, comparison with the accuracy of plain radiographs in patients without AOD could not be performed. As a result, specificity, predictive values, and likelihood ratios can not be discerned from the available literature.

Of the fifteen patients in whom the diagnosis was missed on the initial plain radiographs, the initial neurological condition of three patients was unknown.(1) Of the remaining twelve patients, four were neurologically normal (one Type I, one Type III, two other type).(13,29) Two of those four patients originally reported as normal developed a monoparesis (one Type I, one other type).(7,49) Neither recovered completely. Eight of the remaining twelve patients had neurologic abnormalities from the outset, five of whom worsened. Four of the five transiently worsened, including one Type I injury patient with quadriplegia and Cranial Nerve IX, X, and XII palsies (9) who was only spastic at last follow-up. One patient with a Type I injury developed a hemiparesis that recovered.(27) One Type I injury patient developed quadriplegia who was hemiparetic at follow-up.(46) One lateral dislocation patient with paraparesis and torticollis recovered at last follow-up.(52) One patient (Type I) with a monoparesis initially experienced permanent worsening and was quadriplegic at follow-up.(54)

Although plain radiographs do not reliably identify AOD, the index of suspicion may be increased with the identification of prevertebral soft tissue swelling (STS). Although plain radiographs were obtained in all cases considered, the presence or absence of soft tissue swelling was described in only half. The sensitivity of soft tissue swelling is 0.90 (37 of 41 cases). Acute craniocervical CT imaging was performed in 40 of 79 patients with AOD. However, for 15 of

40 patients the authors did not report whether AOD was diagnosed by CT. The diagnosis of AOD was made by CT in 21 of 25 patients (sensitivity=0.84). Although no other CT findings were reported in 11 of 40 patients, 24 of the remaining 29 patients with AOD studied with CT had hemorrhages (19 subarachnoid hemorrhage, one subdural hemorrhage, four contusions). Five patients had no CT evidence of associated hemorrhage. Nine of fifteen patients in whom the diagnosis of AOD was missed on the first plain radiograph had subsequent acute CT imaging; eight had subarachnoid or other associated hemorrhage.(1,9,8,44) Craniocervical MR imaging was performed in 18 of 79 patients with AOD. The MR findings were not reported for four of the 18 patients studied. The diagnosis of AOD could be made in 12 of 14 cases studied with MRI (sensitivity=0.86).

In summary, physicians often miss the diagnosis of AOD on plain radiographs, (sensitivity=0.57) particularly in the circumstance of non-longitudinal dislocations (non-Type II). Although improved interpretation may increase sensitivity of plain x-rays to 0.76, additional imaging of the craniovertebral junction with CT or MRI is recommended in patients suspected of having AOD, given their superior sensitivity over plain radiographs. Other methods such as fluoroscopy, tomography, and myelography have also been used to confirm the diagnosis of AOD. Neurological abnormalities including lower cranial nerve paresis (particularly cranial nerves 6, 10, and 12), monoparesis, hemiparesis, quadriparesis, respiratory dysfunction including apnea, and complete high cervical cord motor deficits in the setting of normal plain spinal radiographs should prompt additional imaging with CT or MRI. The presence of prevertebral STS on plain radiographs, and subarachnoid hemorrhage on CT at the craniovertebral junction should prompt consideration of the diagnosis of AOD.

## **Treatment:**

Ten patients in the literature did not receive initial treatment for AOD, nine of whom were not correctly diagnosed until neurological worsening occurred.(7,8,10,20,46,48,49,52,54) Five of nine had Type I injuries and four of nine had other injury types. Four of nine had persistent deficits at last follow up which were worse in comparison with their exams on presentation.(7,10,49,54) Two of these patients were normal initially. At last follow-up, one had a CN X deficit with spasticity (Type I) (49) and one a monoparesis.(7) The other two patients had mild initial deficits. One patient with an initial CN VI palsy had a hemiparesis at last follow-up, (10) whereas another with an initial monoparesis was quadriplegic at follow-up (54). Five patients who worsened initially without treatment eventually improved from their initial neurological condition. Finally, one quadriplegic patient with Type II AOD (56) who was not treated improved to quadriparesis at last follow-up. In summary, failure to treat AOD resulted in worsening of all patients with incomplete injuries. Nearly half of these patients failed to improve to their presentation examination baselines.

Of twenty-one patients with AOD initially treated with traction, two worsened transiently and developed worsening quadriparesis and CN VI deficits. Both had resolution of their CN VI deficits but not of their quadriparesis. One patient had a Type II injury (40) and one patient had a rotational dislocation.(10) Four patients were initially normal and remained normal at follow up (2,4,16,33). The remaining fifteen patients had improved neurological function compared to their initial findings at last follow-up. Ten had Type I injuries, five had Type II injuries, two had Type III injuries, and two had other dislocations. In total, one of six patients with Type II injuries and one of three patients with other translational injuries had transient worsening with the use of craniocervical traction. In summary, traction for AOD has been reported to cause

occasional neurological worsening. In both circumstances, the worsening did not persist after discontinuation of traction. Because the frequency of neurological worsening with traction for AOD is approximately 10%, ten times higher than that for subaxial injuries, the use of traction should be considered with caution in patients with AOD.

Of nineteen patients initially treated with external immobilization excluding traction, eight were immobilized in anticipation of internal fixation and fusion and none worsened during the pre-surgical interval (one Type I, four Type II, three other type).(10,11,31,33,38,44) Of the remaining eleven patients treated with external immobilization alone excluding traction, four worsened transiently (three Type I, one Type II). (9,11,13,27) All subsequently underwent craniocervical fixation and fusion. Two of these patients were normal at follow up (one initially normal, one initially hemiparetic) and two were spastic (one initially quadriparetic and one hemiparetic). Of the remaining seven patients managed with external immobilization alone who did not worsen while in external immobilization, two patients managed in collars and one patient treated in a halo were unstable after six to 22 weeks of immobilization (one Type I, two Type II). Two were quadriplegic and one was normal. All three underwent internal fixation and fusion without change in their initial neurological condition at last follow-up. Only four patients with AOD were successfully treated with external immobilization alone (one Type I, two Type II, one other dislocation). Of the twenty-one patients initially treated with traction, six were subsequently managed with external immobilization and none developed neurological worsening. Two of the six (both Type I) remained unstable after three to five months of bracing and were subsequently treated with craniocervical fixation and fusion. Five of those six patients had improvement in their neurological condition at follow-up. The sixth patient remained normal.

In summary, five of thirteen patients with AOD who did not worsen neurologically while treated with external immobilization (with or without traction) failed to achieve bony union with stability without internal fixation and fusion. In addition, six patients transiently worsened with external immobilization (with or without initial traction). Factors affecting fusion or persistent non-union such as degree and type of displacement, patient age, and association with occipital condyle fractures could not be identified. Since eleven of forty patients (28%) managed with external immobilization either deteriorated neurologically or failed to achieve craniocervical stability without surgical internal fixation and fusion, treatment of AOD with external immobilization alone should be considered with caution.

Finally, nineteen patients in the literature were treated with planned early craniocervical fusion with internal fixation. Only one patient worsened neurologically following surgery. This patient with a Type II injury was normally initially and developed a CN X deficit which persisted at follow-up.(44) All but three of the remaining eighteen improved neurologically at follow-up. Four had Type I, ten had type II, and four had other types of dislocation. None of the patients treated with craniocervical fusion and internal fixation had late instability requiring reoperation or further treatment.

## **SUMMARY**

AOD is an uncommon traumatic injury which is difficult to diagnose and is frequently missed on initial lateral cervical radiographs. Patients who survive often have neurological impairment including lower cranial neuropathies, unilateral or bilateral weakness, or quadriplegia. Yet, nearly 20% of patients with acute traumatic AOD will have a normal

neurological examination on presentation. The lack of localizing features may impede diagnosis in the patient with a normal cervical radiograph. A high index of suspicion must be maintained in order to diagnose AOD. Prevertebral soft tissue swelling on a lateral cervical radiograph or craniocervical subarachnoid hemorrhage on axial CT have been associated with AOD and may prompt consideration of the diagnosis. Additional imaging including CT and MR may be required to confirm the diagnosis of AOD if plain radiographs are inadequate. All patients with AOD should be treated. Without treatment, nearly all patients developed neurological worsening, some of whom did not recover. Although treatment with traction and external immobilization has been used successfully in some patients, transient or permanent neurological worsening and late instability have been reported more often in association with these treatments compared to surgical treatment. Consequently, craniocervical fusion with internal fixation is recommended for the treatment of patients with acute traumatic AOD.

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

Although the use of external immobilization for AOD was often associated with late instability, several patients achieved stability without operative management. CT imaging with three dimensional reconstruction for more precise measurement of the magnitude of displacement and MR imaging for differentiation of partial and complete ligament tears from stretch injuries may be useful in identifying a subgroup of patients in whom stability might be achieved with external immobilization alone. Because AOD remains relatively infrequent, cooperative prospective collection of plain radiographic, CT and MR imaging data in patients with AOD is recommended to determine if a subgroup of patients with AOD can be treated with external immobilization alone with fewer occurrences of late instability.

## EVIDENTIARY TABLE I - Imaging Diagnosis of AOD

AUTHOR	AOD TYPE	DIAGNOSIS MADE BY	XRAY FINDINGS	CT FINDINGS	MR FINDINGS
Grabb 1999	I II II	Plain Xray Plain Xray MRI	STS, Power+ STS, Power- STS, Power-	Unreported None performed None performed	Part tear tectorial Tear Post. AOL Part tear tectorial
Naso 1997	I/II	Plain Xray	No mention STS	Unreported	Delayed study
Sponseller 1997	I II	Plain Xray (missed) Plain Xray	No mention STS No mention STS	None performed Unreported	None performed Brainstem contusion
Przybylski 1996 Pang 1980	I II II I/Lateral I/Lateral	MRI Plain Xray (missed) 2 <sup>nd</sup> plain Xray Plain Xray (missed) Plain Xray (missed)	Power/BDI/Xline- Power/BDI/Xline+ Power/BDI-,Xline+ Power/BDI/Xline+ Power/BDI/Xline+	SAH, - Dx SAH, +Dx SAH, +Dx Normal, Head only SAH, +Dx	BS contusion, +Dx BS contusion, +Dx None performed None performed None performed
Yamaguchi 1996	I	Plain Xray	No mention STS	SAH,+ tomo	BS Contusion,+Dx
Guigui 1995	I	Plain Xray	STS	+Dx	None performed
Ahuja 1994	I II II II I/II I/II	Fluoroscopy 5 Plain Xray (3 missed)	STS,Power- STS,Power+ STS,Power+ STS,Power+ STS,Power+ STS,Power+	SAH, unknown None performed SAH, +Dx SAH, unknown None performed SAH, +Dx	None performed None performed None performed None performed None performed None performed
Donahue 1994	I II II II	Plain Xray Plain Xray Plain Xray Plain Xray	STS STS, 5mm distract STS 6mm distract	None performed None performed None performed Intracerebral bleed	None performed None performed None performed None performed
Palmer 1994	II	CT	No mention STS	Unreported	CordContusion,+Dx
Dickman 1993 Papadououlos 1991	II Rotatory Rotatory II/Rotatory	Plain Xray CT MRI 2 <sup>nd</sup> Plain Xray	15mm distraction STS STS STS	None performed + Dx No blood, - Dx +Dx	None performed None performed Epidural, +Dx Epidural, +Dx
Harmanali 1993	II	Plain Xray	No mention STS	None performed	- Dx
Hosono 1993	I	Plain Xray (missed)	STS	Edema, head only	Delayed study
Matava 1993	II II II	Plain Xray Plain Xray Plain Xray	STS No mention STS No mention STS	Delayed study None, +DX SAH, +DX	None performed None performed BS Contusion
Nischal 1993	II II	Plain Xray Plain Xray	STS STS	BS contusion,- Dx - Dx	None performed None performed
Bundshuh 1992	I I	Plain Xray Plain Xray	STS STS, Power/Xline-	SAH, +Dx SAH	SAH, + Dx - Dx
Farley 1992	I	Plain Xray	STS, Power +	None performed	Cord contusion
Belzberg 1991	II	2 <sup>nd</sup> Plain Xray	STS	SAH, +Dx	None performed
Hladky 1991	II II	MRI MRI	No mention STS No STS	Contusion,head only Normal, Head only	+ Dx + Dx
Lee 1991	II I/Rotatory	Plain Xray Plain Xray	STS STS	SAH, +Dx + Dx	None performed None performed

<b>AUTHOR</b>	<b>AOD TYPE</b>	<b>DIAGNOSIS MADE BY</b>	<b>XRAY FINDINGS</b>	<b>CT FINDINGS</b>	<b>MR FINDINGS</b>
Maves 1991	II II III	Plain Xray Plain Xray Plain Xray	No mention STS No mention STS No mention STS	None performed None performed None performed	None performed None performed None performed
Montane 1991	I II II	Plain Xray 2 <sup>nd</sup> Plain Xray 2 <sup>nd</sup> Plain Xray	STS STS No STS	None performed None performed None performed	None performed None performed None performed
DiBenedetto 1990	I	Plain Xray (missed)	STS	ICH, +DX	None performed
Jones 1990	I	Plain Xray	No mention STS	+DX	Premedullary edema
Colnet 1989	Lat/rotatory	Tomography	Late study	SAH, +DX	Delayed study
Jevitch 1989	Lateral	Plain Xray (missed)	No mention STS	Delayed study	None performed
Hummel 1988	I	2 <sup>nd</sup> Plain Xray	No mention STS	Subdural, Head only	None performed
Zampella 1988	II	Plain Xray	No mention STS	SAH, Head only	Delayed study
Georgopoulou s 1987	I	Cineradiography	No mention STS	Delayed study	None performed
Bools 1986	I III	Plain Xray 2 <sup>nd</sup> Plain Xray	STS No mention STS	SAH, +DX None performed	None performed None performed
Collato 1986	I/lateral	Plain Xray (missed)	No STS	SAH, Head only	Delayed study
Putnam 1986	I	Plain Xray	STS, Powers +	SAH, +DX	None performed
Ramsay 1986	I	Plain Xray (missed)	No mention STS	None performed	None performed
Roy-Camille 1986	I I	Late Plain Xray Plain Xray	No mention STS STS	Delayed study None performed	None performed None performed
Zigler 1986	I	Plain Xray	No mention STS	None performed	None performed
Watridge 1985	Lateral	Plain Xray (missed)	No STS	Delayed study	None performed
Banna 1983	Rotatory	Plain Xray	No mention STS	+ Dx	None performed
Kaufman 1982	II II	Plain Xray Plain Xray	STS STS	None performed None performed	None performed None performed
Woodring 1981	I I	Plain Xray Plain Xray (missed)	No mention STS STS	None performed None performed	None performed None performed
Powers 1979	I II	Plain Xray 2 <sup>nd</sup> Plain Xray	Late study No mention STS	None performed None performed	None performed None performed
Rockswold 1979	II	Plain Xray	No mention STS	None performed	None performed
Eismont 1978	III	Plain Xray (missed)	No mention STS	None performed	None performed
Fruin 1977	I	Plain Xray	No mention STS	None performed	None performed
Page 1973	I	Plain Xray	STS	None performed	None performed
Evarts 1970	I	Plain Xray	No mention STS	None performed	None performed
Gabrielsen 1966	I	2 <sup>nd</sup> Plain Xray	STS	None performed	None performed
Farthing 1948	III	Plain Xray	No mention STS	None performed	None performed

1 patient was eliminated because the plain radiograph interpretation was not reported. Ferrara (1)

2 articles (11 patients) were eliminated because the type of dislocation was not reported. Cohen (1), Georgopolous (2/3), Hladky (1/3), Naso (1/2), Sun (6/6)

1 article (5 patients) was eliminated because individual patient data was not reported. Bulas (5/5)

**EVIDENTIARY TABLE II - Treatment of AOD**

<b>AUTHOR</b>	<b>TYPE</b>	<b>INITIAL EXAM</b>	<b>TREATMENT</b>	<b>OUTCOME</b>
Naso 1997	Mixed I/II	Quadriplegia	Supportive	Death five wks
Sponseller 1997	I	Normal	None (neuro worse), Traction, Fusion+Brace Brace failed (6wk), Fusion	Spastic, CN 10
	II	Normal		Normal
Przybylski 1996 Pang 1980	I	Quadriplegia	Collar + Fusion	Quadriplegia
	II	Quadriplegia	Halo failed (22 wk), Fusion	Quadriplegia
	II Mixed I/Lateral Mixed I/Lateral	Normal Hemiplegia Quadriparesis, CN6/7/12	Fusion + Collar Collar + Fusion Fusion + Collar	CN 10 Monoparesis CN 12
Yamaguchi 1996	I	Quadriplegia, CN 10,11,12	Brace failed (10wk), Fusion	Quadriplegia, CN 10,11,12
Geigui 1995	I	Normal	Fusion+Brace	Normal
Donahue 1994	I	Hemiparesis	Halo distracted (temp neuro worse), Fusion Halo + Fusion	Hyperreflexic
	II	CN6	Collar/Traction +	Normal
	II	Quadriplegia, CN7/10	Fusion	Quadriparesis, CN7/10
	II	Quadriparesis, CN3/7	Fusion	Quadriparesis
Palmer 1994	II	Quadriparesis, CN6	Traction (neuro worse), Brace+Fusion	Quadriparesis
Dickman 1993 Papadoulou 1991	II	Quadriplegia, CN9/10 Quadriparesis, CN6	Brace	Unchanged (sepsis death at three mo) Quadriparesis
	Rotatory	CN6	Traction (neuro worse), Fusion+Halo	
	Rotatory	Hemiparesis, CN3/6	None (neuro worse), Fusion+Halo	Hemiparesis
	Mixed II/Rotatory		Halo+Fusion	Normal
Harmanali 1993	II	Hemiparesis, CN12	Fusion+Brace	Normal
Hosono 1993	I	Hemiparesis	Brace(neuro worse), Fusion+Brace	Normal
Matava 1993	II	Hemiplegia, CN6/12	Fusion + Brace	Spastic, CN 6
	II	Hemiparesis, CN6	Fusion + Brace	Normal
	II	CN6/9/10	Fusion + Brace	Spastic
Nischal 1993	II	Quadriparesis, CN3,6,9,10	Brace+Fusion	Hemiparesis, CN3,6,9,10
	II	Quadriplegia, CN9,10	Brace+Fusion	Hemiparesis
Bundshuh 1992	I	Quadriparesis CN6,9,10,12	Traction+Fusion	CN6,12
Farley 1992	I	Quadriplegia, CN10	Traction+Brace	Quadriplegia
Belzberg 1991	II	Quadriparesis, CN6,9,10	Traction+Brace+ Fusion	Monoparesis, CN6
Lee 1991	II Mixed I/Rot	Normal CN6	Traction+Fusion Brace+Fusion	Normal CN6
Montane 1991	I	Hemiparesis	Fusion+Brace	Spastic
	II	Quadriparesis	Traction, Fusion+Brace	Normal
	II	Quadriplegia	Fusion+Brace	Quadriplegia
DiBenedetto 1990	I	Quadriparesis, CN9,10,12	Collar (neuro worse, 6wk), Fusion+Brace	Spastic

<b>AUTHOR</b>	<b>TYPE</b>	<b>INITIAL EXAM</b>	<b>TREATMENT</b>	<b>OUTCOME</b>
Colnet 1989	Mixed lat/rotatory	Hemiplegia, CN 6,9,10	None (neuro worse), Traction+Shunt+Decompression	Hemiparesis
Jevitch 1989	Lateral	Normal	Traction+Brace	Normal
Hummel 1988	I	Hemiparesis	Fusion+Brace	Normal
Zampella 1988	II	Quadriplegia, CN5-12	None	Quadriplegia, CN6
Georgopoulous 1987	I	Normal	None (neuro worse), Fusion+Brace	Normal
Bools 1986	III	Normal	Traction, Fusion+Brace	Normal
Collato 1986	Mixed I/lateral	Normal	None (neuro worse), Fusion+Brace	Monoparesis
Putnam 1986	I	Quadriplegia, CN 6	Brace	Death (sepsis eight mo)
Ramsay 1986	I	Quadriparesis	None (neuro worse), Traction+Brace	Hemiplegia
Roy-Camille 1986	I	CN6,11	None, Brace failed (3 mo), Traction+Fusion	CN6
	I	Quadriplegia, CN6,9-12	Traction+Fusion	Quadriplegia
Zigler 1986	I	Quadriplegia, CN11	Traction+Brace +Fusion	Quadriplegia
Watridge 1985	Lateral	Paraparesis	None (neuro worse), Traction+Fusion+Decompress+Brace	Normal
Banna 1983	Rotatory	Normal	Traction (2 wk)	Normal
Kaufman 1982	II	Quadriplegia	Brace+Fusion	Quadriparesis, CN9,10
	II	Monoparesis	Brace	Normal
Woodring 1981	I	Hemiparesis, CN6	Traction	CN6
	I	Monoparesis	None (neuro worse), Traction+Fusion	Quadriplegia
Powers 1979	I	Hemiparesis, CN6	Traction+Brace	Hemiparesis
	II	Hemiparesis, CN7	Traction+Brace	Normal
Rockswold 1979	II	Hemiparesis, CN6	Traction, Brace+Fusion	Ambulates
Eismont 1978	III	Normal	Collar(neuro worse) Fusion+Brace	Normal
Fruin 1977	I	Hemiparesis CN6,9-12	Traction+Fusion	CN6,11
Page 1973	I	Quadriplegia CN10,12	Traction, Brace failed (5mo),Fusion	Quadriparesis,CN10
Evarts 1970	I	Hemiparesis CN 6,9,10,12	Traction, Brace+Fusion	CN6
Gabrielsen 1966	I	Hyperreflexic,CN6	Traction, Brace failed (3mo),Fusion	Numb scalp
Farthing 1948	III	Normal	Traction+Brace	Normal

3 articles (15 patients) were eliminated because the type of dislocation was not reported. Cohen (1), Georgopoulous (2/3), Bulas (5/5), Naso (1/2), Sun (6/6)

2 articles (8 patients) were eliminated because the initial exam was not reported. Grabb (3), Ahuja (5)

2 articles (6 patients) were eliminated because the treatment was not reported.

Maves (3), Hladky (3)

1 article (2 patients) was eliminated because the outcome was not reported.

Jones (1), Bools (1/2)

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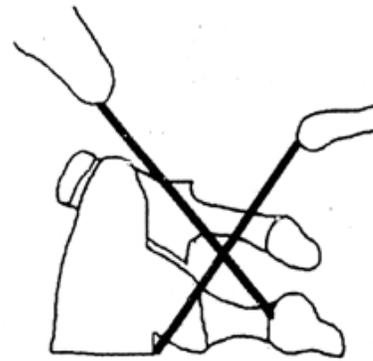
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**Figure 1**

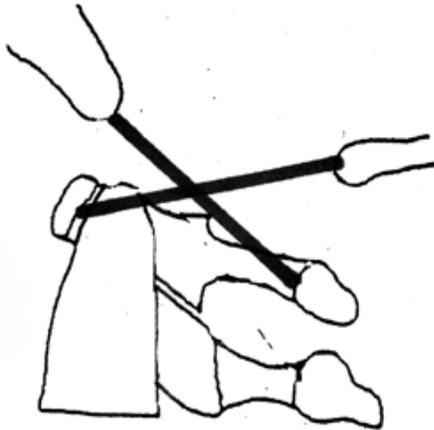
Mid-sagittal diagrams of the craniocervical junction show the various methods for identifying AOD on a lateral cervical radiograph including the Wholey measure (A), Power's ratio (B), Dublin measure (C), X-line method (D), and BAI-BDI method (E).



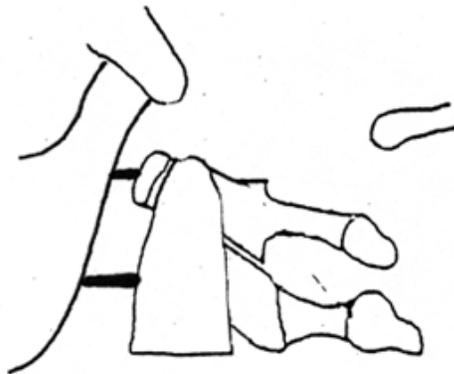
**Figure 1A**



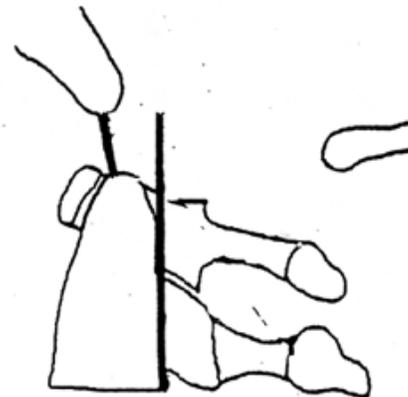
**Figure 1D**



**Figure 1B**



**Figure 1C**



**Figure 1E**

## OCCIPITAL CONDYLE FRACTURES

### RECOMMENDATIONS

#### **Diagnostic:**

Standards: There is insufficient evidence to support diagnostic standards.

Guidelines: CT imaging is recommended for establishing the diagnosis of occipital condyle fractures. Clinical suspicion should be raised by the presence of one or more of the following criteria: blunt trauma patients sustaining high energy craniocervical injuries, altered consciousness, occipital pain or tenderness, impaired cervical motion, lower cranial nerve paresis, or retropharyngeal soft tissue swelling.

Options: MR imaging is recommended to assess the integrity of the craniocervical ligaments.

#### **Treatment:**

Standards: There is insufficient evidence to support treatment standards.

Guidelines: There is insufficient evidence to support treatment guidelines.

Options: Treatment with external cervical immobilization is recommended.

### RATIONALE

Although traumatic occipital condyle fracture (OCF) was first described by Bell in 1817, more frequent observation of this injury has only been reported during the past two decades.(2) Improvements in computed tomographic (CT) imaging technology and use of CT imaging of the head-injured patient that includes the craniovertebral junction have resulted in more frequent

recognition of this injury. However, the overall infrequent occurrence of OCF and missed diagnoses in patients with OCF may result in late neurological deficits in these patients. An analysis of the reported cases of OCF may facilitate development of diagnostic and treatment recommendations for this disorder and are undertaken in this report. Specific questions that were evaluated include: accuracy of plain radiographs and CT imaging in the diagnosis of OCF, as well as the safety and efficacy of various treatment modalities including no treatment, traction, external immobilization, decompression and internal fixation with fusion.

## **SEARCH CRITERIA**

A National Library of Medicine computerized literature search of publications from 1966 to 2001 was performed using the following headings: occipital bone and fracture (spinal, skull, or fracture alone). An exploded search of the headings led to 1,830 and 33,537 citations, respectively. A subset of 218 citations contained both headings. The references of the identified articles were also reviewed to identify additional case reports. The articles were reviewed using the following criteria for inclusion in diagnosis: human survivors, type of fracture, tomographic or plain radiographic findings. The articles were also reviewed using the following criteria for inclusion in treatment: human survivors, type of fracture, management, and outcome. Since fewer than 100 cases of survivors were identified, even single case reports were considered, provided that basic inclusion criteria were met. The observations from the reports were combined because the usual methods for analysis were precluded by the infrequent occurrence of this injury. Forty-seven articles met the selection criteria, providing data on a sum total of 91 patients for this review. All but two articles contained Class III data of either single case studies

or small case series with none containing more than fifteen patients. The two exceptions were prospective studies to evaluate the use of clinical criteria in blunt trauma patients to prompt CT imaging of the skull base (4,26). The duration of follow-up in all articles ranged from several weeks to five years. The data provided by these reports was compiled and make up the basis for this guideline. Summaries are provided in Evidentiary Table format.

## **SCIENTIFIC FOUNDATION**

### **Diagnosis:**

Plain radiographs of the cervical spine were obtained in nearly all 91 patients culled from the literature review. Normal imaging was reported in 42 patients. Eight patients had prevertebral soft tissue swelling (STS), only four of whom did not have associated cervical fractures.(17,28,30,41) Ten patients with cervical fractures or displacements were described without mention of the presence or absence of STS. Three patients had multiple cervical fractures. Associated fractures included atlas-four, Type II odontoid-two, axis-three, C5 fractures-two, C3, C6, and C7 fractures- one each, and unspecified cervical fractures-one. One patient had atlantoaxial widening and one had C5-C6 subluxation. Only two patients were identified with OCF on plain radiographs of the skull or cervical spine.(21,44) The results of plain radiographs were not reported in 28 patients and plain radiographs were not performed in one patient with an old healed fracture identified with CT imaging.(12) The calculated sensitivity of plain radiographs from these reports in the diagnosis of OCF is 3.2% (two of 62). Since the data were obtained from case reports and small case series of patients known to have OCF, comparison with the findings of plain radiographs in patients without OCF could not be

performed. As a result, specificity, positive predictive value, and negative predictive value could not be determined.

The type of fracture was classified according to Anderson and Montesano (1) into Type I (comminuted from impact), Type II (extension of a linear basilar skull fracture), and Type III (avulsion of a fragment) fractures (Figure 1). The 91 patients in this review population provided data on 12 Type I, 24 Type II, 49 Type III unilateral fractures, four bilateral fractures (one Type I, two Type III, and one mixed Type I and Type III), and two old fractures.

All but one patient underwent tomographic imaging (polytomography alone-six, CT imaging alone-83, and both-one). One OCF was missed with polytomography and subsequently identified on CT.(33) Two patients had OCF diagnosed from retrospective review of CT images that were initially interpreted as normal. The diagnosis of OCF could be made in every patient with OCF. Bloom et al performed a prospective study over one year to identify the frequency of OCF in patients meeting certain clinical criteria.(4) Fifty-five consecutive patients with high energy blunt craniocervical trauma underwent thin-section craniocervical junction CT imaging. Supplemental criteria included reduced Glasgow Coma Score (GCS) on admission, occipitocervical tenderness, reduced craniocervical motion, lower cranial nerve abnormality, and retropharyngeal soft tissue swelling (STS). Nine of 55 patients (16.4%) were identified with OCF. Other reports have estimated a 1% to 3% frequency of OCF in patients sustaining blunt craniocervical trauma.(24,31) Similarly, Link et al reported the results of craniocervical CT on 202 patients with a Glasgow Coma Score between three and six.(26) OCF was identified in nine of 202 (4.4%).

Loss of consciousness was observed in 36 of 44 patients. Among 64 patients who had a sufficiently detailed neurological examination reported, 25 were normal, 24 had acute or delayed cranial nerve deficits alone, six had cranial nerve deficits with limb weakness, six had mild to severe limb weakness without cranial nerve deficits, one had a delayed onset of vertigo, one had hyperreflexia, and one had diplopia. Only four patients were found who did not have occipitocervical pain in the absence of significantly impaired consciousness.(28,32,41) One patient was intoxicated, one had severe extremity pain, and the other two had severe facial trauma.

Only 11 patients were investigated with MRI. Early craniocervical MR imaging was performed in eight patients, whereas late MR studies were obtained in three patients. Cervicomedullary hemorrhages were seen in three patients, two had normal imaging, one had a retrodental hemorrhage, one had a torn tectorial membrane, and one had demonstration of the fracture. Displaced fracture fragments were observed in all three patients with delayed MR imaging. Although acute MR imaging has been infrequently reported after OCF, Tuli et al proposed a new classification scheme using MR imaging to differentiate stable from unstable OCF.(41) However, the case example they gave demonstrated concurrent atlantoaxial instability which prompted occipitocervical fusion (rather than atlantooccipital instability and OCF fracture).

In summary, the diagnosis of OCF is rarely made on plain radiographs. Imaging of the craniovertebral junction with CT or other tomographic methods is recommended in patients suspected of having OCF. Blunt trauma patients sustaining high energy craniocervical injuries may be more likely to sustain OCF. Consequently, cranial imaging should include evaluation of

the craniocervical junction. Other clinical criteria including altered consciousness, occipital pain or tenderness, impaired craniocervical motion, lower cranial nerve paresis, or retropharyngeal STS should prompt CT imaging of the craniocervical junction.

### **Treatment:**

Twenty-three patients (Type I-two, Type II-fourteen, Type III-five, unknown type-two) did not receive treatment. Nine of these patients (Type I-one, Type II-four, Type III-four) developed cranial nerve deficits within days to weeks after injury.(6,9,12,13,31,33,34,42,45) One hypoglossal nerve palsy resolved, two hypoglossal nerve deficits improved, three other cranial nerve deficits persisted (two hypoglossal, one glossopharyngeal and one vagal), and three outcomes were not reported. Six additional patients developed delayed deficits or symptoms. Two initially untreated patients (Type II-one, Type III-one) developed multiple lower cranial nerve deficits which improved with six weeks of cervical immobilization.(23) Another initially untreated patient (Type III) developed vertigo after three months that resolved after eight weeks of collar immobilization.(7) One patient (Type III) developed nystagmus and a lateral rectus palsy after precautionary collar immobilization was discontinued. The deficit resolved after resuming cervical immobilization.(14) One patient (Type III) developed double vision during cervical traction which resolved with surgical decompression.(45) Finally, one patient (Type III) developed delayed vagal, spinal accessory and hypoglossal nerve palsies during cervical immobilization in a cervical collar.(8) The cranial nerve X and XI palsies improved. However, the hypoglossal palsy persisted at one year.

Forty-four patients were treated with cervical collar immobilization (Type I-eight, Type II-eight, Type III-twenty-eight). Thirteen patients were treated with halo/Minerva immobilization (Type I-two, Type III-eleven). Treatment was unreported in six patients.

Five patients (Type II-one, Type III-four) underwent surgery. Two patients with Type III OCF were treated with occipitocervical fusion (one with concurrent atlantooccipital dislocation and one with atlantoaxial instability).(21,41) One patient (Type III) with delayed diplopia had symptom resolution after removal of the fracture fragment (45), while one patient (Type II) with lower cranial nerve deficits (37) and one (Type III) with diplopia and hemiparesis (7) remained unchanged several days after surgery. The latter patient subsequently recovered normal function.

In summary, twelve of fifteen patients who developed delayed symptoms or deficits were not initially treated. Only three of these twelve patients were subsequently treated with cervical immobilization. All three improved. In comparison, only three of six patients demonstrated improvement in deficits without treatment. Only one patient (Type III) developed a deficit during treatment that persisted (hypoglossal nerve palsy) despite collar use. Only three patients underwent surgery for decompression of the brainstem, one of whom had immediate and lasting improvement in symptoms post-operatively. Because 12 of 23 patients developed delayed deficits without treatment and another developed a deficit after premature discontinuation of treatment, the literature suggests that patients with Type III OCF should be treated with external immobilization. Treatment of patients with OCF Types I and II may include external immobilization.

## **SUMMARY**

OCF is an uncommon injury requiring CT imaging for diagnosis. Patients sustaining high energy blunt craniocervical trauma, particularly in the setting of loss of consciousness, impaired consciousness, occipitocervical pain or motion impairment, and lower cranial nerve deficits, should undergo CT imaging of the craniocervical junction. Untreated patients with OCF often develop lower cranial nerve deficits that usually recover or improve with external immobilization. Identification of Type III OCF should prompt external immobilization. Additional treatment may be dictated by the presence of associated cervical fractures or instability.

## **KEY ISSUES FOR FUTURE INVESTIGATION**

Although Type III occipital condyle fractures are considered by many authors to be unstable, not all patients, treated or not, developed neurological deficits. CT imaging with three dimensional reconstruction for more precise measurement of the magnitude of fracture displacement and MR imaging for differentiation of partial and complete ligamentous injuries may be useful in identifying subgroups of patients who do not require treatment or conversely require more rigid halo immobilization, rather than collar immobilization. Because OCF injuries remain relatively infrequent, cooperative retrospective collection of plain radiograph, CT and MR imaging data in patients with OCF is recommended.

## EVIDENTIARY TABLES

AUTHOR	AGE	SEX	TYPE	LOC	PAIN	PLAIN	CT	MR	EXAM	TX	OUTCOME
Legros B et al, 2000, <i>J Trauma</i> (23)	71	F	III	-	Unrep	Unrep	L,+	Epidural	Del6,7,10	6wk collar	18mo 10
	44	M	II	-	Unrep	Unrep	R,+	Normal	Del6,9-12	6wk collar	3.5mo 10
Ide C et al, 1998, <i>J Neurosurg</i> (19)	25	M	III	+	+	STS, C1fx	R,+	Tectear	Normal	10wkcollar	10wk Normal
Demish S et al, 1998, <i>Clin Neurol Neurosurg</i> (13)	45	F	II	Unrep	Unrep	Unrep	R,+	Fracture	Del12	None	1yr imp12
Bloom AI et al, 1997, <i>Clin Radiol</i> (4)  Class II	21	M	III	Unrep	Unrep	STS,C67Fx	R,+	Unrep	Normal	>8wk collar	Normal
	36	F	III	Unrep	Unrep	Unrep	L,+	Unrep	Normal	>8wk collar	Pain
	15	F	I/I	Unrep	Unrep	Unrep	B,+	Unrep	Qparesis	>8wk collar	Imp Qparesis
	45	F	III/I	Unrep	Unrep	Unrep	B,+	Unrep	12	>8wk collar	Pain,12
	22	F	II	Unrep	Unrep	Unrep	R,+	Unrep	Normal	>8wk collar	Unrep
	21	M	I	Unrep	Unrep	STS,C125F	R,+	Unrep	Normal	>8wk collar	Unrep
	41	M	I	Unrep	Unrep	x	R,+	Unrep	Normal	>8wk collar	Normal
	6	F	II	Unrep	Unrep	Unrep	L,+	Unrep	Normal	>8wk collar	Normal
	25	F	I	Unrep	Unrep	Unrep	L,+	Unrep	Normal	>8wk collar	Unrep
20	M	I	Unrep	Unrep	STS,C2Fx CFx	R,+	Unrep	Pplegia	>8wk collar	Unrep	
Tuli S et al, 1997, <i>Neurosurgery</i> (41)	64	F	III	Unrep	+	STS	R,+	None	Normal	12wk collar	3mo Normal
	69	F	III	Unrep	-	AAWide	L,+	Fracture	Mparesis,7	OC Fusion	Improved
	27	M	Old	Unrep	-	Normal	L,+	None	Normal	None	3yr Normal
Cottalorda J et al, 1996, <i>J Pediatr Orthop</i> (10)	15	F	I	Unrep	+	Normal	R,+	None	Normal	7wkMinTrCo 1	4mo Normal
Lam CH and Stratford J, 1996, <i>Can J Neurol Sci</i> (22)	20	F	III	Unrep	Unrep	Normal	R,+	Contuse	Hpa,12	3moHalo	5yr imp 12
Urculo E et al, 1996, <i>J Neurosurg</i> (42)	62	M	III	Unrep	Unrep	Normal	R,+	Fracture	Del 9,10	None	6mo same

AUTHOR	AGE	SEX	TYPE	LOC	PAIN	PLAIN	CT	MR	EXAM	TX	OUTCOME
Noble ER and Smoker WRK, 1996, <i>Am J Neuroradiol</i> (31)	33	M	I	Unrep	Unrep	Unrep	?,+	None	Del 12	None	Unrep
	26	M	I	Unrep	Unrep	Unrep	?,+	None	GCS15	None	Unrep
	16	M	II	Unrep	Unrep	Unrep	?,+	None	GCS13	None	Unrep
	32	M	II	Unrep	Unrep	C2Fx	?,+	None	7,12	None	Unrep
	53	F	II	Unrep	Unrep	Unrep	?,+	None	GCS8	None	Unrep
	47	F	II	Unrep	Unrep	Unrep	?,+	None	GCS15	None	Unrep
	37	M	II	Unrep	Unrep	Unrep	?,+	None	GCS8	None	Unrep
	11	M	II	Unrep	Unrep	Unrep	?,+	None	GCS13	None	Unrep
	33	M	II	Unrep	Unrep	Unrep	?,+	None	GCS15	None	Unrep
	23	M	II	Unrep	Unrep	Unrep	?,+	None	Unrep	Unrep	Unrep
	39	M	III	Unrep	Unrep	IIOdFx	?,+	None	7	Halo	Unrep
	88	M	III	Unrep	Unrep	C1,IIOdFx	?,+	None	GCS15	Halo	Unrep
	29	M	III	Unrep	Unrep	Unrep	?,+	None	Unrep	Unrep	Unrep
14	F	III	Unrep	Unrep	Unrep	?,+	None	GCS11	Collar	Unrep	
17	F	III	Unrep	Unrep	Unrep	?,+	None	GCS7	None	Unrep	
Castling B and Hicks K, 1995, <i>Br J Oral Maxillofacial Surg</i> (9)	21	M	II	+	+	Normal	R,+	None	Del 12	None	2yr Normal
Emery E et al, 1995, <i>Eur Spine J</i> (15)	26	M	III	Unrep	+	Normal	L,+	Fracture	Hyperreflexic	Collar	4mo Normal
Paley MD and Wood GA, 1995, <i>Br J Oral Maxillofacial Surg</i> (34)	21	M	III	Unrep	+	Normal	L,+	Normal	Del 12	None	6mo imp 12
Stroobants J et al, 1994, <i>J Neurosurg</i> (40)	27	M	III	-	+	Normal	R,+	None	Normal	10wk collar	21moNormal
	12	F	III	-	+	C1Fx	L,+	None	Normal	4wk minerva	Normal
Wasserberg J and Bartlett RJV, 1994, <i>Neuroradiol</i> (45)	39	M	III	+	Unrep	Normal	L,+	None	Del 12	None	12
	24	M	III	+	+	Normal	L,+	None	Del Diplopia	Tx, Decomp	Normal
	16	M	III	+	Unrep	Normal	R,+	None	Brain injury	Tx, collar	3mo 12
	34	M	III	Unrep	Unrep	Normal	R,+	None	Unrep	Tx, halo	Unrep
Young WF et al, 1994, <i>Neurosurgery</i> (47)	26	F	III	+	Unrep	Normal	L,+	None	Hpa, 9-12	12wk halo collar	14mo imp9-12
	20	M	III	+	Unrep	Normal	R,+	None	GCS7		1yr Hpa
Mann FA and Coheen W, 1994, <i>Am J Radiol</i> (27)	23	M	III	-	+	Normal	R,+	None	Normal	6wk collar	Normal
Olsson R and Kunz R, 1994, <i>Acta Radiologica</i> (32)	43	M	III	Unrep	-	Normal	L,+	None	Normal	Collar	Normal

AUTHOR	AGE	SEX	TYPE	LOC	PAIN	PLAIN	CT	MR	EXAM	TX	OUTCOME
Sharma BS et al, 1993, <i>Clin Neurol and Neurosurg</i> (37)	35	M	II	Unrep	Unrep	Normal	L,+	None	9,10	Decomp	3mo imp 9,10
Massaro F and Lanotte M, 1993, <i>Injury</i> (29)	21	M	III	Unrep	Unrep	Normal	L,+	None	Hsensory, 12	8wk minerva	2yr 12
Raila FA et al, 1993, <i>Skeletal Radiol</i> (35)	25 67	M M	III III	+ -	+ +	Normal C1abnormal	L,+ L,+	None None	Normal Normal	6wk collar collar	Normal Normal
Bettini N et al, 1993, <i>Skeletal Radiol</i> (3)	39 24 21 21	F M F M	I II III III/III	Unrep + + Unrep	+ Unrep Unrep +	C3fx Normal Unrep Normal	L,+ R,+ ?,+ B,+	None None Contuse None	Normal Coma Coma Normal	Unrep Unrep Unrep Unrep	Unrep Unrep Unrep Unrep
Leventhal MR et al, 1993, <i>Orthopaedics</i> (25)	42 19 43 17 36 17	F F M F M M	II III III II I I	+ + Unrep + + +	Unrep + + Unrep GCS8 GCS4	Normal Normal C5fx L1fx T1fx Normal	L,+ L,+ R,+ R,+ R,+ R,+	None None None None None None	6,7 Normal Normal GCS10 GCS8 GCS4	3mo collar Collar 3mo collar 3mo collar 3mo halo 3mo collar	Unrep Unknown Normal Normal Normal Normal
Mody BS and Morris EW, 1992, <i>Injury</i> (30)	21	M	III	+	Unrep	STS	L,+	None	Unrep	Tx,6wk collar	18mo no sxs
Bozboga M et al, 1992, <i>Spine</i> (7)	34 37	F M	III III	+ +	+ Unrep	Normal Unrep	L,+ L,+	None None	Lhpa,diplopia Del vertigo	Late Decomp Del 8wk collar	4yr Normal 3yr Normal
Bridgman SA and McNab W, 1992, <i>Surg Neurol</i> (8)	32	M	III	+	+	Normal	L,+	None	Del 10-12	Collar	1yr imp 10-12
Wani MA et al, 1991, <i>J Trauma</i> (44)	67	M	II	+	Unrep	+ condfx	L,None	None	9-12	None	10,12
Wessels LS, 1990, <i>S Afr J Surg</i> (46)	26 7mo 27	M M M	III II II	+ + +	+ Unrep Unrep	Unrep Unrep Unrep	R,+ L,+ R,+	None None None	7-12 5,7-12 7-12	Collar Collar Collar	6wk imp 7-12 4mo 7-12 6wk imp
Mariani PJ, 1990, <i>Ann Emerg Med</i> (28)	30	M	III	+	-	STS	R,-	None	Normal	8wk collar	Normal
Jones DN et al, 1990, <i>Am J Neuroradiol</i> (21)	43	M	III/III	+	Unrep	+ confx	B,+	Contuse	Qplegia	OCF	4wk Qplegia
Desai SS et al, 1990, <i>J Trauma</i> (14)	33	M	III	-	+	Normal	L,+	None	6	Collar	4mo Normal

AUTHOR	AGE	SEX	TYPE	LOC	PAIN	PLAIN	CT	MR	EXAM	TX	OUTCOME
Valaskatzis EP and Hammer AJ, 1990, <i>S African Med J</i> (43)	19	M	III	+	+	Normal	R,+	None	Normal	6wk collar	Normal
Orbay T et al, 1989, <i>Surg Neurol</i> (33)	37	M	III	Unrep	+	Normal	L,+ (tomo-)	None	Del 12	None	15mo 12
Savolaine ER et al, 1989, <i>J Orthop Trauma</i> (36)	71	F	III	+	+	Normal	R,+	None	Hplegia, 6	Tr, Halo	Lmparesis
Anderson PA and Montessano PX, 1988, <i>Spine</i> (1)	3 18 22 23 25 37	M F M M M M	I III III III III II	+	Unrep Unrep Unrep Unrep Unrep Unrep	Normal Normal Normal Normal Normal Normal	R,+ ?,+ R,Tomo+ L,+ ?,Tomo+ L,+	None None None None None None	Uncon Unrep Uncon Uncon Unrep Uncon	Soft Minerva Halo Collar Minerva Collar	24mo normal 36 mo 12mo normal death 17mo 12mo normal
Curri D et al, 1988, <i>J Neurosurg Sci</i> (11)	16	F	III	+	Unrep	Normal	R,+	None	Decerebrate	Collar	6mo Unrep
Hashimoto T et al, 1988, <i>Neurosurgery</i> (18)	71	M	II	-	Unrep	Normal	L,+	None	9-12	None	6mo 9-12
Deeb ZI et al, 1988, <i>J Computed Tomography</i> (12)	25 66	F F	II Old	Unrep Unrep	Unrep +	Normal None	DelL,+ DelL,+	None Fracture	12 Normal	None None	Unrep Unrep
Spencer JA et al, 1984, <i>Neurosurgery</i> (38)	19	M	I	+	GCS8	Normal	L,+	None	GCS8	ColHalo	B910
Goldstein SJ et al, 1982, <i>Surg Neurol</i> (16)	24	F	III	Unrep	+	C56slx	L,Tomo+	None	Normal	2mocoll	NI
Harding-Smith J et al, 1981, <i>J Bone Joint Surg</i> (17)	18	M	III	+	Unrep	STS	R,Tomo+	None	Uncon	Collar	16moNI
Bolender N et al, 1978, <i>Am J Radiol</i> (6)	23 22	M M	III II	Unrep Unrep	Unrep Unrep	Normal Normal	R,Tomo+ R,Tomo+	None None	9-12 Del 6,9,10	None None	Unrep Unrep

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**FIGURE 1** Classification of occipital condyle fractures according to Anderson and Montesano. Type I fractures (A) may occur with axial loading. Type II fractures (B) are extensions of a basilar skull fracture. Type III fractures (C) may result from an avulsion of the condyle during rotation, lateral bending, or a combination of mechanisms.

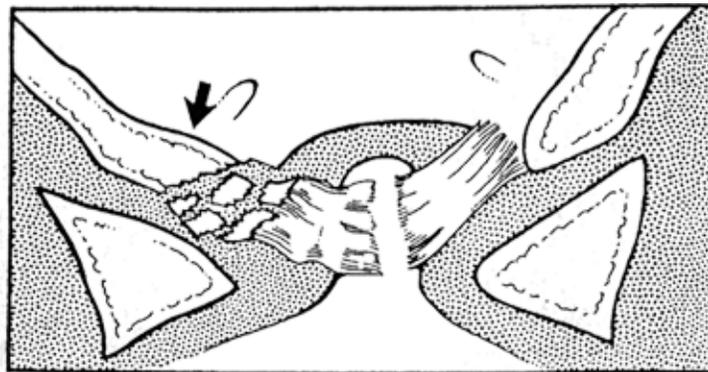


Figure 1A

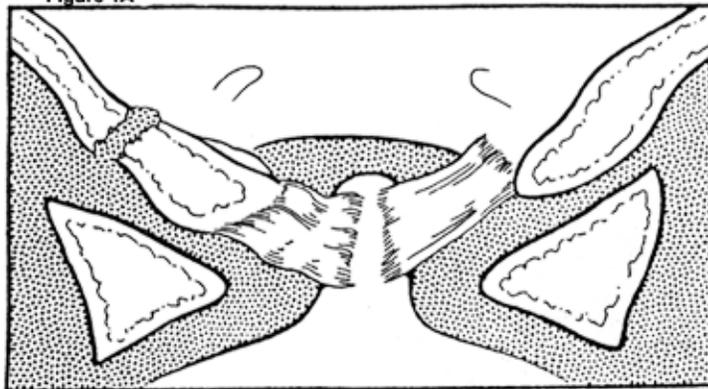


Figure 1B

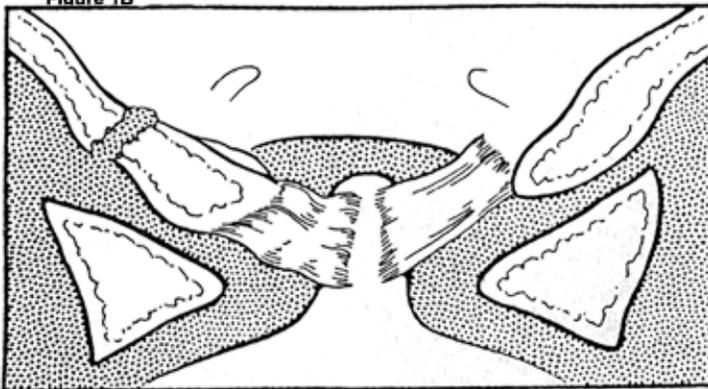


Figure 1C

## ISOLATED FRACTURES OF THE ATLAS IN ADULTS

### RECOMMENDATIONS

Standards: There is insufficient evidence to support treatment standards.

Guidelines: There is insufficient evidence to support treatment guidelines.

Options: Treatment options in the management of isolated fractures of the atlas are based on the specific atlas fracture type. It is recommended that isolated fractures of the atlas with an intact transverse atlantal ligament be treated with cervical immobilization alone. It is recommended that isolated fractures of the atlas with disruption of the transverse atlantal ligament be treated with either cervical immobilization alone or surgical fixation and fusion.

### RATIONALE

The atlas vertebra is subject to a variety of acute fracture injuries and may be associated with other cervical fracture and ligamentous traumatic injuries (3,8,25,26,31). While the treatment of atlas fractures in combination with other cervical fracture injuries is most commonly linked to the treatment of the associated injury (8), isolated fractures of the atlas occur with sufficient frequency to warrant review.

The medical literature addressing the management of fractures of the atlas was examined using evidence-based medicine techniques to determine the optimal treatment for isolated atlas fractures including: Isolated anterior or posterior arch fractures, anterior and posterior arch fractures (burst fractures), lateral mass fractures, comminuted fractures, and transverse process fractures.

## **SEARCH CRITERIA**

A National Library of Medicine computerized literature search from 1966 to 2001 was undertaken using Medical Subject Headings in combination with “vertebral fracture”: atlas and human. This strategy yielded 360 references. The abstracts were reviewed and articles addressing clinical management and follow-up of atlas fractures were selected for inclusion. The relative infrequency of these fractures, the small number of collected case series and the numerous case reports with pertinent information required rather broad inclusion and exclusion criteria. Several papers addressing relevant background information such as biomechanics and radiology were included. The bibliographies of the selected papers were also reviewed to provide additional references and to assess completeness of the literature review.

These efforts resulted in 32 manuscripts describing acute traumatic atlas fractures. Ten Class III articles, eight case series and two case reports documenting treatment of patients with atlas fractures are summarized in Evidentiary Table format. The remaining referenced 22 articles are included in the bibliography and contribute to the scientific foundation.

## **SCIENTIFIC FOUNDATION**

Atlas fractures account for approximately 1% to two % of all fractures of the human spinal column and roughly 2% to 13 % of all acute cervical spine fractures.(8,21,30)

The first known fracture of the atlas was demonstrated at autopsy by Cooper in 1822 and has been the subject of a series of historical publications.(28) In 1920, Jefferson reviewed 42 previously described cases of atlas fracture and added four new cases.(15) While his paper documents a variety of atlas fracture patterns, it is best known for the characterization of the “Jefferson fracture”, a burst fracture injury of the atlas ring.(10) In 1945 Hinchey and Bickel

added 112 cases of atlas fracture to the literature. Sherk and Nicholson summarized an additional 73 cases in 1970.(30)

Spence et al, in 1970 reported their findings of a study of the mechanism of atlas fracture and potential rupture of the transverse atlantal ligament.(31) Using ten cadaveric specimens, the authors studied the application of force required to fracture C1 and to rupture the transverse ligament (range 38 - 104 kg, mean 58 kg). The sum of the excursion of the C1 lateral masses over the C2 lateral masses following traumatic injury ranged from 4.8 to 7.6 mm (mean 6.3 mm). The authors concluded that if the sum of lateral mass displacement (LMD) of C1 over C2 on the anterior-posterior radiographic image is greater than 6.9 mm, then the transverse atlantal ligament is "probably torn". In a follow-up clinical and biomechanical study, Fielding et al confirmed these findings.(5) These two studies, completed in the pre-MRI era, are the basis for the widely quoted "rules of Spence" (i.e., > 6.9 mm LMD = transverse atlantal ligament disruption) offered to assist in the management of patients with isolated atlas fractures. Subsequently, Heller et al reported their observations on 35 open mouth odontoid films using calibration markings to assess radiographic magnification.(12) They found an 18% magnification factor on open mouth odontoid view x-rays. Applying this information to the evaluation of atlas burst fractures by means of the "rules of Spence" suggests that the sum of the lateral mass displacement measurement indicating atlantal transverse ligament disruption should be increased from 6.9 mm to 8.1 mm. This study pointed out the difficulty in using plain radiographic measurements to assess the integrity of the transverse atlantal ligament following acute traumatic atlas fracture.

Hadley et al reported a series of 57 patients with atlas fractures representing 6.6 % of their series of cervical fractures in 1988. They managed 32 patients with isolated atlas fractures, none of whom sustained neurological injury.(8) The authors based their treatment recommendations on the degree of LMD in each patient. Twelve patients had non-displaced atlas fractures. Of these, ten

were treated with a rigid collar, one with a soft collar and one with a suboccipital-mandibular immobilizer (SOMI). The duration of treatment for these patients was 8-12 weeks. Fifteen patients had LMD of less than seven mm. Eight were treated with a rigid collar, three with a SOMI and four with a halo immobilization brace for a duration of 10-12 weeks. The remaining five patients had LMD greater than seven mm and were managed with a halo orthosis. These five patients were treated for a duration of 12-16 weeks. Of the 32 patients, 29 were available for long-term follow-up, median 40 months. Three complained of neck pain. All were successfully treated. No patient required subsequent surgical fixation. The authors concluded that isolated fractures of the atlas are effectively managed with external immobilization alone for a period of 12 weeks (median duration). Atlas fractures with a LMD greater than 6.9 mm required more rigid immobilization (halo orthosis), than those with LMD less than 6.9 mm (cervical collar). Levine and Edward described their experience with 34 patients with isolated atlas fractures in 1991. They followed a similar treatment algorithm with similar success.(21)

Fowler et al reported a series of 48 consecutive atlas fracture patients representing 5.5% of all cervical fractures in their experience.(6) In their series, 33% of their patients had other associated cervical spine fractures. Atlas fractures were divided into burst (n = 30), posterior arch (n = 17) and anterior arch fractures (n = 1). None of the patients with an isolated atlas fracture presented with neurologic deficit. These authors suggested treatment with closed traction reduction of isolated atlas fractures if the LMD is greater than 7.0, followed by immobilization in a rigid collar. No patient in this series underwent surgical fixation. All were effectively treated with this management scheme at last follow up, although the duration of treatment was not specified. In 1991 Kesterson et al reported their series of 17 cases of atlas fractures.(16) Thirteen were isolated atlas fractures and were considered stable. All were successfully managed with rigid cervical immobilization (nine collar, one SOMI, one halo, two Minerva). Again, the duration of treatment

was not specified. Several other authors have described the successful treatment of isolated atlas fractures with rigid cervical immobilization employing similar management principles (9,13,20,29,32).

Landells and Van Peteghem described a series of thirty-five patients with atlas fractures comprising 4.7 % of their institutional experience with acute cervical fracture injuries.(18) The authors categorized atlas fractures into three types. Type I fractures involved a single arch and occurred in sixteen of their thirty-five patients. Type II fractures were burst fractures and represented 13 of the 35 isolated fractures they treated. Type III fractures were atlas lateral mass fractures identified in six of the 35 patients. The authors used the original "rules of Spence" to assist with the identification of stability and noted four patients with LMD greater than 6.9 mm. Regardless of the fracture type or stability, all fractures except one were initially treated with external immobilization for an unreported length of time. The one exception was a patient with a Type I fracture who underwent early surgery with C1-2 wiring and fusion. The reason for the exception is not made clear in the text. Only one of 34 patients treated with external immobilization required surgery for late instability. The authors observed no relationship between successful treatment and the amount of initial lateral mass displacement. They recommended that atlas fractures be initially treated with rigid external immobilization. They noted that late instability can occur and recommended clinical follow-up of these patients.

Clinically observed atlas fracture patterns can be reproduced in cadaveric experimental models of axial loading.(11) In a series of biomechanical studies, Panjabi and Oda reproduced these atlas fracture patterns with axial loading and found that the burst fracture was associated with post-injury hypermobility at C1-C2. They described a 42 % increase in flexion-extension motion, a 24 % increase in lateral bending and a five % increase in axial rotation.(24-26) They found that in all instances of transverse atlantal ligament disruption, the atlanto-dental interval was greater than

three mm. The authors concluded that the atlanto-dental interval was the most reliable predictor of transverse ligament disruption in adults following acute C1 fracture.

McGuire and Harkey in 1995 described two cases of unstable atlas burst fractures treated with surgical fixation and fusion.(22) The fractures were felt to be unstable based on a predental space greater than five mm, and lateral mass displacement greater than nine mm, respectively. Both were treated with posterior C1-2 transarticular screw fixation and fusion with good result. The authors reported that transarticular screw fixation obviated the need for halo immobilization post-operatively. Several other authors have reported successful surgical fixation and fusion for atlas fractures when associated with disruption of the transverse ligament with resultant instability.(17,18,27) These few patients were treated with posterior C1-C2 wiring and fusion procedures and were managed in rigid orthoses (halo, Guilford brace) for 12-16 weeks post-operatively.

More recently it has been proposed that magnetic resonance imaging is a more sensitive indicator of transverse atlantal ligament disruption than the "rules of Spence".(3,4,7) Dickman et al described two types of isolated transverse atlantal ligament injuries they identified on MR imaging: Type I, in which the substance of the ligament is injured without associated fracture of the atlas, and Type II, which involves an avulsion fracture of the atlas at the insertion of the transverse atlantal ligament.(3) The authors concluded that patients with Type I injuries be treated with early surgical fixation due to the inherent instability at C1-C2 following ligamentous disruption. They favor rigid external immobilization for patients with Type II ligament fracture injures. Applying MR imaging to their series of 39 patients with atlas and/or axis fractures, the authors reported that the use of standard cervical radiographs and the "rules of Spence" would have failed to identify 60% of the fractures they found with associated disruption of the transverse atlantal ligament (as determined by MR).(4)

More recently, Lee, Green and Petrin described 16 patients with atlas fractures.(20) These included six isolated anterior or posterior arch fractures (Landell's Type I), four burst fractures (Landell's Type II) and six lateral mass fractures (Landell's Type III). Twelve of the 16 fracture injuries were isolated atlas fractures and were judged to be stable as determined by integrity of the transverse ligament either by MR imaging or by lateral mass displacement criteria. All 12 were successfully treated with rigid collar immobilization for 10-12 weeks. The authors recommended a treatment algorithm of cervical immobilization for stable atlas fractures, and surgical fixation and fusion for unstable atlas fractures and unstable C1-C2 combination fracture injuries. Their series, however, did not include any patient with an unstable isolated atlas fracture, nor any patient with an isolated atlas fracture who required surgical management.

Unusual isolated atlas fractures have been described in the literature, often as radiographic curiosities.(1,11,14,19,23) In none of the cases in which clinical information was provided, was surgical treatment necessary. Fractures of the transverse process of the atlas have been described, including one of the cases described by Jefferson in 1920.(2,15) Although injury to the vertebral artery has been associated with fractures through the C1 transverse foramen, the bony C1 injury has not required surgical fixation and has been treated with immobilization alone.

## **SUMMARY**

There are no Class I or Class II studies which address the management of patients with isolated atlas fractures. All of the articles reviewed described case series or case reports providing Class III evidence supporting several treatment strategies for patients with acute C1 fracture injuries.

Isolated anterior or posterior atlas arch fractures and fractures of the atlas lateral mass have been effectively treated with external cervical immobilization devices. Rigid collars, SOMI braces

and halo ring-vest orthosis have all been utilized for a duration of treatment of 8-12 weeks with good result. No study has provided evidence for using one of these devices over the other.

Combined anterior and posterior arch fractures of the atlas (burst fractures) with an intact transverse atlantal ligament (implying C1-C2 stability) have been effectively managed with use of a rigid collar, a SOMI brace, or a halo orthosis for a duration of 10-12 weeks.

Combined anterior and posterior arch fractures of the atlas (burst fractures) with evidence of transverse atlantal ligament disruption have been effectively treated with either rigid immobilization alone (halo orthosis) for a period of 12 weeks, or surgical stabilization and fusion. The type of C1-C2 internal fixation and fusion procedure performed may influence the need for and duration of post-operative immobilization.

Criteria proposed to determine transverse atlantal ligament injury with associated C1-C2 instability include: Sum of the displacement of the lateral masses of C1 on C2 of greater than 8.1 mm on plain films (“rules of Spence” corrected for magnification), a predental space of greater than 3.0 mm in adults, and magnetic resonance imaging evidence of ligamentous disruption or avulsion.

**Table:**

<b>Atlas Fracture Type</b>	<b>Treatment Options</b>
Anterior or posterior arch fractures	Collar
Anterior and posterior arch (burst) <ul style="list-style-type: none"> <li>- Stable (transverse atlantal ligament intact)</li> <li>- Unstable (transverse atlantal ligament disrupted)</li> </ul>	Collar, Halo Halo, C1-2 Stabilization and fusion
Lateral mass fractures <ul style="list-style-type: none"> <li>- Comminuted fracture</li> <li>- Transverse process fractures</li> </ul>	Collar, Halo Collar

## **KEY ISSUES FOR FUTURE INVESTIGATION**

The ability to identify the atlas fracture types at greatest risk of non-union and subsequent instability is a key issue in determining appropriate management. Prospective data collection generating case-control studies at multiple institutions would be feasible and useful in examining this issue. The relative infrequency of isolated atlas fractures would make a randomized study less likely to be implemented. A uniform and clinically useful definition of instability in association with isolated atlas fractures would be of benefit. The subgroup of patients with isolated atlas fractures with transverse ligament disruption that can be managed either by external immobilization alone or surgical fixation and fusion should be examined in terms of long-term success, economic benefit, patient satisfaction and return to pre-injury activities. The relatively few patients with atlas fractures treated with surgical stabilization and fusion described in the literature limits the ability to address these issues at present.

## EVIDENTIARY TABLE

First Author Reference	Description of Study	Data Class	Conclusions
Lee TT et al, 1998, <i>Spine</i>	Retrospective review including 12 cases of isolated atlas fracture	Class III	All treated successfully with external immobilization. Treatment algorithm proposed: Stable - treat with collar 12 weeks Unstable- proposes surgical fixation (Instability defined as lateral mass displacement greater than seven mm or MRI evidence of transverse ligament disruption)
McGuire RA and Harkey HL, 1995, <i>J Spinal Disord</i>	Two cases of unstable atlas burst fracture treated with posterior transarticular screw fixation and fusion	Class III	Considered unstable based on a predental space greater than five mm and a LMD greater than nine mm. Both treated successfully. A cervical collar was used post-operatively.
Levine AM and Edwards CC, 1991, <i>J Bone Joint Surg (Am)</i>	Retrospective review of 34 patients with atlas fractures Median followup 4.5 years	Class III	If lateral mass displacement less than seven mm (collar) and with greater than seven mm either halo alone or reduced in traction and maintained until healed (6 weeks in traction and six weeks in halo) No patients treated surgically
Kesterson L et al, 1991, <i>J Neurosurg</i>	Retrospective review including 13 patients with isolated atlas burst (Jefferson) fracture	Class III	All successfully treated with immobilization.
Fowler JL et al, 1990, <i>J Spinal Disord</i>	Retrospective review of 48 consecutive atlas fractures divided into burst (30), posterior arch (17) and anterior arch fractures (1)	Class III	Authors suggest reduction by traction if LMD greater than 7.0, followed by collar. No patients underwent surgical fixation.
Hadley MN et al, 1988, <i>Neurosurgery</i>	Retrospective review including 32 isolated fractures of the atlas. Median follow-up 40 months on 29 of 32.	Class III	The following treatment patterns were recognized: LMD greater than seven mm (5 patients) treated with Halo, LMD 0 to seven mm (15 patients) - four treated in halo, 11 treated in SOMI, LMD none (12 patients) treated in rigid collar. None of these isolated C1 fractures sustained neurologic injury or required surgery. 3 complained of neck pain otherwise all were successfully treated. Authors recommendation: Isolated C1 fractures can be managed without early surgical fixation. If the LMD is greater than 6.9 mm then Halo immobilization is indicated.

First Author Reference	Description of Study	Data Class	Conclusions
Landells CD and VanPeteham PK, 1988, <i>Spine</i>	Retrospective review of 35 patients with fracture of the atlas.	Class III	The authors outline a classification scheme: Type I - single arch (16)- most prevalent and most often associated with other fractures Type II - burst fracture (13) - most often in isolation only one of 13 with deficit, Type III - lateral mass fracture (6) Treatment not standard but 34 patients were treated with rigid external immobilization. Only one patient treated with early surgery (Type I fracture treated with C1-2 fusion). One patient required surgery in followup. 56 % of patients reported significant symptoms at one year (neck pain, scalp dysesthesias). Authors argue for conservative measures with traction and immobilization with careful follow-up.
Segal LS et al , 1987, <i>J Bone Joint Surg (Am)</i>	Retrospective review including eight isolated atlas fracture. Median follow-up 46 months.	Class III	Two of four patients with a comminuted fracture, described as a unilateral avulsion of the transverse ligament attachment and adjacent arch fracture, developed a non-union and remained symptomatic at follow-up. The authors recommend these patients be considered for the "most effective immobilization". None of the patients underwent surgical fixation.
Kornberg M , 1986, <i>Orthop Rev</i>	Report of single case of unstable atlas burst fracture.	Class III	Author feels fusion appropriate for unstable burst fractures of the atlas (LMD - greater than 6.9 mm) and describes a case of posterior arch disruption in which they were still able to perform C1-2 posterior fusion as one arch remained connected to lateral mass.
Schlicke LH and Callahan RA, 1981, <i>Clin Orthop</i>	Report of a single case of unstable atlas burst fracture.	Class III	Case of unstable burst fracture of the atlas with LMD of 12 mm. The authors propose a treatment algorithm of considering surgery for disruption of the transverse ligament (LMD greater than 6.9 mm).

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## ISOLATED FRACTURES OF THE AXIS IN ADULTS

### RECOMMENDATIONS:

#### Fractures of the Odontoid

Standards: There is insufficient evidence to support treatment standards.

Guidelines: Type II Odontoid fractures in patients 50 years of age and older should be considered for surgical stabilization and fusion.

Options: Type I, Type II and Type III fractures may be managed initially with external cervical immobilization. Type II and Type III odontoid fractures should be considered for surgical fixation in cases of dens displacement five mm or greater, comminution of the odontoid fracture (Type IIA) and/or inability to achieve or maintain fracture alignment with external immobilization.

#### Traumatic Spondylolisthesis of the Axis (Hangman's fracture)

Standards: There is insufficient evidence to support treatment standards.

Guidelines: There is insufficient evidence to support treatment guidelines.

Options: Traumatic spondylolisthesis of the axis may be managed initially with external immobilization in the majority of cases. Surgical stabilization should be considered in cases of severe angulation of C2 on C3, (Francis Grade II and IV, Effendi Type II), disruption of the C2-3 disc space, (Francis Grade V, Effendi Type III) or inability to establish or maintain alignment with external immobilization.

## **Fractures of the Axis Body (Miscellaneous Fractures)**

Standards: There is insufficient evidence to support treatment standards.

Guidelines: There is insufficient evidence to support treatment standards.

Options: External immobilization is recommended for treatment of isolated fractures of the axis body.

## **RATIONALE**

Fractures of the axis represent unique cervical vertebral injuries due to the unique anatomy and biomechanics of the C2 vertebra and the stresses applied to the dynamic atlanto-axial complex during trauma. Fractures of the axis may be associated with other cervical fractures or ligamentous injuries. Isolated fractures of the axis are common and warrant independent consideration. Fractures of the axis have been divided into three general subtypes: Fractures of the odontoid process, traumatic spondylolisthesis of the axis (Hangman's fractures), and miscellaneous non-odontoid non-Hangman's fractures of the C2 vertebra. Each of these fracture subtypes has been further subdivided based on the anatomic features and the functional significance of the individual fracture injury. The purpose of this review is to identify evidence-based management strategies for each injury subtype of traumatic fractures of the second cervical vertebra.

## **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": "axis", "vertebrae", "fracture" and "human". Seven hundred and eleven articles were identified. Those

manuscripts focusing on the clinical management of acute traumatic axis fractures were selected for review. The bibliographies of these papers were scanned for additional references to confirm completeness of the literature review. Relevant papers addressing the mechanism of injury or the biomechanics and radiology of the C2 vertebra were included.

The articles were reviewed and classified using established methodology. Thirty-eight articles for odontoid fracture, 17 for traumatic spondylolisthesis and eight for miscellaneous axis fractures comprised the basis for the scientific foundation of this guideline. Data from articles describing axis fractures and/or their management were categorized and are provided in Evidentiary Table format. Fifteen additional articles are referenced for supporting information.

## **SCIENTIFIC FOUNDATION**

### **Odontoid Fractures:**

#### *Overview*

The most common traumatic axis injury is fracture through the odontoid process, either through the tip of the dens (Type I), through its base (Type II) or involving the odontoid but extending into the C2 body (Type III). (1,6,41)

The anatomy and biomechanics of the C1-C2 complex provide for weight bearing support for the head on the spine and for the most motion of any intervertebral unit in the cervical spine. Motion at C1-C2 is primarily rotational, accounting for one half of the axial rotation of the head on the neck.(76) Translational motion of C1 on C2 is restricted by the transverse atlantal ligament which approximates and secures the odontoid process to the anterior arch of the ring of C1. With a fracture of the odontoid process, restriction of translational movement of C1 on C2 may be lost. Anterolisthesis or retrolisthesis of the C1-odontoid complex may occur relative to

the body of C2. If substantial subluxation of C1 on C2 occurs, spinal cord injury may result. The atlanto-axial complex is one of the most common sites of dislocation in fatal cervical spinal injuries.(21)

Prior publications utilizing evidence-based methodology for evaluating the literature on odontoid fracture management have focused on fusion as the primary outcome criterion with a minimum follow-up of 18 months.(45,72) Articles on odontoid fractures containing this information were included. While it has been argued that the radiographic determination of fusion may be difficult and subject to observer variability, it appears to be the most appropriate outcome measure and is described in the majority of clinical articles addressing odontoid fractures. It is recognized that outcome measures incorporating patient satisfaction, quality of life measures, and function would perhaps be superior, however, this information is sparse and less objective than the fusion criteria described in the literature.

#### *Classification of Odontoid Fractures:*

In 1974, Anderson and D'Alonzo classified fractures of the odontoid into three types.(1) This categorization has met with general acceptance and remains in use with minor modification. Based on a series of 49 patients managed from 1954 through 1972 with an average follow-up of 22 months, the authors defined three odontoid fracture types: Type I fractures are oblique fractures through the upper portion of the odontoid process. Type II fractures cross the base of the odontoid process at the junction with the axis body. Type III fractures are fractures through the odontoid that extend into the C2 body. The authors considered the Type III odontoid fracture to be more accurately described as a fracture of the body of the axis. Using this scheme the authors identified and treated two Type I fractures, 32 Type II fractures and 15 Type III fractures.

In 1988 Hadley et al added another fracture subtype to this classification scheme.(40) They described the Type IIA odontoid fracture as a comminuted fracture involving the base of the dens with associated free fracture fragments. The incidence of a Type IIA fracture was estimated at 5% of all Type II fractures, (three of 62 Type II fractures in their series) and was associated with severe instability and inability to obtain and maintain fracture reduction and realignment. The authors proposed that Type IIA odontoid fractures be managed with early posterior surgical fixation and fusion of C1-C2.

### *Treatment*

A variety of treatment strategies have been proposed for odontoid fractures based on the fracture type, the degree of initial dens displacement, the extent of angulation of the dens with respect to the body of C2, and the age of the patient. These include non-operative and operative methods.(1,15,16,31,35,39-41,45,51,58,72) Patients with odontoid fracture injuries have been treated with external immobilization using a variety of orthoses with varying results.(1,15,16,31,35,39-41,45,51,58,72) Surgical options include posterior cervical fusion with or without transarticular screw fixation or anterior odontoid screw fixation techniques.

### *Non-operative Treatment*

No treatment: In 1985 the Cervical Spine Research Society published a multicenter review addressing the management of odontoid fractures. This report includes 18 patients with type II odontoid fractures and three patients with type III odontoid fractures who received no treatment.(16) None of these cases achieved subsequent bony fusion. The authors concluded that non-treatment of odontoid fractures should be eliminated as a management choice.

Traction: Reviews by Traynelis and Julien et al include evidentiary tables describing seven articles containing Class III medical evidence addressing the treatment of odontoid fractures with traction and subsequent immobilization in a cervical collar.(1,15,16,31,35,45,51,58,72) All patients with Type I odontoid fractures achieved radiographic fusion (three of three). Eighty-seven percent of patients with Type III fractures achieved fusion (55 of 63 patients). The failure rate for patients with Type II fractures treated in this fashion was 43% (42 of 97). Fifty-seven percent achieved bony union. It appears that traction followed by cervical collar immobilization may be considered a management option for patients with odontoid fractures, particularly those with Type I and Type III fractures. The low fusion success rate reported for Type II odontoid fractures managed with traction and collar immobilization (57%) implies that perhaps collar immobilization is not the ideal strategy for Type II fracture patients.

Cervical collar: Several authors have proposed treatment of odontoid fractures with cervical collars. Polin et al in 1996 describes a series of 36 Type II fractures treated with either a Philadelphia collar or halo vest immobilization.(24) The fusion rate was lower in the patients treated with collars (53%) compared to 74% for patients managed in halos. An earlier report from the same institution described a similar rate of fusion (57%) in a study including seven Type II fractures treated with a collar alone. (75)

The infrequent Type I odontoid fracture appears to have an acceptable rate of fusion with rigid cervical collar immobilization, approaching 100% in one study.(1,15,16) Type III odontoid fractures have been treated with cervical collars as well but have a less favorable union rate with fusion rates ranging from 50% to 65% in small series. (16,75)

Halo Immobilization: In a series of publications resulting in the largest institutional series of axis fractures published to date, 340 cases of axis fractures were reviewed including 199

odontoid fractures (Type I - two, Type II – 116, Type IIA - four, Type III - 77. (37,39,41) Excellent results were obtained with rigid external immobilization in the Type I and Type III fracture patients (two of two and 68 of 69 with successful fusion). Of the Type II patients, 95 were treated with external immobilization for a median of 13 weeks. The authors reported a 28% failure rate. Seven failures were successfully treated with additional external immobilization and 18 patients underwent subsequent posterior C1-C2 fusion. The authors found that a displacement of the dens six millimeters or greater was associated with a high non-union rate, irrespective of patient age, direction of displacement, or neurological deficit (86% failure rate, chi square 33.74,  $p < .001$ ). The degree of dens displacement and a negative correlation with fusion was noted by at least four other investigators.(16,25,27,49) The amount of odontoid displacement observed ranged from two to six millimeters in their studies.

Julien et al reviewed nine articles that dealt with treatment of odontoid fractures (total 269 patients) using halo/Minerva fixation for 8-12 weeks.(12,15,16,25,27,31,45,49,58,63) All patients with Type I odontoid fractures were found to have successful fusion (three of three). (15,31,63) One hundred and sixty-eight patients with Type II odontoid fractures were treated with halo immobilization. One hundred ten had successful fusion (65%). There was a 30% non-union rate (50 of 168 patients). Eight patients were described as having a malunion. Eighty-four percent of patients with Type III odontoid fractures (67 of 80) achieved a solid fusion. There was an 8% failure rate (6 of 80 patients) and seven cases were described as malunions. The authors of these series generally concluded that rigid external immobilization can be considered a viable treatment option for Type I, Type II, and Type III odontoid fractures. Rigid external immobilization appears to be most successful for patients with Type I, Type III and nondisplaced Type II odontoid fractures but should be considered with caution in elderly patients.

### *Operative Treatment*

Posterior Cervical Fixation: Posterior cervical fixation and fusion has been successfully utilized in the treatment of acute traumatic odontoid fractures. Although no criteria defining the indications for surgical fixation have been established, a number of retrospective case series suggest treatment options. (15,16,18,25,31,50,58,74) These papers describe a total of 147 patients who underwent posterior cervical fixation and fusion for Type II odontoid fractures and 29 patients treated similarly for Type III fractures. One patient with a Type I fracture was treated successfully with posterior fusion. The overall fusion rates for Type II and Type III fractures managed with surgical fixation and fusion were 87% and 100% respectively in these series. The report of Maiman and Larson described a fusion rate across the fracture line of only 35%, but a fusion rate of 100% at the posterior operative site. (50)

The aforementioned series typically describe an instrumented (wire or cable) posterior C1-2 arthrodesis followed by cervical immobilization in a rigid orthosis. More recently, transarticular screw fixation and fusion of C1-2 has been employed for traumatic odontoid fractures, particularly in cases of failed fusion following initial management (14,44). The reported surgical morbidity and mortality is 2% to 4%, and includes failure of fracture reduction, vertebral artery injury, and the new onset of neurological deficit. Loss of motion at the atlantoaxial joint following posterior C1-2 fusion results from of dorsal C1-C2 arthrodesis. Despite this, several authors favor posterior C1-2 fusion rather than anterior odontoid screw fixation as ideal treatment of unstable odontoid fractures. (2,14,57)

Anterior Cervical Fixation: Anterior single and double screw fixation of odontoid fractures has been accomplished with success. The technical challenges associated with this procedure have limited widespread application. If successful, this technique has the potential to maintain rotational motion at the atlanto-axial joint. It has been suggested that this is an appropriate strategy when the odontoid fracture line is either horizontal or oblique and posterior, and that it is contraindicated in situations where the fracture line is oblique and anterior.(2,3,20,56) In cases of transverse atlantal ligament disruption, anterior screw fixation can result in an unsatisfactory outcome despite union of the odontoid fracture due to persistent transverse atlantal ligament incompetence. Julien et al summarized a series of articles that describe retrospective experiences with anterior screw fixation for odontoid fractures.(9,15,22,31,44,45) The combined fusion rate of Type II fractures treated in these reports is 89% (112 of 126 patients). Patients with type III odontoid fractures achieved radiographic fusion in 20 of 20 patients (100%). In a recent series reported by Subach et al, 26 patients with Type II fractures (mean age 35) underwent anterior odontoid fixation with single screw followed by immobilization in a cervical collar (median 7.2 weeks). (69) Twenty-five of 26 patients achieved successful fusion (96%). The one failure was attributed to inadequate fracture reduction. That patient required subsequent posterior C1-C2 fusion. Jenkins, Corric and Branch in 1998 described a retrospective non-randomized series of 42 patients undergoing anterior screw fixation for Type II odontoid fractures. They compared single-screw versus two-screw techniques.(44) The fusion rate in their experience was similar for single-screw fixation (81%) compared to two-screw fixation (85%). Use of lag screws to achieve anterior odontoid fixation is recommended. Complications of the procedure include retropharyngeal wall injury, screw fracture, infection, and screw misplacement

with injury to surrounding vascular and neural structures.(9,22,31) Attempts at anterior odontoid fixation utilizing a transoral approach was associated with multiple significant complications.(15)

Apfelbaum et al compared anterior screw fixation for recent and remote odontoid fractures at two institutions.(3) One hundred and forty seven patients with Type II (n = 138) and Type III (n = nine) odontoid fractures underwent anterior screw fixation either within six months of injury (129 patients) or greater than 18 months post injury (18 patients). The fusion rates were 88% in the less than six months group versus 25% in the remote fracture injury group (p <0.05), with a mean follow-up of 18 months. A positive correlation was identified between fusion and fractures oriented in the horizontal or posterior oblique planes. No effect of age, sex, number of screws placed or degree of dens displacement was identified. Their experience suggests that anterior odontoid screw fixation for odontoid fractures is most effective when performed early after injury, particularly within six months of fracture.

#### *Odontoid Fracture Management in the Elderly Patient*

One of the controversial issues in the management of odontoid fractures is the influence of age on treatment selection. A number of studies have examined the circumstance of acute odontoid fracture in the older patient. Three case series argue against surgical fixation in the elderly patient.(37,63,67) Seven other case series favor surgical fixation in this age group. There is also one case-control study by Lennarson et al providing Class II medical evidence for surgical treatment of elderly patients.(47)

Ryan et al described 30 patients over 60 years of age with Type II odontoid fractures. The fusion success rate in patients over age 60 treated with external immobilization was only 23%.(61) The authors felt that the high fracture nonunion rate was secondary to inadequate

immobilization and delays in diagnosis in the majority of cases. If these issues were eliminated, no significant difference in outcome between surgical and non-surgical management would have been demonstrated. They concluded that surgical fixation and fusion for elderly patients with odontoid fractures should be reserved for unusual circumstances.

Greene et al reported the largest series (120 patients) of retrospectively reviewed cases of traumatic odontoid Type II axis fractures.(37) Patients with dens displacement of six millimeters or greater in their experience had a non-union rate of 86%, compared to a non-union rate of 18% for patients with displacement less than six millimeters. The authors reported no significant relationship between fracture non-union and age using chi square analysis. It might be argued that statistical tests of association would be more appropriate in this circumstance and age may have been shown to be a factor had they been used.

Andersson et al described 29 patients age 65 and older with odontoid fractures managed with surgical and non-surgical means.(2) In their series, six of seven patients (86%) achieved successful fusion following posterior cervical C1-C2 arthrodesis. Worse results were observed in patients treated with anterior odontoid screw fixation (20% fusion rate) and in patients managed with external immobilization alone (20% fusion rate). These authors favored posterior cervical fusion over other management options in elderly patients with Type II odontoid fractures.

Pepin et al reported their experience with 41 acute odontoid fractures (Type I - one, Type II - 19, Type III - 21).(58) The authors found that halo immobilization was poorly tolerated in patients over 75 years of age. They suggested that early C1-2 fixation and fusion was appropriate in this group.

Hanigan et al described 19 patients over 80 years of age with odontoid fractures (Type II - 16, Type III - three).(42) Five patients with displacement greater than five mm were treated

with posterior cervical fixation and fusion with good results. Three of the five had stable non-unions. They reported a mortality rate of 26% in patients managed conservatively with prolonged immobilization rather than surgical fixation and fusion. On the other hand, they noted that no patient treated with external immobilization alone developed clinically significant instability.

Pitzen and colleagues described their experience with surgical therapy in seven patients with odontoid fractures over 70 years of age.(60) Two patients died of related medical comorbidity. Five patients did well and were mobilized early. The authors concluded that early surgical fixation in this age group is the preferred management strategy. This view is shared by several other investigators including Seybold et al, Campanelli et al, and Muller et al (14,57,67).

Bednar et al reported a prospective assessment of elderly patients with odontoid fractures managed with early operative stabilization and fusion.(5) Eleven patients were included in their study. The authors found a 91% fusion rate (10 of 11 patients). One patient died of unrelated causes. The authors argued in favor of early surgical intervention for elderly patients with odontoid fractures.

In 1997 Berleman et al offered a retrospective review of their experience with 19 patients over 65 years of age with Type II odontoid fractures treated with anterior odontoid screw fixation.(7) Radiographic fusion with nearly five-year follow-up was obtained in 16 of 19 patients (85%). The authors concluded that anterior odontoid screw fixation is a successful therapy for elderly patients with Type II odontoid fractures.

In the only case-control, Class II evidence study published on this topic, Lennarson et al examined 33 patients with isolated Type II odontoid fractures treated with halo vest immobilization.(47) Age greater than 50 was found to be a significant factor for failure of fusion in a halo immobilization device. Patients age 50 and greater had a risk for non-union 21 times

higher than that found for patients under age 50 years. No significant effect on outcome was found due to other medical conditions, sex of the patient, degree of fracture displacement, direction of fracture displacement, length of hospital stay or length of follow-up.

## **Traumatic Spondylolisthesis of the Axis (Hangman's Fracture)**

### *Overview*

Traumatic fractures of the posterior elements of the axis often related to hyperextension injuries from motor vehicle accidents, diving, and falls, are reminiscent of the injury induced to the axis by judicial hangings.(65,78) A distinction has been made between the two fracture types because the mechanisms of injury are different. The mechanism of injury associated with judicial hanging is one of distraction and hyperextension. The more common Hangman's fracture injury induced by motor vehicular trauma is typically a result of hyperextension, compression and possible rebound flexion. The incidence of head injury is high with the latter Hangman's fracture injury type.

Wood-Jones described the cervical fracture-dislocation injury induced by hanging in 1913.(78) Garber used the term "traumatic spondylolisthesis" of the axis in 1964. He described eight patients with "pedicular" fractures of the axis following motor vehicle accidents.(33) The term "Hangman's Fracture" has been attributed to Schneider et al who described a series of eight patients and noted the similarity between the fracture of the posterior elements of the axis to the pattern of fracture injury induced by judicial hanging.(65) Williams documented four cases of Hangman's fracture injury in 1975, noting that three occurred due to motor vehicle accidents and the fourth due to a fall.(77) A variety of authors have suggested that a more appropriate term for describing this axis injury type may be "traumatic spondylolisthesis of the axis" due to the

differences in mechanism of injury between hanging and the deceleration injuries of falls and motor vehicle accidents.(29,30) The majority of traumatic spondylolisthesis fractures of C2 due to motor vehicle accidents appear to result from hyperextension and compression, rather than the hyperextension and distraction associated with hangings. These differences in the mechanism of injury, along with the wide range of neurological deficits identified with these injuries prompted a series of investigators to attempt to better characterize and classify traumatic spondylolisthesis injuries of the axis.

#### *Classification of Hangman's Fractures*

In 1981 Pepin and Hawkins published a two-type classification scheme for Hangman's fractures. Type I was described as a non-displaced fracture of the posterior elements alone. Type II was a displaced fracture involving the posterior elements and the body of C2.(59) They successfully treated 42 patients without surgery using their scheme, which involved reduction (Type II injuries) and immobilization. They noted a low incidence of associated spinal cord injury but a frequent association with head injury. Although simple and effective, Pepin and Hawkins' scheme has not gained popular acceptance and is not widely used.

In the same year Francis, Pepin, Hawkins and others published a collaborative experience with 123 patients with traumatic spondylolisthesis of the axis. Injuries were divided into one of five grades based on displacement and angulation of C2 on C3.(30) Grade I was defined as displacement less than 3.5 mm and angulation less than 11 degrees. Grade V was defined as complete C2-3 disc disruption. Grade IV in their scheme had greater than 3.5 mm of C2-3 disruption but less than half of C3 vertebral width with greater than 11 degrees of C2-3 angulation. Grades II and III were injury types graded between Grades I and IV.

Effendi et al described three types of fractures of the ring of the axis based on a series of 131 patients.(26) Their classification scheme was based on the mechanism of injury: Type I Axial loading and hyperextension, Type II Hyperextension and rebound flexion, Type III Primary flexion and rebound extension. Type I fractures were defined as isolated hairline fractures of the ring of the axis with minimal displacement of the body of C2. Type II fractures were defined as displacement of the anterior fragment with disruption of the disc space below the axis. Type III fractures were defined as displacement of the anterior fragment with the body of the axis in a flexed position in conjunction with C2-3 facet dislocation. This Type III fracture is associated with a flexed forward position of the axis body. The incidence of Type I, II and III fracture injury in their series was 65%, 28% and 7% respectively.

Levine and Edwards modified Effendi's classification scheme in 1985.(48) They added flexion-distraction as a mechanism of injury (Type IIA) and offered a tailored treatment strategy for each of the four injury types.

In the largest series of axis fractures yet described, both the classification schemes of Effendi et al and that of Francis et al were utilized to characterize 74 Hangman's fractures.(37) The most common fracture pattern identified was the Effendi Type I (72%) and the Francis Grade I (65%). The investigators found a strong correlation between Effendi Types I and III and Francis Grades I and IV, respectively.

Not all authors feel that all Hangman's fractures fit into one or both of these classification schemes. In the review by Burke et al of 165 acute injuries of the axis vertebra, 62 (38%) were traumatic spondylolisthesis of the axis: including thirteen Effendi I, 35 Effendi II and three Effendi III injuries.(13) Eleven patients (18%) had a fracture pattern not previously described in which one or both fractures involved a portion of the posterior cortex of the body of the axis.

### *Incidence of Traumatic Spondylolisthesis and Associated Injuries*

In Greene's series of 1,820 cervical fractures, 340 (19%) were of the axis and 74 (4%) were Hangman's type.(37) In the series of acute fractures of the axis vertebra described by Burke et al, injuries of the axis were associated with other fractures of the cervical vertebra in 8% of cases.(13) Ryan and Henderson studied 657 patients with cervical spine fractures over a 13-year period. Hangman's type fractures occurred as isolated fractures in 74% of their series.(62) Only nine percent were associated with fractures of C1. An additional nine percent were associated with subaxial cervical spine fractures. In the series of Guiot et al of ten complex combined atlantoaxial fractures only one involved a Hangman's injury.(38)

Although the incidence of spinal cord and nerve root injury as a result of a Hangman's fracture is reportedly low, unstable Hangman's injuries do occur with some frequency.(12,59) If the patient survives the initial injury, it has been proposed that the relatively spacious intracanalicular diameter affords some protection against spinal cord compression.(55) Starr et al described an atypical fracture pattern occurring through the posterior aspect of the vertebral body with continuity of the posterior cortex or pedicle with narrowing of the spinal canal due to the associated subluxation.(68) In their series of 19 patients, this Hangman's fracture variant occurred in six patients including two patients with resultant paralysis. In the series described by Francis et al, eight of 123 patients they managed had neurological deficits (6%).(30) Tan's retrospective series of 33 Hangman's fractures included 14 patients with no neurological deficit on admission.(70) The other 19 (57%) had neurological deficits ranging from quadriparesis to urinary retention. Twenty-eight patients (85%) returned to employment at one-year follow-up.

Mirvis' series of 27 patients had associated neurological findings in 26 % of patients with Hangman's fractures.(54)

Combination fractures of C1 and C2 in association with a Hangman's type C2 injury appear to have a higher incidence of associated neurological injury, likely due to increased instability and a more severe traumatic injury pattern. (23, 38)

### *Treatment*

The majority of patients with traumatic Hangman's fractures reported in all the literature reviewed was treated with cervical immobilization with good results. The three largest experiences reported are the multi-institutional series of Effendi et al, Francis et al, and the single institutional experience described by Greene et al.(26,30,37) Management strategies and surgical indications vary somewhat between investigators.

In the series reported by Effendi et al in 1981 there were 85 Type I fractures, sixty-two of which were managed with external immobilization. They reported thirty-seven Type II fractures; seventeen of the patients were treated with bracing and fifteen with surgical fusion. Of the patients managed surgically, four patients were treated with a C2-3 anterior fusion and eleven were treated with dorsal internal fixation and fusion. Nine patients had Effendi Type III fractures. Three died without definitive treatment, one was managed in a brace and five were treated surgically with fusion, one anterior and four posterior. The authors concluded that the majority of Hangman's fractures were best managed non-operatively. They commented that they may have over-treated patients early in their series offering surgery when external immobilization may well have been successful.(26) They decided that surgery should be reserved for unusual Type III fractures and those patients with failure of fusion despite three months of halo immobilization.

In Pepin's series also reported in 1981, all 42 patients with Hangman's fractures they treated healed successfully with external immobilization alone.(59) Francis et al described and classified Hangman's fracture injuries in 123 patients from four institutions.(30) Injuries were categorized into Grades I through V based on displacement and angulation. There were 19 Grade I, nine Grade II, 46 Grade III, 42 Grade IV and seven Grade V fractures. All patients were initially managed with traction with conversion to a halo orthosis, or were treated in a halo vest without traction. Healing occurred in 116 patients (95%) with halo immobilization alone. Seven patients received surgical management with fusion for non-union despite halo immobilization (four had an anterior C2-3 fusion, two had a posterior C1-3 fusion, and one had a posterior C2-4 fusion). The authors assessed the injury type with respect to success with non-operative management. Three of nine Grade II injury patients (33%) and two of seven Grade V injury patients (28%) developed non-union despite halo management and required subsequent surgical treatment. No Grade I or Grade III injury patients and only one Grade IV injury patient failed halo treatment. The authors concluded that primary surgical treatment for Hangman's fracture injuries is not indicated. All patients should be provided late follow-up to assess for non-union, particularly Grade II and Grade V injury patients. When surgical management is considered, the authors recommend an anterior C2-3 fusion.

In Levine and Edwards series of 52 patients with Hangman's fractures, all isolated Type I, II and IIa injuries were managed non-operatively.(48) Three of five Type III patients underwent surgical stabilization and fusion for failure to obtain or maintain fracture reduction in a halo. The authors singled out the subgroup of the Effendi Type II fracture that significantly distracted with the application of craniocervical traction. They felt Type II injuries were likely the result of

flexion-distraction forces. The three patients with Type II fractures in their series were successfully treated with “mild compression - extension in a halo vest under fluoroscopic control”.

Greene et al noted a strong correlation between Effendi Type I and Francis Grade I Hangman’s injury and between Effendi Type III and Francis Grade IV fractures in their series of 74 patients.(37) Sixty-five of 74 patients were treated non-operatively with external immobilization for a median of 12 weeks. There were two early deaths. Seven patients required early surgical fixation and fusion for inability to maintain fracture alignment in a halo brace. All seven early surgical patients were either Effendi Grade II or III and five of the seven were Francis Grade III or IV. Overall, 33% of patients with unstable Effendi Types II and III or 36% of Francis Grade III, IV and V injuries required early surgical treatment. Eventually all seven achieved solid fusion without evidence of instability. The authors compared their experiences with those of Effendi et al and Francis et al, and concluded that conservative management (external immobilization) should be the initial treatment in virtually every patient with a Hangman’s fracture. Early surgical management should be reserved for unstable injuries ineffectively immobilized in a halo device.

In a combined clinical and cadaveric anatomic study, Mestdagh et al described their experience with 41 fractures of the posterior neural arch of the axis.(53) Eleven cases were treated surgically with anterior C2-3 interbody fusion and thirty patients were treated with external immobilization. Thirty patients were available for follow-up. Cervical mobility was better in patients managed conservatively. Displacement of up to five mm at the Hangman’s fracture site in a cadaveric study was compatible with stability without disruption of the ligaments or the C2-C3 disc space. The authors concluded that conservative management was the ideal treatment for Hangman’s fractures except in cases of marked instability or failure of union.

Grady et al reported their experience with 27 patients with Hangman's fractures. Sixteen were managed in a halo device, eight in a rigid collar and three with bed rest only.(36) All achieved fusion with no residual symptoms. The authors concluded that use of a Philadelphia collar alone for Hangman's fractures is a reasonable alternative to halo immobilization particularly for injuries with minimal C2-C3 displacement.

In 1987, Govendor and Charles prospectively studied 39 patients with traumatic spondylolisthesis of the axis.(34) Injuries were classified for stability based on the criteria of White and Panjabi.(76) All patients were successfully treated with collar immobilization regardless of assessment of stability. The authors argue against basing treatment on dynamic imaging as advocated by Effendi et al and Levine and Edwards. (26,48)

A variety of other reports favor non-operative management of Hangman's fractures.(4,11,17,28,34,48,51,53,66,71)

### *Surgical Management*

Surgical options for unstable Hangman's fracture injuries, particularly those that fail to heal despite external immobilization, include anterior C2-3 interbody fusion and dorsal C1-C3 fusion procedures.

In the series of Effendi et al forty-two of 131 patients with Hangman's fractures were treated surgically.(26) Ten were treated with an anterior C2-3 fusion and 32 underwent a posterior fusion. All were successfully stabilized at last follow-up.

In the Francis et al series of 123 Hangman's fracture patients, only seven patients were treated surgically. Four underwent anterior C2-3 fusion, two had a posterior C1-3 fusion, and one underwent posterior C2-C4 fusion.(30) The authors noted that six out of the seven patients

requiring fusion for non-union had C2-C3 angulation greater than 11 degrees. All seven patients achieved bony stability.

A number of case-series of Hangman's fractures offer similar experiences with surgical management. McLaurin et al described their experience with early fusion in two patients with Hangman's fractures in order to allow early mobilization. The authors acknowledged that both injuries would likely have healed with external immobilization alone.(52) Salmon described 20 patients with Hangman's fractures treated with posterior interlaminar wiring and fusion with no morbidity. (64)

Verheggen et al, in their 1998 report, argued strongly for surgical fixation and fusion of Effendi Type II and III Hangman's fractures. In their opinion the optimal management of these injuries remains controversial. They described 16 patients with Hangman's fractures they treated with surgical fixation of the posterior arch of the axis with screw fixation. They found that this fixation technique resulted in superior functional results as compared to historical controls.(73) They favor this management strategy in the setting of the Levine and Edwards type IIa fracture.(48) Their viewpoint is challenged by Sybert in his comments which accompany their article.(73)

Borne et al in 1984 published their approach to the management of pedicular fractures of the axis.(10) They utilized a technique of bilateral posterior screw fixation. They described excellent results and a 100% fusion rate. Despite this, their technique has not gained widespread acceptance.

### **Fractures of the Axis Body**

A number of authors have addressed the management of non-odontoid, non-Hangman's fractures of the axis. They have been labeled as miscellaneous fractures of the axis, non-odontoid

non-Hangman fractures, or simply axis body fractures.(6,32,37,41) There have been several attempts at classifying the various fracture types within this diverse group.

Benzel et al reported on 15 patients with fracture of the axis body and divided them into three types: coronal, sagittal and transverse oriented.(6) The latter group was felt to represent the same group as the Anderson and D'Alonzo Type III odontoid fracture. The authors proposed that the Type III odontoid fracture classification be discarded since it is misleading. The original authors had the same thought. Benzel and colleagues offered a mechanism of injury for each of the three fracture types they described. No treatment or outcome data was included in their report.

Greene et al described 67 patients with miscellaneous axis fractures of all types.(37) Of the 61 patients available for follow-up (median follow-up 14 months), all but one was successfully managed with a variety of non-operative means. The authors note that this is a diverse injury group and describe a treatment algorithm based on features of fracture stability. Only one patient with a miscellaneous axis fracture required surgical intervention for delayed non-union.

Fujimara et al classified 31 axis body fractures based on radiographic injury pattern: avulsion, transverse, burst or sagittal.(32) All nine cases of avulsion fracture and the two cases of transverse fracture healed with external immobilization. Two of the three burst fractures were treated with C2-3 anterior interbody fusion. Of the 17 sagittal fractures, 15 healed with non-operative treatment. The remaining two patients required surgical fusion. The authors recommend initial non-operative treatment for all non-odontoid, non-Hangman's axis fractures.

Craig et al added nine cases of axis fractures involving the superior articular facet.(19) In seven patients there was an associated odontoid fracture. This fracture occurred in either the coronal or sagittal plane resulting in shearing of the anterior or lateral portion of the facet

complex. The lateral mass of the atlas was noted to occasionally sublux into the facet fracture. The authors recommended immobilization for non-displaced fractures and the consideration of surgical reduction, fixation and fusion for difficult to reduce fractures.

Bohay et al described three unusual fractures of the posterior body of C2, all of which responded to non-operative management.(8) Jakim and Sweet contributed a single case.(43)

Korres et al described fourteen patients with avulsion fractures of the anterior inferior portion of the axis that they believed to be extension-type injuries.(46) These cases represented only 3% of the cervical spine fractures they managed over a 12-year period. All fourteen of these body fracture types were successfully managed with cervical immobilization (mean follow-up of 8.5 years).

## **SUMMARY**

### **Fractures of the Odontoid**

There is no Class I medical evidence addressing the issues of management of acute traumatic odontoid fractures. A single Class II evidence paper reviews the management of Type II odontoid fractures in halo immobilization devices. This study demonstrated a 21-fold increase in risk of non-union with halo immobilization in patients over the age of 50 years. All other articles reviewed contain Class III evidence that supports several treatments.

Type II Odontoid fractures in patients 50 years of age and older should be considered for surgical stabilization and fusion. Type I, Type II and Type III fractures may be managed initially with external cervical immobilization. Type II and Type III odontoid fractures should be considered for surgical fixation in cases of dens displacement five mm or greater, comminution of the odontoid fracture (Type IIA) and/or inability to achieve or maintain fracture alignment with

external immobilization. Isolated Type I and Type III odontoid fractures may be treated with cervical immobilization, resulting in fusion rates of 100% and 84%, respectively. Anterior surgical fixation of Type III fractures has been associated with a 100% fusion rate. Type II odontoid fractures may be treated with external immobilization or surgical fixation and fusion. Halo immobilization and posterior fixation have both been used successfully for these injuries. Anterior odontoid-screw fixation has been reported with up to a 90% fusion success rate, except in older patients. Treatment of Type II odontoid fracture with a cervical collar alone or traction followed by cervical collar immobilization may also be undertaken, but have lower success rates.

## Traumatic Spondylolisthesis of the Axis

There is no Class I or Class II medical evidence addressing the management of traumatic spondylolisthesis of the axis. All articles reviewed contain Class III evidence that supports a variety of treatments. The majority of Hangman's fractures heal with 12 weeks of cervical immobilization either with a rigid cervical collar or a halo immobilization device. Surgical stabilization is an option in cases of severe angulation (Francis Grade II and IV, Effendi Type II), disruption of the C2-3 disc space (Francis Grade V, Effendi Type III), or the inability to establish or maintain fracture alignment with external immobilization.

## Fractures of the Axis Body (Miscellaneous Axis Fractures)

There is no Class I or Class II medical evidence addressing the management of traumatic fractures of the axis body. All articles reviewed contain Class III evidence that supports the use of external immobilization as the initial treatment strategy.

**Table I: Initial Management of Isolated Axis Fracture in the Adult**

Fracture Type	Treatment Options
Odontoid fracture Type I Type II Type IIA Type III	Collar, halo Consider for early surgery or halo, collar Consider for early surgery or halo Collar, halo, surgical fusion
Traumatic Spondylolisthesis of the Axis (Hangman's Fracture) <u>Stable</u> Effendi Type I, Francis Type I, II <u>Unstable</u> Effendi Type II, III, Francis Type III, IV, V	Halo, collar Halo, consider surgical stabilization
Miscellaneous Axis Fractures	Collar or halo

## **KEY ISSUES FOR FUTURE INVESTIGATION**

More data are necessary to determine treatment standards and/or guidelines for the definitive management of odontoid fractures. For Type I and Type III fractures the available Class III evidence suggests that a well-designed multicenter case-control study could provide sufficient evidence to define their appropriate management in the early post-injury period. For Type II fractures, the literature suggests that both operative and nonoperative management remain treatment options. A randomized or case-control study would be of benefit in establishing definitive treatment recommendations for this fracture type.

Traumatic spondylolisthesis of the axis and miscellaneous axis fractures are treated successfully with external immobilization in the majority of cases. A multicenter case-controlled study of patients with these injury types would help to define optimal treatments of each specific fracture subtype.

## EVIDENTIARY TABLE one - Odontoid Fracture

Author / Year	Study Design	Data Class	Comments
Anderson S et al, <i>Eur Spine J</i> , 2000	Retrospective non-randomized report of 29 patients with odontoid fractures over age 65 managed with posterior fusion, anterior odontoid fixation or immobilization.	III	Posterior fusion resulted in 7/7 fused (100 %). Anterior odontoid screw resulted in three / 11 fused (27%) and Halo immobilization resulted in three /10 fused (30%). The authors argue for posterior fusion in the elderly patient with an odontoid fracture.
Apfelbaum RI et al, <i>J Neurosurg</i> , 2000	Retrospective review of two institution experience with anterior odontoid screw fixation. 147 odontoid fractures (Type II n = 138, Type III n = 9) divided into recent (within six months, 129 patients) and remote (greater than six months from injury, 18 patients) groups.	III	The fusion rate was significantly higher in the recent group comparing fusion rates of 88 % versus 25 % (p <0.05) with a mean follow-up of 18 months. A positive correlation was seen between fusion and fractures oriented in the horizontal or posterior oblique planes. No effect of age, sex, number of screws placed or displacement was demonstrated.
Dai LY et al, <i>Eur Spine J</i> , 2000	Review of 57 cases of failed management for odontoid fracture	III	50 treated with occipitocervical fusion and seven with atlantoaxial fusion. Two cases of non-union after atlantoaxial fusion alone. 38 achieved an excellent result.
Lennarson PJ et al, <i>Spine</i> , 2000	33 patients with isolated Type II odontoid fracture treated with halo vest immobilization. Cases defined as non-unions in halo and controls defined as unions.	II Case-control	Patients 50 years or older had a risk for failure 21 times higher than age under 50. No significant difference in medical conditions, sex, amount of fracture displacement, direction of fracture displacement, length of hospital stay, length of follow-up between groups.
Julien T et al, <i>Neurosurg Focus</i> , 2000	Evidence-based review of management of odontoid fractures	III	Authors conclude there is insufficient evidence for standards or guidelines. Type I and III odontoid fractures can be managed with external immobilization (100 % and 84 % fusion respectively). Anterior fixation for Type III odontoid fractures appears to improve the fusion rate to nearly 100 %. Type II fractures can be managed with external immobilization with an expected fusion rate of approximately 65%. Surgical instrumentation and fusion appears to improve the fusion rate and include posterior cervical fusion (74 %) or anterior odontoid screw fixation (90 %) with acceptable morbidity.
Campanelli M et al, <i>Surg Neurol</i> , 2000	7 patients with displaced Type II odontoid fractures underwent posterior transarticular screw fixation.	III	6 / seven (86%) achieved rigid immobilization. One vertebral injury. The authors conclude that this is a reasonable option.

Author / Year	Study Design	Data Class	Comments
Muller EJ et al, <i>Eur Spine J</i> , 1999	Retrospective review of 23 patients over 70 years of age with odontoid fractures.	III	Complication rate significantly increased in the elderly group (52 versus 33 %) primarily due to non-union after non-operative treatment. The authors suggest that the elderly patient is at high-risk for morbidity and mortality and suggest early halo fixation or primary stabilization.
Morandi X et al, <i>Surg Neurol</i> , 1999	17 cases of odontoid screw fixation	III	Fusion in 16 of 17 (94%). The authors suggest patient selection for anterior fixation be based on the orientation of the fracture line.
Subach BR et al, <i>Neurosurg</i> , 1999	26 patients (mean age 35) with Type II fractures treated with anterior odontoid screw fixation (single screw) plus collar (median 7.2 weeks).	III	25/26 fusion (96%). One patient required posterior fusion for inadequate reduction.
Seybold EA and Bayley JC, <i>Spine</i> , 1998	Retrospective review of 37 Type II and 20 Type III odontoid fractures divided into age groups. Age less than 60 and greater than 60.	III	Under age 60: Type II, 7/12 fused (58%) Over age 60: Type II, 8/11 fused (73%) Type III – all treated with halo, 95% fusion regardless of type Fusion rates did not differ significantly between the two groups. The authors noted a decreased tolerance in the elderly patient for halo immobilization and as a result favor surgery in select cases.
Jenkins JD et al, <i>J Neurosurg</i> , 1998	Comparison of one versus two screws in non-selected patients with Type II odontoid fractures divided into two groups: 20 (single screw) versus 22 (two screws). Follow up nine months.	III	The difference in fusion rate, 81% (one screw) versus 85%(two screws), was not significant.
Berlemann U and Schwarzenbach O, <i>Acta Orthop Scand</i> , 1997	19 patients with Type II odontoid fractures over 65 years treated with anterior odontoid screw fixation with a followup of 4.5 years.	III	Bony fusion in 16/19 (84%) and 15 / 19 (79%) were asymptomatic. The authors favor anterior fixation in this age group.
Traynelis VC, <i>Clin Neurosurg</i> , 1997	Evidence based review of Type II odontoid fractures.	III	First evidence based report on odontoid fracture management. Indicated that insufficient data was available for standards or guidelines and that four treatment options for Type II odontoid fractures were available including: traction followed by immobilization, immobilization with Halo or Minerva, posterior cervical fusion or anterior screw fixation. The author notes that the higher fusion rate reported with anterior screw fixation might be offset by its higher complication rate and learning curve.

Author / Year	Study Design	Data Class	Comments
Greene KA et al, <i>Spine</i> , 1997	Retrospective review of 340 cases of axis fractures, including 199 odontoid fractures.	III	<p>Type I: two patients, 2/2 healed with Halo immobilization (12 weeks)</p> <p>Type II: 120 patients, 20 treated with early surgery - eight had Type II with greater than six mm, four Type IIA with instability despite external immobilization (one Type IIA treated successfully with a halo), seven patients underwent surgical fusion to avoid halo immobilization</p> <p>95 treated with external immobilization (median of 13 weeks) - 88 available for followup - solid fusion failed in 25 (28.4 %) - seven successfully treated with additional immobilization and 18 successfully treated with posterior fusion (late surgery) - significant factors dens displacement greater than six mm (chi square 33.74, p &lt; .0001) giving an 86 % failure rate in the halo treatment group</p> <p>5 died</p> <p>Type III: 77 patients, 69 managed non-operatively with external immobilization - 68 fused (median 12 weeks) - the one that failed also had a C1 posterior arch fracture and required posterior fusion</p> <p>6 patients were treated with early surgery - five because the halo failed to maintain alignment and one because of a combined C2-3 subluxation - two had concomitant lateral mass fractures of the atlas with avulsion of the ligamentous insertion on the tubercle</p> <p>2 died</p> <p>Conclusions: The highest non-union rate was observed in Type II odontoid displaced six mm or more.</p> <p>Surgery recommended for 1). Acute fracture instability despite external immobilization, 2). Transverse ligament disruption and 3). Type II odontoid fracture with greater than six mm displacement.</p>
Polin RS et al, <i>Neurosurgery</i> , 1996	Retrospective review of 36 Type II fractures treated with Philadelphia collar (16) or Halo vest immobilization (20).	III	<p>Type II</p> <p>54 % fused with collar</p> <p>74 % fused with Halo</p>

Author / Year	Study Design	Data Class	Comments
<p>Chiba K et al, <i>J Spinal Disord</i>, 1996</p>	<p>104 pts., with odontoid fractures:  Type I - two pts.  Type II -62 pts.  Type III - 32 pts.</p> <p>2 groups:  Fresh group, 72 pts. whose fractures were identified within three weeks of traumatic event</p> <p>Old group, 32 pts. who had an extended period before definitive treatment 32 pts.  1 type I, 21 type II, and eight type III.</p>	<p>III</p>	<p><u>Type I</u>: two patients, Collar 2/2, Both fused (100%)  <u>Type II</u> (62 patients), Immobilization 10/62  Surgery 52/62  In fresh fracture group treated with surgery 31/32 fused (97%).  In delayed fracture group treated with surgery 13/19 fused (68 %).  <u>Type III</u> (32 patients), Surgery 15 /32 (47%) fused  Immobilization 17 /32 (53%) fused, ten /15 (66%) treated with surgery fused  11 / 17 (65%) treated with immobilization fused  (Every patient treated with a halo fused 5/ five 100 %)</p> <p><u>Surgical Procedures</u> (66 patients)  <i>Posterior cervical fusion</i>, (10) Patients  Type II: 9/9 successful fusions (100%)  Type III: 1/1 successful fusions (100%)  <i>Anterior screw fixation</i>: (46 patients).  36 type II, ten type III, 42/45 pts. achieved bony union (93.3%)  <i>Transoral fusion</i>, (9 patients), 6/8 type II successful fusions (75%), 1/1 type III successful fusions (100%)</p> <p><u>The authors of this large series conclude:</u>  Type I fractures can generally be managed non-operatively.  Anterior screw fixation recommended for most Type II and unstable Type III fractures.  Contraindications include old established non-unions, irreducible fractures, caudal displacement, severe osteoporosis.  Type III fractures can be treated with Halo immobilization or anterior screw fixation.  Established non-unions and irreducible fractures should be treated with posterior fusion.  Transoral fusion reserved for rare cases of anterior cord compression.</p>
<p>Bednar DA et al, <i>J Spinal Disord</i>, 1995</p>	<p>Prospective report of early surgical stabilization in 11 geriatric patients with odontoid fractures.</p>	<p>III</p>	<p>The authors suggest that mortality can be reduced by surgical intervention and avoiding the use of Halo immobilization.</p>
<p>Dickman C and Sontag V, <i>J Neurosurg</i>, 1995</p>	<p>14 pts. with either acute or subacute type II fractures treated with anterior odontoid screw fixation.  Radiographic criteria for fusion- postoperative radiographs and CT scans.</p>	<p>III</p>	<p>Type II  14/14 successful fusions (100%).</p>

Author / Year	Study Design	Data Class	Comments
Dickman CA et al, <i>J Neurosurg</i> , 1995	Describes salvage procedures for failed atlantoaxial non-unions.	III	Report includes two cases in which anterior atlantoaxial transarticular screws were used and eight cases of posterior transarticular screws.
Coyne TJ et al, <i>J Neurosurg</i> , 1995	15 pts. treated with posterior wire fusion and immobilized postoperatively in either Philadelphia-collar or Halo. Minimum follow-up two years, mean 4.7 years  Radiographic criteria for fusion- absence of C1-2 movt. on lateral flex./ext. radiographs and evidence of continuity of trabecular bone formation between C1 and C2 across the graft.	III	Type II: 13/14 successful fusions (93%) Type III: 2/2 successful fusion (100%)
Hanigan J et al, <i>J Neurosurg</i> , 1993	19 patients over 80 years of age with odontoid fractures ( Type II - 16, Type III - 3).	III	Five patients with displacement greater than five mm required posterior surgical fixation with good results. There was a 27 % mortality in the conservative treatment group with prolonged immobilization noted as one of the contributing factors.
Waddell J and Reardon G, <i>Can J Surg</i> , 1993	24 patients with odontoid fracture: 20 type II and four type III fractures. 16 of the 20 type II fractures were treated with C 1-2 arthrodesis (Gallie procedure). All type III fractures were treated non-operatively.	III	Type II: 15/16 successful fusions (94%), one pt. was lost to follow-up Type III: 3/4 successful fusions (75%), 1/4 nonunion (25%)
Ryan M and Taylor T, <i>J Spinal Disord</i> , 1993	30 patients over 60 years of age with Type II fractures.	III	The fusion rate in the patients over age 60 treated with immobilization was only 7/29 (23 %). Despite the low fusion rate for this age group, the authors favor halo immobilization over surgical fixation..
Bucholz RW, <i>Clin Orthop</i> , 1989	26 pts. 0 type I, 17 type II, nine type III. Pts. were immobilized in Halo for a minimum of three months and if no movt. on flex./ext. radiographs, placed in a Philadelphia-collar for an additional four weeks. Radiographic criteria for fusion - no movt. or subluxation at the fracture site on flex./ext. radiograph.	III	Type II 15/17 successful fusions (88%) 2/17 nonunions (12%) Type III 9/9 successful fusions (100%)  Three deaths: 2pts. had type II fractures while being treated in Halo and one pt. with type III fracture.

Author / Year	Study Design	Data Class	Comments
Hadley MN et al, <i>Neurosurg</i> , 1988	Retrospective study including 62 patients with Type II odontoid fractures, including three with comminution at the base.	III	The subgroup of Type II odontoid fracture with comminution at the base was defined as the Type IIA odontoid fracture. The clinical significance of this observation was that fracture fused poorly with immobilization and was considered for early surgery.
Govender S and Grootboom M, <i>Injury</i> , 1988	Review of 41 patients with odontoid fractures: 26 type II, 15 type III.  One month in traction (2-4 Kg), then a rigid collar for 6-8 weeks, and assessed at three months.  Radiographic criteria for fusion: bony continuity across fracture site and no movt. on flex./ext. tomograms	III	Type II - 19/26 successful fusions (73%) 2/26 fibrous unions (8%) 5/26 nonunions (19%)  Type III - 15/15 successful fusions(100%)  - No mortality  - seven Halo pin site infections  - three pts. had skin excoriation over chin secondary to halter traction.
Fujii E et al, <i>Spine</i> , 1987	Retrospective review of 52 pts. with odontoid fractures, including data on 24 treated with immobilization, 10 treated with anterior screw fixation and seven treated with posterior fusion. Radiographic criteria for fusion- AP and lateral tomograms.	III	<u>Immobilization</u> Type I, 1/1 successful fusion (100%) Type II, 3/7 successful fusions (43%) Type III, 10/14 successful fusions (72%) <u>Posterior fusion</u> Type II, 7/7 successful fusions (100%) <u>Anterior screw fixation</u> 6/8 type II successful fusion (75%) 2/2 type III successful fusion(100%)
Lind B et al, <i>Spine</i> , 1987	Review of 14 pts. with odontoid fractures managed with Halo immobilization and evaluated at 12 weeks with flex/ext. radiographs. Included type nine type II and five type III fractures with a two year follow-up. Radiographic criteria for fusion - lateral flex./ext. radiographs.	III	10/11 successful fusions (91%) combined type II and type III fractures. Authors support the use of Halo immobilization as the initial treatment for Type II and III odontoid fractures.

Author / Year	Study Design	Data Class	Comments
Dunn ME and Seljeskog EL, <i>Neurosurg</i> , 1986	Retrospective report of 80 pts. with odontoid fractures including data on 74 patients treated primarily with rigid bracing for 3-6 months followed by additional collar support for six weeks and 41 patients undergoing posterior cervical fusion. Minimum follow-up period was six months, 80% of the patients had follow-up longer than eight months. Radiographic criteria for fusion - lateral flex./ext. radiographs at 3-4 months.	III	Rigid immobilization Type II 40/59 successful fusions (68%) 19/59 nonunions (32%) Type III 15/15 successful fusions (100%)  Posterior fusion 40/41 successful fusions (98%) for combined type II and type III fractures.
Clark CR and White AA, <i>J Bone Joint Surg Am</i> 1985	Multicenter review including 144 patients managed by 27 different surgeons. Fusion rates reported based on fracture type and treatment. Radiographic criteria for fusion-evidence of trabeculation across the fracture site and absence of movt. on lateral flex./ext. radiograph.	III	<u>No treatment</u> Type II: 0/18 successful fusions (0%) Type III: 0/3 successful fusions (0%) <u>Collar</u> Type II: 0/3 successful fusions (0%) Type III: 5/10 successful fusions (50%) <u>Traction</u> Type II: 2/3 successful fusions (66%) Type III: 7/8 successful fusions (88%) <u>Halo</u> Type II: 25/38 successful fusions (66%) Type III: 13/16 successful fusions (81%) <u>Anterior fusion</u> Type II: 7/8 successful fusions (88%) Type III: 2/2 successful fusions (100%) <u>Posterior fusion</u> Type II: 25/26 successful fusions (96%) Type III: 4/4 successful fusions (100%)

Author / Year	Study Design	Data Class	Comments
Pepin JW et al, <i>Clin Orthop</i> , 1985	Retrospective review of 41 patients with odontoid fractures including 26 treated conservatively with tongs, four-poster brace, collars and/or Halo vests (0 type I, 13 type II, 13 type III). 12 patients underwent posterior cervical fusion (1 type I, four type II and seven type III). Radiographic criteria for fusion - union on plain radiograph and tomogram as well as lateral flex./ext. views. Nonunion was defined as movement of the dens fragment on lateral flex/ext. radiographs	III	<u>Halo/Traction</u> Type II: 6/13 successful fusions (46%) Type III: 11/13 successful fusions (85%) <u>Posterior cervical fusion</u> Type I: 1/1 successful fusions (100%) Type II: 4/4 successful fusions (100%) Type III: 7/7 successful fusions (100%) The authors noted that the Halo vests were poorly tolerated in patients over age 75.
Wang GJ et al, <i>Spine</i> , 1984	Retrospective review of 25 patients with odontoid fractures treated with a variety of cervical immobilization techniques.	III	Type I: 1/1 fused with Halo (100 %) Type II: 4/7 fused with collar (57 %) 4/5 fused in Halo (80 %) Type III: 2/2 fused with collar (100 %) 10/12 fused with Halo (83 %)
Bohler J, <i>Surg Annu</i> , 1982	15 pts. With odontoid fractures, both acute and chronic treated with anterior screw fixation followed by a period of cervical fixation in a plastic collar for a period of 4-16 weeks. Pt. distribution 0 type I, eight type II, and seven type III. Radiographic criteria for fusion- not given	III	Type II 8/8 successful fusions (100%) Type III 7/7 successful fusions (100%)

Author / Year	Study Design	Data Class	Comments
Maiman DJ and Larson SJ, <i>Neurosurg</i> , 1982	Retrospective review of 49 cases of odontoid fracture, including 34 type II fractures treated with early posterior wire/graft stabilization. Post-op immobilization with a Minerva for an average of five weeks. Two type III fractures were included. Radiographic criteria for nonunion: tomographic evidence of avascular necrosis, gross instability with a demonstrable gap at the fracture line and no evidence of healing. Fusion results evaluated six months post-surgery	III	The authors observed a 100 % fusion rate at the posterior surgical site, but only a 35 % fusion rate across the fracture site.
Ryan MD and Taylor TK, <i>J Bone Joint Surg Br</i> , 1982	Retrospective review of 23 pts. with odontoid fractures over a ten year period including one type I, 16 type II and six type III. Radiographic criteria for fusion - no movement on lateral flex./ext. radiographs.	III	Halo/Minerva/ SOMI Type I: 1/1 successful fusion Type II: 9/15 successful fusions (60%) Type III: 6/6 successful fusions (100%)
Ekong CE et al, <i>Neurosurg</i> , 1981	Retrospective review of 22 cases of odontoid fracture treated with Halo immobilization for three months. Type I, - 0 Type II - 16 Type III - 6 Includes outcome on 17 patients with an average follow-up of 30 months. Radiographic criteria for fusion - lateral flex./ext. radiographs.	III	Type II: 6/12 successful fusions (50%) Type III: 4/5 successful fusions (80%)
Marar BC and Tay CK, <i>Aust NZ J Surg</i> , 1976	Review of 26 cases of odontoid fracture including 24 type II and two type III treated with cervical traction for up to ten weeks. Radiographic criteria for fusion - fibrous union at fracture site.	III	Type II - 9/24 successful fusions (37.5%) Type III - 2/2 successful fusions (100%)

Author / Year	Study Design	Data Class	Comments
Anderson LD and D'Alonzo RT, <i>J Bone Joint Surg, Am</i> , 1974	Retrospective review of 49 patients with odontoid fractures classified into Type I, II and III based on fracture.	III	<u>Non-operative treatment</u> (37 patients) Type I: Collar / Brace, 2/2 successful fusions (100%) Type II: Halo, 14/22 successful fusions (64%) 8/22 nonunions (36%) Type III: Halo, 12/13 successful fusions (92%) 1/13 nonunion (8%) <u>Operative treatment</u> (12 patients) Type II: eight / ten successful fusions (80 %) Type III: 2/2 (100 %)

## EVIDENTIARY TABLE two - Traumatic Spondylolisthesis of the Axis

Author/ Year	Study Design	Class	Comments
Barros TE et al, <i>Spinal Cord</i> , 1999	Case report of surgical fixation in Hangman's fracture	III	Surgical treatment for Hangman's fracture is an option.
Verheggen R and Janses J, <i>Surg Neurol</i> , 1998	Retrospective study of 16 patients treated with early posterior screw fixation of the neural arch following Hangman's fracture.	III	The authors suggest that this is the optimal therapy for Edwards and Levine (Effendi) Type II and III fractures describing excellent results in their series.
Greene KA et al, <i>Spine</i> , 1997	340 cases axis fractures, including 74 patients with traumatic spondylolisthesis of the axis. Followup available on 72.	III	Most common: Effendi Type I (72 %) Francis Grade I (65%) 65 treated successfully with immobilization (12 weeks) 7 required early surgery (posterior fusion) due to poor alignment in the halo. (Effendi II - six and Effendi III – 1, by Francis Grading one -I, 1-II, 2- III, 3-IV) 33 % of all Effendi types II and III and 36 % of all Francis types III, IV, V patients required surgery. Strong correlation observed between Effendi I and Francis I and Effendi III and Francis IV. Conclusions: Immobilization is generally sufficient treatment. Surgery may be considered for severe Francis or Effendi type Hangman's fractures.
Corric D et al, <i>J Neurosurg</i> , 1996	Retrospective review of Hangman's fracture including 39 non-displaced (less than six mm C2 on C3) treated with non- rigid immobilization (Philadelphia collar for and average of 12 weeks) and ten displaced (greater than six mm) treated with halo (3), collar (6) or surgery (1)	III	Non-displaced group 39 / 39 fused using collar alone. Displaced group also fused regardless of treatment. C1-3 fusion required in one patient for failure of closed reduction.
Starr JK et al, <i>Spine</i> ,1993	Review of 19 cases of axis fracture including six cases of a pattern occurring through the posterior aspect of the vertebral body continuity of the posterior cortex with subluxation resulting in narrowing of the spinal canal.	III	Occurred in six of 19 patients including two with spinal cord injury from the associated subluxation.
Tan ES and Balachandran N, <i>Paraplegia</i> , 1992	Retrospective study of 33 patients with Hangman's fracture. Classified by Effendi: Type I – 21, Type II – 11 and Type III – 1.	III	20 / 26 had no neurologic deficit on admission. 28 / 33 with complete recovery after one year.
Torreman M, <i>Ned Tijdschr Geneeskde</i> , 1990	Long term study of 23 patients with Hangman's fractures treated with immobilization. Average follow-up 9.6 years.	III	100 % long-term fusion rate with cervical immobilization.

Author/ Year	Study Design	Class	Comments
Govendor S and Grootboom M, <i>Injury</i> , 1987	Prospective study of 39 patients.	III	All patients successfully managed with traction and immobilization.
Grady MS et al, <i>Neurosurg</i> , 1986	Retrospective review of 27 patients including 16 managed with halo, eight with a collar and three with bedrest.	III	All achieved fusion with no residual symptoms. The authors recommend the use of a Philadelphia collar alone in fractures with minimal displacement.
Levine AM and Edwards CC, <i>J Bone Joint Surg Am</i> , 1985	Retrospective case series of 52 patients with traumatic spondylolisthesis of the axis classified using the Effendi criteria.	III	Isolated Type I, II and IIa were all managed non-operatively. three of five Type III patients underwent surgical stabilization for failure to obtain or maintain reduction in a halo. The authors identify the Type IIa subgroup of the Effendi Type II patients who distract significantly with the application of traction and note the mechanism of injury for this group is likely flexion-distract. 3/3 Type IIa were treated with gentle extension and compression under fluoroscopic guidance followed with Halo immobilization.
Borne GM et al, <i>J Neurosurg</i> , 1984	Retrospective review of 18 cases of "pedicle" fracture of the axis treated with direct internal fixation.	III	Aggressive surgical approach for fixation of pedicle-isthmus fractures of the axis with 100 % fusion rate.
Mestdagh H et al, <i>Rev Clin Orthop Reparatrice Appar Mot</i> , 1984	Combined clinical and anatomic study describing 41 fractures of the posterior neural arch of the axis. Eleven cases treated with anterior C2-3 interbody fusion. 30 treated with traction and immobilization. Follow-up available on 30.	III	Cadaveric study demonstrated fractures with displacement of up to five mm were stable. Cervical mobility maintained better in the conservative management group. The authors recommend conservative measure except in cases of marked instability or non-union.
Francis WR et al, <i>J Bone Joint Surg Br</i> , 1981	Classification paper based on 123 cases of fractures of the posterior arch of the axis. Grade based on displacement and angulation.	III	Grade I (15% of total series) 0 % non-union with immobilization Grade II (7 %) 33 % non-union Grade III (37 %) 0 % non-union Grade IV (34 %) 2 % non-union Grade V (6 %) 28 % non-union
Bucholz RW, <i>Clin Orthop</i> , 1981	Autopsy study of 170 cases of traumatic death.	III	38 had cervical spine fractures and eight /38 had traumatic spondylolisthesis of the axis.
Pepin JW and Hawkins RJ, <i>Clin Orthop</i> 1981	Defined an early classification scheme for Hangman's fracture based on 42 cases.	III	Type I - an non-displaced fracture of the posterior elements Type II - displaced fracture involving posterior and anterior structures. All 42 patients were successfully treated non-surgically.

Author/ Year	Study Design	Class	Comments
Effendi B et al, <i>J Bone Joint Surg Br</i> , 1981	Classification paper based on 131 cases patients with fractures of the ring of the axis. Fractures divided into three groups based on mechanism of injury, displacement and stability.	III	<p>Type I (65 % of total group) Isolated hairline fractures of the ring of the axis with minimal displacement of the body of C2 caused by axial loading and hyperextension</p> <p>Type II (28 %) Displacement of anterior fragment with disruption of the disc space below the axis caused by hyperextension and rebound flexion</p> <p>Type III (7 %) Displacement of anterior fragment with C2-3 facet dislocation caused by primary flexion and rebound extension.</p> <p>Although five patients underwent surgery the authors conclude that the vast majority of these patients are best managed with cervical immobilization.</p>
Brashear R et al, <i>J Bone Joint Surg Am</i> , 1975	29 patients with Hangman's fractures followed for an average of six years.	III	No case of neurologic deficit. 23/23 (100%) treated with immobilization achieved fusion. Supports non-operative management.

### EVIDENTIARY TABLE three – Miscellaneous Axis Fractures

Author / Year	Study Design	Data Class	Comments
Greene KA et al, <i>Spine</i> 1997	340 cases axis fractures, including 67 non-odontoid, non-Hangman's fractures (miscellaneous ) most involving the body of lateral masses.	III	60 / 61 ( 98 % ) were successfully treated with external mobilization in all but one patient - (1.6 % non-fusion rate) Four died patients died and one underwent early surgery for five mm luxation C2 on C3.
FujimaraY et al, <i>J Orthop Trauma</i> , 1996	31 cases of axis body fractures categorized into four types based on radiographic imaging.	III	Four types: <u>Avulsion</u> : (9/9 fused with immobilization) <u>Transverse</u> : (2/2 healed with immobilization) <u>Burst</u> : (2/3 treated with C2-3 fusion) <u>Sagittal fractures</u> : (15/17 healed with immobilization). 8 sagittally oriented fracture patients still had pain despite a bony union.
Benzel EC et al, <i>J Neurosurg</i> , 1994	Retrospective report of 15 patients described with fractures of the axis body.	III	The authors propose classification into Type 1: (coronal, n = 12), Type 2: (sagittal, n = 3) Type 3: (oblique and equivalent to the Type III odontoid fracture).
Korres DS et al, <i>Eur Spine J</i> , 1994	14 cases of avulsion fracture of the anterior inferior portion of the axis secondary to an extension type injuries. Mean follow-up of 8.5 years.	III	3 % of the cervical spine trauma cases over a 12 year period. All patients treated successfully with cervical immobilization
Bohay D et al, <i>J Orthop Trauma</i> , 1992	Describes three cases of vertical fractures of the axis.	III	Notes this as an unusual variant fracture of the axis body. All treated with immobilization.
Craig JB and Hodgson BF, <i>Spine</i> , 1991	Describes nine cases of superior facet fracture of the axis vertebra	III	5 treated with reduction and immobilization. three required open reduction and posterior fusion.
Burke JT and Harris JH, <i>Skeletal Radiol</i> , 1989	Review of 165 patients with axis fractures. 31 miscellaneous body fractures identified and classified on mechanism of injury.	III	Identified 31 patients with axis body fractures. 21 / 38 (68 %) were extension teardrop and ten / 31 (32 %) were hyperextension.
Jakim I and Sweet MB, <i>J Bone Joint Surg Br</i> , 1988	Case report of a transverse fracture of the axis and literature review. A classification scheme is proposed.	III	Three types of axis body fractures were described: the Type III odontoid fracture of Anderson and De Alonzo, the transverse body fracture and the avulsion fracture.

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## MANAGEMENT OF COMBINATION FRACTURES OF THE ATLAS AND AXIS IN ADULTS

### RECOMMENDATIONS

Standards: There is insufficient evidence to support treatment standards.

Guidelines: There is insufficient evidence to support treatment guidelines.

Options: Treatment of atlas-axis combination fractures based primarily on the specific characteristics of the axis fracture is recommended. External immobilization of the majority of C1-C2 combination fractures is recommended. C1-Type II odontoid combination fractures with an ADI of five mm or greater and C1- Hangman's combination fractures with C2-C3 angulation of 11 degrees or greater should be considered for surgical stabilization and fusion. In some cases, the surgical technique must be modified as a result of the loss of the integrity of the ring of the atlas.

### RATIONALE

Combined fractures of the atlas and axis often present management challenges due to the unique anatomy and biomechanics of the atlantoaxial complex and the untoward stresses applied to the atlanto-axial region during trauma. While the majority of isolated atlas and axis fractures have been managed with cervical immobilization, the occurrence of the two fractures in combination often implies a more significant structural and mechanical injury. Although reports of combination C1-C2 fractures are relatively infrequent, sufficient evidence exists to allow a review of the management of a variety of combinations of atlas and axis fractures. The purpose of this report is to examine the available literature to determine successful treatment strategies for individual C1-C2 combination fracture types.

## **SEARCH CRITERIA**

A National Library of Medicine computerized literature search from 1966 to 2001 was undertaken using Medical Subject Headings in combination with “vertebral fracture”: “atlas”, “axis” and “human”. This strategy yielded 1,071 references. The abstracts were reviewed and articles focusing on clinical management and follow-up of combination fractures of the atlas and axis were selected for inclusion. The relative infrequency of these fractures, the small number of case series and the numerous case reports with pertinent information required rather broad inclusion criteria. Several papers addressing relevant biomechanics and radiology were included. The bibliographies of the selected papers were reviewed to provide additional references.

These efforts resulted in 48 manuscripts describing the clinical features and the management of acute traumatic atlas and axis combination fractures. They are summarized in Evidentiary Table format. No Class I or II evidence has been generated on the management of these fractures. Treatment options have been formulated based on Class III medical evidence.

## **SCIENTIFIC FOUNDATION**

### **Overview**

In 1920 Sir Geoffrey Jefferson reviewed 46 cases of atlas fractures.<sup>(30)</sup> While his paper is best known for the characterization of the C1 burst fracture or “Jefferson fracture”, Jefferson’s series included 19 fractures that were described as “combination fractures” of the atlas and the axis. He noted increased morbidity and mortality for combination injury patients. Eleven of the

19 patients he described with C1-C2 combination injuries suffered significant neurological injuries.

In 1986, Levine and Edwards reported their approach to the management of C1-C2 traumatic fracture injuries.(34) They suggested that if an atlas or axis injury was identified, that a careful search for other related injuries was indicated. They stressed that each patient and each injury needed to be evaluated independently. They described staged treatment for certain injuries to allow healing of one fracture (usually the atlas) before definitively managing the combination injury (typically the axis fracture). Several of their observations are worthy of consideration in the management of combination fracture injuries of the atlas and axis today.

### **Incidence**

Combination fractures of the C1-C2 complex are relatively common. In reports focusing primarily on odontoid fractures, the occurrence of a concurrent C1 fracture in the presence of a Type II or Type III odontoid fracture has been reported in 5% to 53% of cases.(12,23,25,26,28,36,39,40,43-45,47,49)

Odontoid fractures have been identified in 24% to 53% of patients with atlas fractures. (18,32,35,45) In the presence of a Hangman's fracture, the reported incidence of a C1 fracture ranges from 6% to 26%.(13,17,33,38,41,45)

Greene et al reported on 340 axis fractures and found 48 concurrent atlas fractures (combination injuries) for an incidence of 14%.(23) Ryan et al reviewed 717 spine fractures and found combination atlas-axis fractures in 15% of odontoid fractures and in 9% of Hangman's fractures. (45)

Gleizes et al reviewed 784 patients with proximal cervical spine injuries in 2000. (21) One hundred sixteen patients had injuries to C1 and/or C2. Thirty-one patients had C1 fractures in association with a C2 fracture (combination injury) representing 4% of the total c-spine fracture population, and 27% of all C1-C2 fracture injuries.

### **Morbidity and Mortality**

Various authors have suggested that the morbidity and mortality of C1-C2 combination fractures is greater than that associated with isolated fractures of either the atlas or the axis. (12,18,19,25,26,31,49) Fujimara et al observed neurological deficits in 82 of 247 patients (34%) with injuries to the C1-C2 complex.(19) Those patients suffering deficit had either burst fractures or fractures of the posterior arch of C1, or a fracture of the C2 body coupled with an odontoid or Hangman's fracture. Several authors have described a high mortality rate with combination fractures, in particular C1 fractures combined with Type II odontoid fractures.(18,25,26,49) Fowler et al found that six of seven patients (86%) with C1-Type II odontoid combination fractures died in the early treatment period.(18) Similarly, Hanssen et al observed that five of six patients (83%) with this same combination fracture pattern died within the first 40 days of injury.(26) Both Hanigan et al and Zavonne et al reported early deaths associated with C1-Type II odontoid fractures. (25,49)

In other reports on C1-C2 combination fractures, the description of morbidity and mortality has been less remarkable.(12,18) Dickman et al suggested a 12% incidence of neurological deficit for C1-C2 combination fractures, compared with a 0% (0 of 32) and a 2% incidence (two of 125) for isolated atlas and axis fractures, respectively.(12) Kesterson et al described four patients with C1-C2 combination fractures.(31) Only one patient had a

neurological deficit (25%). Irrespective of the author, the described incidence of morbidity and mortality associated with combination C1-C2 fractures appears to be more than that associated with isolated atlas and axis fractures.

## **Treatment**

Since the original description of C1-C2 combination fracture injuries by Jefferson, nearly every series reviewing either isolated fractures of the atlas or the axis includes some mention of C1-C2 combination fractures. It is difficult to determine the specific treatment provided to and outcome for most of those patients. Several authors have focused their reports specifically on combination C1-C2 fractures and their management.(12,21,24)

In 1989 Dickman et al identified 25 cases of acute atlas-axis combination fractures in an overall series of 860 patients with acute cervical fracture injuries.(12) In their experience, C1-C2 combination fractures represented 3% of their total cervical fracture population. Combination injuries represented 43% of acute atlas fractures (25 of 58) and 16% of acute axis fractures (25 of 150). The fractures of C1 and C2 were identified using plain film radiographs in 76% and 92% of the cases respectively. Computed tomography characterized the combination fracture patterns in all cases. Twelve percent of patients (three of 25) had neurological deficits upon admission. Two patients had acute central cord syndrome and one patient had a complete neurological injury. The etiology of the injury was a motor vehicle accident in 60% of cases and a fall in 28%. Four main types of atlas-axis fracture combination were identified: C1-Type II odontoid (ten cases, 40%), C1 miscellaneous axis fracture (seven cases, 28%), C1-Type III odontoid (five cases, 20%) and C1- Hangman's type fracture (three cases, 12%). The distribution of the atlas fractures was reported as multiple ring fractures in 40%, posterior ring

fracture in 28%, unilateral ring fracture in 24% and lateral mass fracture in 8%. Non-operative therapy was the initial management strategy in 20 of 25 of patients (84%). Eighteen were placed in a halo orthosis and two in a SOMI brace, for a median duration of 12 weeks (range 10–22 weeks). Four patients were treated with early surgical stabilization and fusion based on an atlanto-axial interval of six mm or greater. Three were treated with posterior C1-C2 wiring and fusion. Follow-up was accomplished in 23 of 25 patients (92%). Nineteen of the 20 patients (95%) treated either with a halo or SOMI orthosis achieved stability and fusion. One patient with an initial atlanto-axial interval of five mm, failed halo immobilization and was treated with posterior C1-C2 fusion. All patients treated surgically achieved stability utilizing a posterior fusion technique, four early and one delayed (100%). No patient deteriorated during or as a result of treatment. Six patients complained of persistent neck pain or limitation of neck motion. The authors offered a treatment algorithm based on the type and displacement of the axis fracture. They believe every patient with a C1 or C2 fracture should be studied with CT to rule out a combination injury. When present, atlas fractures in combination with Type II or III odontoid fractures with an atlantoaxial interval of five mm or greater should be considered for early surgical management. They stressed that the integrity of the C1 ring must be assessed to determine if C1-C2 wiring techniques can be utilized. Their perspectives were offered prior to the popularization of C1-C2 transarticular screw fixation techniques.

Guiot et al in 1999 described a series of ten patients with combination atlas-axis fractures treated with surgical stabilization and fusion.(24) Fifty percent (five of ten) of these patients

had failed halo immobilization and were referred specifically for operative intervention. Ninety percent were patients with C1-Type II odontoid fractures and the remaining patient had a C1-Type III odontoid combination fracture injury. One patient died in the follow-up period of unrelated causes. There were no other significant complications with a follow-up period of 28.5 months. All nine other patients accomplished successful fusion, utilizing an odontoid screw alone in five patients, an odontoid screw plus C2 pedicle screws in one, posterior transarticular screws in two, and anterior transarticular screws in one patient. The authors' indications for surgery included patients with fractures that could not be reduced or maintained with external immobilization, and unstable fractures with a high likelihood of non-union (including evidence of disruption of the transverse atlantal ligament).

### **Treatment of C1-Type II odontoid combination fractures**

The treatment of specific fracture combinations has been the subject of numerous reports. The C1-Type II odontoid fracture combination appears to be the most frequent and the subject of the most variability in treatment strategy. As noted with the management of isolated Type II odontoid fractures, optimal treatment remains controversial (see Management of Isolated Axis Fractures in Adults). Management techniques for C1-Type II odontoid combination fractures include semi-rigid immobilization (collar), traction followed by mobilization in a brace, rigid immobilization (halo, Minerva, SOMI), posterior fusion with and without instrumentation and anterior odontoid screw fixation.

While Esses et al describes a single cases of C1-Type II odontoid combination fracture managed successfully in a cervical collar,(16) the lower fusion rate described for Type II odontoid fractures managed in a collar alone should be considered when electing this treatment

option (see Management of Isolated Axis Fractures in Adults). Sherk and Nicholson described a single patient successfully treated with traction reduction followed by immobilization in a Minerva brace.(48) Segal et al treated two patients with traction followed by rigid immobilization.(46) A variety of authors have described the treatment of C1-Type II odontoid combination fractures with rigid immobilization (halo, SOMI, Minerva).(8,12,14,26,32,36,46) Dickman et al described five of six patients successfully treated in this way (83% success rate).(12) All six patients had an atlanto-axial interval of less than six mm. One patient with an atlantoaxial interval of 5mm failed halo immobilization and required posterior C1-C2 fusion at 12 weeks post-injury. Segal et al described three cases of C1-Type II odontoid combination fracture successfully treated with halo immobilization.(46) Andersson et al described two patients with this combination fracture injury over age 65 successfully treated with a halo device.(2) Seybold et al added two more patients treated with a halo resulting in successful union.(47) Additional single cases managed with halo immobilization have been described. (8,14,26,32,36,43)

The C1-Type II odontoid combination fracture has been successfully managed with surgical stabilization and fusion. Dickman et al treated four patients with C1-Type II odontoid combination fractures with early surgical fusion based on an atlanto-axial interval of six mm or greater.(12) Three patients had posterior C1-C2 fusion and one patient underwent occipital-cervical fusion for multiple fractures of the posterior atlantal arch. Andersson et al treated one patient with C1-Type II odontoid combination fracture with posterior C1-C2 fusion in a series of elderly patients.(2) Coyne et al also treated one patient with this injury pattern with a C1-C2 posterior fusion.(10) Several authors have suggested that the C1 arch fracture be allowed to heal prior to undertaking definitive atlantoaxial arthrodesis for this sub-type of combination fractures.

Others have suggested using onlay bone graft for C1-C2 fusion followed by halo immobilization in the setting of posterior C1 arch incompetence.(29,34,37) Lee et al described the surgical management of two patients with C1-Type II odontoid combination fractures in whom posterior C1-C2 fusion was performed.(32) Guiot et al described two patients with this combination injury pattern treated posteriorly with C1-C2 transarticular screw fixation and fusion.(24)

Multiple investigators have utilized anterior odontoid screw fixation in the treatment of C1-Type II odontoid combination fractures. Montesano et al described four cases in 1991 successfully managed in this fashion.(39) Berlemann and Schwazenbach published an additional four cases.(5) The report by Guiot et al included six patients in which odontoid screw fixation was accomplished.(24) These authors added anterior transarticular fixation in one patient. In 1999 Henry et al described a fusion success rate of 90% in ten patients with C1-Type II odontoid combination fractures treated with anterior odontoid screw fixation.(28) Apostolides et al described a single case in which three screws were placed all from an anterior trajectory to stabilize the C1-C2 (bilaterally) articulation and the odontoid fracture.(3)

Occipito-cervical fusion has been reported in the management of C1-Type II odontoid combination fractures.(1,2,12,27,31,42) It appears this approach is reserved for patients with disruption of the C1 arch and gross C1-C2 instability.

In summary, a variety of treatment options have been effective in C1 – Type II odontoid combination fractures. External orthoses have been successfully used in the management of the majority of these injuries. Combination fractures of this subtype with C1-C2 instability as defined by atlantal-dens interval of five mm or greater have a high failure rate with external immobilization alone and have been successfully managed with operative reduction, internal fixation, and fusion.

### **Treatment of C1- Type III odontoid combination fractures**

Dickman et al described five patients with C1-Type III odontoid combination fractures.(12) All were successfully treated with halo immobilization for an average of 12 weeks. Ekong et al identified two similar cases.(14) One was managed successfully in a halo. The second failed halo immobilization and required a delayed posterior C1-C2 fusion. Guiot et al reported a patient with a C1-Type III odontoid-Hangman's combination fracture they successfully treated with ventral odontoid screw fixation followed by posterior pedicle screw fixation and fusion.(24) It appears external immobilization is effective in the management of these injuries in the majority of patients.

### **Treatment of C1-Hangman's combination fractures**

Most reported combination injuries of the atlas and the posterior elements of the axis have been successfully managed with semi-rigid or rigid external immobilization (+/- initial traction).(7,9,12,15,32,46,49) Coric et al and Lee et al described the successful treatment of nine total patients with this combination fracture type with a cervical collar only.(9,32) Dickman et al reported three patients with C1-Hangman's combination fractures successfully treated with either a halo or SOMI immobilization device.(12) The reports of Elliot et al, Brashear et al, Segal et al, Govendor and Charles, and Zavanone et al each describe patients with similar injuries successfully treated with non-operative techniques.(7,15,22,46,49) As with an isolated unstable Hangman's fracture, surgical fixation may be an option. The report by Fielding et al included 15 patients with C1-Hangman's combination fractures.(17) These authors recommended that fractures with angulation between C2 and C3 of 11 degrees or greater be treated surgically.

These combination fractures with angulation greater than 11 degrees were associated with an 85 % non-union rate with non-operative management in their experience. This combination injury subtype appears to be managed effectively with external immobilization alone. Unstable injuries, as defined by C2-C3 angulation of 11 degrees or greater, may require surgical management.

### **Treatment of C1-Miscellaneous C2 body combination fractures**

Combination fractures of the atlas associated with miscellaneous axis body fractures have been treated with both rigid and non-rigid immobilization.(6,11,12,20,32,44) Dickman et al reported seven cases of this combination fracture subtype treated successfully with either a halo or SOMI brace.(12) The cases described by Fujimara et al, Lee et al, Craig et al and Bohay et al were all managed successfully with a cervical collar alone.(6,11,20,32) A single case described by Polin et al was treated with traction and subsequent halo immobilization.(44) Non-operative management of this combination injury subtype is effective.

### **SUMMARY**

Combination fractures involving fractures of both the atlas and axis occur relatively frequently. A higher incidence of neurological deficit is associated with C1-C2 combination fractures compared to either C1 or C2 fractures in isolation. The C1-Type II odontoid combination fracture appears to be the most common combination injury subtype, followed by C1-miscellaneous axis, C1-Type III odontoid and C1-Hangman's combination fractures.

No Class I or Class II evidence addressing the management of patients with combination atlas and axis fractures is available. All of the articles reviewed describe case series or case

reports containing Class III evidence supporting a variety of treatment strategies for these unique fracture injuries.

In most circumstances, the specifics of the axis fracture will dictate the most appropriate management of the combination fracture injury. As reported for isolated atlas and axis fractures, the majority of atlas-axis combination fractures can be effectively treated with rigid external immobilization. Combination atlas-axis fractures with an atlanto-axial interval of five mm or greater or angulation of C2-C3 of 11 degrees or greater may be considered for surgical fixation and fusion. The integrity of the ring of the atlas must often be taken into account when planning a specific surgical strategy utilizing instrumentation and fusion techniques. If the posterior arch of C1 is inadequate, both incorporation of the occiput into the fusion construct (occipitocervical fusion) and posterior C1-C2 transarticular screw fixation and fusion have been successful.

**Table I:**

<b>Combination Fracture Type</b>	<b>Treatment Options</b>
C1-Type II odontoid fracture Stable Unstable (ADI = five mm or greater)	Collar, Halo, surgical fixation/fusion Halo, Surgical fixation/fusion
C1-Type III odontoid fracture	Halo
C1-miscellaneous axis	Collar, Halo
C1-Hangman's fracture Stable Unstable (C2-C3 angulation 11 degrees or greater)	Collar, Halo Halo, Surgical fixation/fusion

## **KEY ISSUES FOR FUTURE INVESTIGATION**

The identification of which of the atlas-axis combination fracture sub-types are at greatest risk for non-union and subsequent instability would be useful in determining appropriate management for combination fracture injuries. A uniform and clinically useful definition of cranial, C1, and C2 instability in association with these fractures would be of benefit. Prospective data collection and case-control studies at multiple institutions would provide meaningful data addressing these issues. The relative infrequency of combined atlas-axis fractures would make a randomized study difficult. Patients with a C1-Type II odontoid combination fracture should be studied comparing operative and non-operative management and should be evaluated in terms of management morbidity, long-term success, economic benefit, patient satisfaction and return to pre-injury activities.

## VII. EVIDENTIARY TABLES

First Author Reference	Description of Study	Data Class	Conclusions
Andersson S et al, 2000, <i>J Bone Joint Surg</i>	Case series of patients over 65 with odontoid fractures	Class III	Includes three patients with C1-Type II odontoid fractures. Treatment: Halo – 2 Posterior cervical fusion – 1
Gleizes V et al, 2000, <i>Eur Spine J</i>	Retrospective epidemiological review of coincidence of fractures in the upper cervical spine	Class III	784 cervical spine injuries 116 upper cervical spine injuries (C1-C2) (15%) 31/116 (26 %) combination of C1 and C2  70 % of all atlas fractures occurred in combination with another fracture  30 % of all Hangman's and odontoid fractures occurred in combination with another fracture  41.9 % of patients with combination fractures of the upper cervical spine underwent surgical fixation versus 21.7 % of those with isolated injuries
Muller EJ et al, 2000, <i>Eur Spine J</i>	Case series of 39 cases of Hangman's fractures.	Class III	Included two with C1 ring fractures (5.1 %)
Guiot B and Fessler R, 1999, <i>J Neurosurg</i>	Retrospective review of ten patients undergoing surgical fixation for combination C1-C2 fractures  5/10 referred specifically for surgical fixation after failed external immobilization  Average followup 28.5 months	Class III	Type: C1-Type II odontoid – nine (90%) C1-Type III odontoid and Hangman's – one (10%)  Technique: Odontoid screw – six (60 %) Odontoid screw plus C2 pedicle screws one (10 %) C1-C2 Transarticular screws (posterior) two (20%) C1-C2 Transarticular screws (anterior) one (10%)  Outcome: One unrelated death All others fused successfully without other complication.
Henry AD et al, 1999, <i>J Bone Joint Surg</i>	Case series of 61 cases of Type II odontoid fractures treated with anterior screw fixation in which followup was available.	Class III	Includes ten combination fractures of C1-C2 (16 %)  C1 burst (Jefferson) – Type II odontoid – three (5 %) C1 anterior arch – Type II odontoid – three (5 %) C1 posterior arch – Type II odontoid – four (6 %)  All patients in the series were treated with anterior odontoid screw fixation with a 92 % success rate.  No problems attributed directly to the presence of the atlas fracture.
Morandi X et al, 1999, <i>Surg Neurol</i>	Case series including 17 odontoid fractures treated with anterior screw fixation.	Class III	Included two cases of C1-posterior arch fracture plus a posteriorly displaced Type II odontoid.

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
Lee TT et al, 1998, <i>Spine</i>	Retrospective review of 16 cases of atlas fracture	Class III	Includes eight patients with combination C1 and C2 fractures C1-Type II odontoid – two cases One treated with halo immobilization Two treated with posterior C1-C2 fusion  C1-Hangman’s – two cases Both treated with cervical collar  C1-C2 body fracture – three cases All three treated with cervical collar  Authors conclude that the management of the combination fracture should be based on the C2 fracture and that halo immobilization is not always required
Seybold EA and Bayley JC, 1998, <i>Spine</i>	Case series of 57 odontoid fractures.	Class III	Includes three cases of C1 ring fracture plus Type II odontoid. The authors successfully managed two patients with a halo. One patient treated in a collar with a “poor” result. The overall fusion rate for the Type II odontoid fractures in this series was 65 %. No specific effect from the C1 fracture was noted.
Apostolides P et al, 1997, <i>J Neurosurgery</i>	Case report	Class III	Patient with anterior ring of C1 fracture and a Type II odontoid failed halo immobilization  Treated successfully with anterior C1-C2 transarticular fixation and an odontoid screw
Berlemann U and Schwarzenbach O, 1997, <i>Acta Orthop Scand</i>	Retrospective review of 19 patients over age 65 with odontoid fractures	Class III	Includes four patients with C1 fractures and Type II odontoid fractures all treated with anterior odontoid screw fixation
Greene KA et al, 1997, <i>Spine</i>	Large review of 340 axis fractures.	Class III	48 patients with an axis fracture also had an atlas fracture (14 %). Specifics on management are not presented but the authors indicate that the management in these cases was based on the C2 fracture.
Castillo M and Mukherji SK, 1996, <i>Am J Neuroradiol</i>	Case report	Class III	Includes one case of Jefferson fracture plus Type II odontoid treated with halo
Coric D et al, 1996, <i>J Neurosurg</i>	Case series of 57 patients with Hangman’s fractures.	Class III	Includes seven cases of combination fracture (C1 –Hangman’s). All were treated based on degree of displacement. If less than six mm, were treated with non-rigid immobilization.
Fujimara Y et al, 1996, <i>Paraplegia</i>	Case series of axis body fractures	Class III	Describes three patients with C1- miscellaneous body fracture all treated with cervical immobilization. Authors recommend non-operative treatment except in cases of severe angulation. Philadelphia collar used if minimal angulation.
Polin RS et al, 1996, <i>Neurosurgery</i>	Case series of 62 patients with odontoid fractures.	Class III	Includes five cases of combination C1-C2 fracture (8 %)  C1-Jefferson – Type II odontoid - four C1-Misc C2 body fracture – 1  All patients in series managed with either halo or collar.
Coyne TJ et al, 1995, <i>Spine</i>	Retrospective review of 32 patients with odontoid fractures includes one combination fracture	Class III	One case of Jefferson- Type II odontoid treated with Gallie fusion

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
Fujimara Y et al, 1995, <i>Paraplegia</i>	Retrospective review of 247 admissions with upper cervical spine fractures - focuses on 82 patients with neurological deficit	Class III	In patients with combined injury of C1 and two neurological deficit occurred in patients with posterior arch fracture, burst fracture of the atlas or body fracture of the axis associated with either an odontoid fracture or Hangman's
Benzel E et al, 1994, <i>J Neurosurg</i>	Case series	Class III	Includes one case of C1- vertically oriented C2 miscellaneous body fracture (treatment not described).  The author discusses the possible mechanism included hyperextension and axial loading.
Pederson AK and Kostuik JP, 1994, <i>J Spinal Disord</i>	Case report of 70 year old man with fracture dislocation of C1-C2 with 20 mm atlantoaxial displacement	Class III	Successfully treated with O-C4 decompression and posterior fusion with complete recovery.
Hanigan WC et al, 1993, <i>J Neurosurg</i>	Case series of 19 patients over 80 years of age with odontoid fractures.	Class III	Included two patients with a C1-Jefferson – Type II odontoid.  One died in the hospital after being placed in traction. One with a stable fibrous non-union following treatment in a halo.
Bohay D et al, 1992, <i>J Orthop Trauma</i>	Case series	Class III	Includes a case of a C1 burst fracture plus a vertical C2 body fracture successfully treated in a cervical collar alone.
Hays MB and Bernhang AM, 1992, <i>Spine</i>	Case series of unusual fractures of the atlas	Class III	Includes two cases of combination fractures.  C1 (anterior arch)-Type II odontoid fracture failed halo treatment resulting in an O-C2 fusion.
Jeanneret B and Magerl F, 1992, <i>J Spinal Disord</i>	Case series of 59 patients with odontoid fractures 30 of which were treated surgically.	Class III	Includes two cases in which the posterior arch of C1 was not intact.  C1-Jefferson – Type II odontoid - 1 C1 – posterior arch – Type III odontoid - 1  Authors feel strongly that if the posterior arch of C1 is not intact that C1-C2 transarticular fixation is indicated.  In the comment following the article, the point is made that an onlay graft between C1 and C2 posteriorly without wiring of C1 followed by halo immobilization has been used in this situation.
Ryan MD and Henderson JJ, 1992, <i>Injury</i>	Epidemiological report of 717 spine fractures	Class III	Atlas fractures occurred with odontoid fractures (53%) and with Hangman's (24 %)  Odontoid fractures occurred with atlas fractures (15%)  Hangman's fracture occurred with atlas fracture (9%)
Craig JB and Hodgson BF, 1991, <i>Spine</i>	Case report	Class III	Jefferson plus superior facet of axis treated with collar.

First Author Reference	Description of Study	Data Class	Conclusions
Esses SI and Bednar DA, 1991, <i>Spine</i>	Case report - atlas and odontoid fracture	Class III	Jefferson plus type II odontoid in a 34-year-old male - treated successfully with collar only (seen after a one month delay in diagnosis)
Kesterson L et al, 1991, <i>J Neurosurg</i>	Case series, retrospective review	Class III	Includes four patients with combination fracture of the atlas and type II odontoid treated with O-C2 fusion  One of these four patients had a significant neurological deficit (25%)  The authors suggest surgery if unstable and define instability as atlantoaxial interval of greater than five mm or lateral mass displacement greater than seven mm
Levine AM and Edwards CC 1991, <i>J Bone Joint Surg</i>	Case series of 34 patients with atlas fractures.	Class III	Includes 15 patients with a combination C1-C2 fracture (44%)  C1- TII or TIII odontoid – eight (24 %) C1- Hangman's – seven (21%)  Describes two cases in the C1-odontoid fracture group in which the posterior C1 arch altered the treatment plan. In one case a Gallie fusion failed and the second no wiring was used, just onlay bone graft.
Montesano PX et al, 1991, <i>Spine</i>	Case series of 14 Type II odontoid fractures treated with anterior odontoid screw fixation. Follow-up 24 months.	Class III	Seven had a C1 fracture (50%)  The overall fusion rate was 93 %.  No problems attributed directly to the C1 fracture.
Zavonne M et al, 1991, <i>J Neurosurg Sci</i>	Case series of 23 C1-C2 fractures.	Class III	Included two combination fracture (9%)  C1-Type II odontoid – patient died C1- Hangman's – treated successfully with traction reduction and Minerva
Fowler JL et al, 1990, <i>J Spinal Disord</i>	Case series of 48 atlas fractures from series of 867 C-spine fractures (5.5 %).	Class III	Included 18 cases with a combination C1-C2 fracture (38 % of total series)  C1 burst (Jefferson) –Type II odontoid – six (33 %) C1 burst (Jefferson) -Type III odontoid – one (6 %) C1 burst (Jefferson) - Miscellaneous axis – two (11 %) C1 burst (Jefferson) – Hangman's – 0 (0 %)  C1 arch –Type II odontoid – eight (44 %) C1 arch -Type III odontoid – one (6%) C1 arch - Miscellaneous axis – one (6 %) C1 arch – Hangman's – three (16 %)  These authors present data supporting the increased mortality associated with combination C1-C2 fractures. Six of the seven early deaths (86 %) had a C1 fracture associated with either a Type II or Type III odontoid fracture.

First Author Reference	Description of Study	Data Class	Conclusions
Dickman C et al, 1989, <i>J Neurosurg</i>	Retrospective review of 25 patients with fractures of both C1 and C2  Comprises three % of the overall cervical spine injury cohort (25 of 860)	Class III	Four Types Noted: C1 –Type II odontoid – ten (40 %) C1- Miscellaneous axis – seven (28 %) C1-Type III odontoid – five (20%) C1 – Hangman’s – three (12 %)  Neurological deficit in three / 25 (12 %) Treatment determined by type of C2 fracture  Non-operative (84 %) Halo – 18 SOMI – 2 One of the C1-Type II patients failed halo and required C1-C2 fusion  Operative (initial management) C1-Type II odontoid with six mm displacement – three patients treated with posterior C1-C2 fusion – one patient treated with O-C2 fusion because of multiple fractures in C1
Fielding JW et al, 1989, <i>Clin Orthop</i>	Case series of 123 Hangman’s fractures	Class III	Included: C1 arch – Hangman’s – ten cases (8 %) C1 burst (Jefferson) – Hangman’s – two (2%) C1 lateral mass – Hangman’s – three (3%)  Specifics not given for each subtype but overall the authors recommend treatment based on the C2 fracture despite the presence of the C1 fracture. Regardless of the C1 fracture, the authors favored an anterior C2-3 fusion for those patients with angulation greater than 11 degrees as this group had an 85% non-union rate with cervical immobilization.
Govendor S and Charles RW, 1987, <i>Injury</i>	Cases series of upper cervical fractures.	Class III	Includes two cases of combination C1 posterior arch fracture – Hangman’s fracture – treated successfully with a cervical collar (non-rigid cervical immobilization)
Hanssen AD and Cabanela ME, 1987, <i>J Trauma</i>	Case series of 42 odontoid fractures.	Class III	Includes seven combination fractures (17 %).  C1- Jefferson – Type II odontoid – 6 five of six (83 %) died within first 40 days 1 of six developed a stable non-union  C1- posterior arch – Type II odontoid – 1 Healed with halo immobilization
Lind B et al, 1987, <i>Spine</i>	Case series of 14 odontoid fractures managed in a halo orthoses.	Class III	Includes one case of C1-Jefferson- Type II odontoid managed in a halo vest for 12 weeks.
Mirvis SE et al, 1987, <i>Radiology</i>	Radiographic review 27 C2 fractures.	Class III	Noted nine associated C1 fractures (26 %).
Segal LS et al, 1987, <i>J Bone Joint Surg</i>	Case series of 18 patients with atlas fractures.	Class III	Six cases were combination C1-C2 fractures. C1-Jefferson – odontoid fracture - five cases 3 treated with halo, two with traction followed by halo C1-Jefferson – Hangman’s – one case treated with a collar

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
Levine AM and Edwards CC, 1986, <i>Orthop Clin North Am</i>	Review article on management of C1-C2 trauma.	Class III	<p>Comments on combined injuries:</p> <ol style="list-style-type: none"> <li>1. The presence of three injuries to the C1-C2 complex associated with a high likelihood of neurological injury</li> <li>2. If find one injury or fracture, look carefully for another</li> <li>3. Mechanism of injury usually consistent with the injury observed</li> <li>4. Each injury needs to be evaluated individually, for example the presence of two fractures does not always indicate instability (posterior arch of C1 plus a non-displaced Hangman's fracture)</li> <li>5. Staging of treatment may be required (as described by Lipson et al below) with allowing one fracture to heal before treating definitively</li> </ol>
Levine AM and Edwards CC, 1985, <i>J Bone Joint Surg</i>	Case series of 53 patients with Hangman's fracture. Describes stable (Type I Hangman's) and unstable (Type II Hangman's) groups.	Class III	<p>Included nine cases of Type I Hangman's (stable) plus C2 fracture:  Type II odontoid – 2  Type III odontoid – 3  Posterior arch – 1  Burst (Jefferson) – 2  Lateral mass – one</p> <p>Only one case Type II Hangman's (unstable) with C2 fracture  Posterior arch – 1</p> <p>Only one case treated surgically Type I Hangman's plus Type II odontoid treated with posterior C1-C2 fusion.</p>
Pepin JW and Hawkins RJ, 1985, <i>Clin Orthop</i>	Case series of 41 odontoid fractures	Class III	<p>Includes nine cases of odontoid fracture in combination with another spinal fracture of which the C1-Jefferson – Type II odontoid was the most common</p> <p>All treated with either C1-C2 fusion or halo.</p> <p>Author recommends fusion in the elderly.</p>
Effendi B et al, 1981, <i>J Bone Joint Surg</i>	Case series of 131 Hangman's fracture with classification	Class III	<p>Includes combination fractures:  C1 posterior arch – Hangman's – eight patients (8/131, six %)  Odontoid fracture – Hangman's – two patients (2/131, two %)  Specific outcomes not presented but all fused with either anterior or posterior C1-C2 fusion or halo –</p> <p>Overall mortality was nine %</p>
Ekong CE et al, 1981, <i>Neurosurgery</i>	Case series of 22 patients with odontoid fractures	Class III	<p>Included:  C1- Jefferson -Type II odontoid — 1  C1-Jefferson -Type III odontoid — 2</p> <p>All treated with halo with one of the C1 Jefferson -Type III odontoid patients failing and requiring C1-C2 posterior fusion</p>

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
Lipson SJ, 1977, <i>J Bone Joint Surg</i>	Case series of three cases of atlas fracture plus Type II odontoid	Class III	The authors recommend combination therapy of halo immobilization from ten to 12 weeks until the posterior arch of the atlas fracture has healed, followed by atlanto-axial fusion (Gallie-type) to definitively manage the odontoid fracture.
Brashear R et al, 1975, <i>J Bone Joint Surg</i>	Case series of Hangman's fracture	Class III	Included two patients with C1 posterior arch fracture plus Hangman's treated with reduction and Minerva for 3-6 months.
Anderson LD and D' Alonzo RT, 1974, <i>J Bone Joint Surg</i>	Case series of odontoid fractures	Class III	Included one patient with combined C1-Type II odontoid fracture treated with O-C2 fusion
Elliot JM et al, 1972, <i>Radiology</i>	Case series	Class III	C1 posterior arch – Hangman's – two cases treated with immobilization
Sherk HH and Nicholson JT, 1970, <i>J Bone Joint Surg</i>	Case report	Class III	One case each of a combination C1- TII odontoid and a C1- Hangman's - both were treated with immobilization (reduction in traction followed by a Minerva brace) successfully.

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## OS ODONTOIDEUM

### RECOMMENDATIONS

#### **Diagnosis:**

Standards: There is insufficient evidence to support diagnostic standards.

Guidelines: There is insufficient evidence to support diagnostic guidelines.

Options: Plain radiographs of the cervical spine (A-P, open mouth odontoid, and lateral) and plain dynamic lateral radiographs performed in flexion and extension are recommended. Tomography (computerized or plain) and/or MR of the craniocervical junction may be considered.

#### **Management:**

Standards: There is insufficient evidence to support treatment standards.

Guidelines: There is insufficient evidence to support treatment guidelines.

Options:

- Patients with os odontoideum without symptoms or neurological signs may be managed with clinical and radiographic surveillance.
- Patients with os odontoideum, particularly with neurological symptoms and/or signs, and C1-2 instability may be managed with posterior C1-2 internal fixation and fusion.
- Postoperative halo immobilization as an adjunct to posterior internal fixation and fusion is recommended unless successful C1-C2 transarticular screw fixation and fusion can be accomplished.
- Occipital-cervical fusion with or without C1 laminectomy may be considered in patients with os odontoideum who have irreducible cervicomedullary compression and/or evidence of associated occipital-atlantal instability.

- Transoral decompression may be considered in patients with os odontoideum who have irreducible ventral cervicomedullary compression.

## **RATIONALE**

The definition of an os odontoideum (os) is uniform throughout the literature: an ossicle with smooth circumferential cortical margins representing the odontoid process that has no osseous continuity with the body of C2 (16,22). The etiology of os odontoideum remains debated in the literature with evidence for both acquired and congenital causes (18,23,25). The etiology of os, however, does not play an important role in its diagnosis or subsequent management.

## **Diagnosis**

Os odontoideum can present with a wide range of clinical symptoms and signs, as well as be an incidental finding on imaging. The literature has focused on three groups of patients with os odontoideum, 1) those with occipital-cervical pain alone, 2) those with myelopathy, and 3) those with intracranial symptoms or signs from vertebrobasilar ischemia (4). Patients with os odontoideum *and* myelopathy have been subcategorized further into those with: 1) transient myelopathy (commonly following trauma), 2) static myelopathy, and 3) progressive myelopathy. (10) Because patients with occipital-cervical pain, myelopathy, or vertebrobasilar ischemia likely will have etiologies other than os, the diagnosis of os odontoideum is not usually considered until imaging is obtained. The presence of an os is usually first suggested after obtaining plain cervical spine radiographs. Most often plain cervical spine radiographs are sufficient to obtain a diagnosis.(15)

Os odontoideum has been classified into two anatomic types, orthotopic and dystopic. Orthotopic defines an ossicle that moves with the anterior arch of C1, while dystopic defines an ossicle that is functionally fused to the basion. The dystopic os may sublux anterior to the arch of C1.(10) Tomograms and computerized tomography have been used to better define the bony anatomy of the os and the odontoid process. Plain dynamic radiographs in flexion and extension have been used to depict the degree of abnormal motion between C1 and C2. Most often there is anterior instability with the os subluxing forward in relation to the body of C2. However, at times one will see either no discernible instability, or “posterior instability” with the os moving posteriorly into the spinal canal during neck extension.(10,20)

With respect to diagnosis, the issues regarding the imaging of os odontoideum are two: First, while plain radiographs are often diagnostic for os, the sensitivity and specificity of plain cervical radiographs for os odontoideum have not been reported. The utility of confirmatory studies such as computerized and plain tomography and MR has not been well defined. Second, following the diagnosis of os odontoideum on plain cervical x-rays, instability and osseous anomalies associated with os can influence clinical management. The best methods of further evaluating or excluding these complicating factors deserve definition.

## **Management**

The natural history of untreated os covers a wide spectrum. The literature provides many examples of both asymptomatic and symptomatic patients with known os odontoideum who have never been treated, and who have had no reported new problems in follow-up over many years. (22) Conversely, examples of sudden spinal cord injury in association with os following minor trauma have also been reported.(17) The natural history of os odontoideum is variable, and

predictive factors for deterioration, particularly in the asymptomatic patient have not been identified. Indications for surgical stabilization include: simply the existence of an os, os in association with occipital cervical pain alone and/or os in association with neurological deficit. (10,22) Other factors that may assist in determining the need for stabilization and/or decompression include C1-2 instability, associated deformities, and spinal cord compression. A variety of techniques have been used to stabilize C1 and C2 in patients with os odontoideum. (2,3,5,6,10,20,22,26,27) Fusion success rates and complication rates for these various procedures may provide evidence as to whether a preferred method of C1-2 arthrodesis is supported by the literature.

Finally, neural compression is an important consideration in patients with os odontoideum. Neural compression may be anterior from a combination of bone and soft tissue, or posterior from the dorsal arch of C1. Surgical techniques to stabilize and fuse across the craniocervical junction with or without C1 laminectomy, and techniques that provide ventral decompression have been reported in the treatment of os odontoideum with irreducible neural compression.(6,24) The literature will be examined in light of the risks and benefits these techniques may provide to patients with os odontoideum.

## **SEARCH CRITERIA**

A National Library of Medicine computerized literature search from 1966 to 2001 was performed through MEDLINE using the key phrase “os odontoideum”. The search identified 121 articles. Articles written in English were reviewed. Twenty-seven articles that described the clinical aspects and management of patients with os odontoideum were identified and used to generate these guidelines. Not one of the articles meeting selection criteria represented Class I or

Class II studies. All 27 provided Class III evidence regarding the diagnosis and/or management of os odontoideum. These 27 articles represent the basis for this review and are summarized in Evidentiary Table format.

## **SCIENTIFIC FOUNDATION**

### *Diagnostic Evaluation*

There is no literature that describes the sensitivity and specificity of imaging studies for os odontoideum. Dai et al, in their review of 44 patients with os used tomography, CT, and MR, in addition to “routine” plain cervical radiographs (AP, lateral, open-mouth, flexion and extension x-rays) in 39, 27, and 22 patients, respectively.(6) Matsui et al, described only the plain radiographs of 12 patients with os.(16) They excluded patients with Down’s syndrome and Klippel-Feil anomalies. The authors made no mention of any other studies to obtain or confirm the diagnosis in these 12 patients. Likewise, Watanabe et al, (27) and Spierings and Braakman (22) described the plain radiographs of 34, and 37 patients respectively with os, without reference to other imaging studies. Fielding et al, described 35 patients with os odontoideum in which “Each patient had extensive roentgenographic investigation, including multiple roentgenograms of the cervical spine and *often* flexion-extension lateral roentgenograms and flexion-extension laminagrams.” No mention was made as to whether additional studies beyond static plain c-spine x-rays were necessary to confirm the diagnosis of os odontoideum in their series of patients.(10)

The literature supports the ability of plain cervical spine radiographs to establish the diagnosis of os odontoideum. There is no compelling evidence in the literature that supports the need of additional studies to confirm the diagnosis of os.

Specific characteristics or associated abnormalities of os odontoideum, including C1-C2 instability, soft tissue masses, spinal canal diameter, associated osseous anomalies, spinal cord appearance, and vertebral artery compromise have been investigated with a variety of imaging studies. The imaging of abnormal motion and spinal cord compression in association with os odontoideum has received the most attention in the reported clinical series.

Instability of C1-2 in association with os odontoideum has been investigated with multiple imaging modalities. Employing flexion and extension lateral cervical spine x-ray studies in 33 patients, Fielding et al, reported 22 patients (67%) with anterior instability who had a mean atlanto-dens interval (ADI) of 10.3 mm, five patients (15%) with posterior instability (mean posterior translation of the os during extension of 8.4 mm), three patients (9%) had less than three mm of C1-2 motion, and three patients (9%) with no detectable C1-C2 motion.(10) Eight patients (23%) had both anterior and posterior instability. The authors noted that cineradiography was helpful in examining range of motion at C1-C2 in these patients, but it was not of benefit in the measurement of the degree instability. Of note is that almost one fifth of the patients in their series manifested no radiographic evidence of C1-2 instability.

Spierings and Braakman studied 21 of their 37 patients with os odontoideum with flexion and extension cervical spine radiographs or tomograms. They measured the maximal distance the os moved in the sagittal plane, the inner diameter of the atlas, and the minimal spinal canal diameter (the distance between the posterior aspect of the C2 body and the dorsal arch of C1 during flexion). They compared these measurements in two groups, those with and without myelopathy.(22) The degree of C1-C2 instability did not correlate with neurological status, but the measured minimal spinal canal diameter was significantly smaller ( $P < 0.05$ ) in the group with myelopathy. They identified 13 millimeters as the critical anterior-posterior (AP) spinal

diameter. Watanabe et al, made similar measurements in 34 patients using plain lateral cervical radiographs in flexion and extension.(27) Like Spierings and Braakman, the degree of instability in their patients did not correlate with the presence of myelopathy. Shirasaki et al, described radiographic findings on lateral flexion and extension radiographs in nine patients with os odontoideum.(20) They reported that a distance of 13 millimeters or less between the os and the dorsal arch of C1 “specifically defined severe cervical myelopathy” in their patients. They too, found that the degree of C1-C2 instability did not correlate with the presence of myelopathy. Yamashita et al, studied atlantoaxial subluxation with plain radiography and MR, and correlated the imaging studies with the degree of myelopathy in 29 patients (four with os odontoideum). They found that the degree of myelopathy did not correlate with the distance of subluxation of C1 on C2 on plain radiographs.(28) The degree of cord compression on MR did correlate well with the degree of myelopathy measured clinically. Matsui et al, classified os odontoideum into three types according to the shape of the os on plain radiographs.(16) Three types were described: round, cone, and blunt-tooth. They compared these three os types to the degree of clinical myelopathy and found the degree of myelopathy correlated most closely with the “round” os type. Kuhns et al, described the MR appearance of os odontoideum in four children and identified signal changes within the posterior ligaments consistent with trauma.(14) They could not discern whether these changes represented a primary or secondary phenomenon with respect to atlanto-axial instability.

These studies provide two consistent conclusions: 1) the degree of C1-C2 instability does not appear to correlate with neurological status in patients with os odontoideum; and 2) sagittal spinal canal diameter on plain radiographs of 13 millimeters or less is strongly associated with myelopathy.

Beyond plain spine radiographs and flexion-extension x-rays, imaging to assist with operative planning of unstable os odontoideum receives brief mention in several reports.(11,17,24,26) Important factors to consider before proceeding with surgical intervention for this disorder are: the ability to reduce C1-C2, spinal cord compression, an assimilated atlas, an incomplete C1 ring, the course of the vertebral arteries at C1 and C2, and the presence of an associated congenital fusion of the cervical spine (e.g. Klippel-Feil). Plain radiographs, tomography, and CT scans provide information regarding the ability to achieve anatomic alignment of C1 on C2, and the presence or absence of a congenital fusion. MR is the best modality for viewing cord compression even after apparent C1-C2 realignment.(28) CT can provide important information about the bony anatomy at the craniocervical junction including the completeness of the atlas ring and the position of the transverse foramina at C1 and C2.(19) Hosono et al, made interesting observations on the different motions of the posterior arch of C1 in relation to C2 in patients with os odontoideum. They observed two patterns of motion, linear and sigmoid. They felt that in those patients with a sigmoid shaped motion pattern posterior wiring techniques may not provide adequate stability.(13) The selection of and necessity for additional imaging studies in the evaluation of os odontoideum appears to be made on a patient-by-patient basis. The literature provides no convincing evidence as to which patients should undergo supplemental imaging (tomography or MR) after the diagnosis of os odontoideum has been made.

### Management

The universal theme of the various management strategies offered in the treatment of patients with os odontoideum has been either confirming or securing cervical spinal stability at

the C1-2 levels. The earliest reports of os odontoideum describe small pediatric case series treated surgically. In 1978, Griswold et al, described four children with os odontoideum that underwent posterior C1-2 wiring and autologous iliac fusion (12). Three children had successful arthrodesis. The fourth child did not achieve fusion/stability despite three attempts. In the same year, Brooks and Jenkins described their technique of C1-2 wiring and fusion and reported three children with os who were immobilized postoperatively in Minerva jackets.(3) All three patients achieved successful fusion. In summary, six of the seven children with os odontoideum described in these two reports were successfully treated.

Two larger series, reported in the early 1980's, included adults and children with os odontoideum, and described both operative and nonoperative management strategies for these patients. Fielding et al, described 35 patients with os odontoideum, of which 27 had radiographic evidence of instability.(10) Twenty-six of these 27 patients underwent successful posterior C1-C2 fusion (Gallie type). Fusions were noted to be "solid" after two months of immobilization in children and three months in adults. One patient with instability refused surgery and remained well at two years follow-up. The eight remaining patients with no evidence of C1-C2 instability managed non-operatively remained well at last follow up of one to three years. Spierings and Braakman described 37 patients they managed with os. Seventeen were treated surgically.(22) They provide 20 patients for analysis of the natural history of os odontoideum. Information about radiographic stability was provided for only 21 of the 37 patients they reported. Sixteen patients in their series presented with neck pain only or had an incidentally discovered os. Nine of these 16 patients had flexion and extension radiographs. Of these nine patients, seven had abnormal motion of eight millimeters or greater. With a median follow-up of seven years none of these 16 patients developed a neurological deficit. Four

additional patients who presented with myelopathy were treated non-operatively with follow-up from six months to 14 years. Three of these four patients presented with transient myelopathy and had no recurrence at last follow-up, despite abnormal motion of C1 on C2 of eight mm to 16 mm. The fourth patient had a stable monoparesis at last follow-up. Of the 17 patients who underwent surgery, one patient had neurological worsening and two died. Eight of these 17 patients treated surgically had a posterior C1-2 fusion. Nine patients underwent occipital-cervical fusion with C1 laminectomy. The authors did not report a single failed fusion. They had a combined surgical morbidity and mortality of 18% (three of 17 patients). The authors conclude that patients with os odontoideum without C1-C2 instability can be managed without surgical stabilization and fusion with good result. While they did not provide operative treatment to every os patient with C1-C2 instability, those with myelopathy and greater amounts of instability were more likely to be operated upon. If these two series are considered representative of patients with os odontoideum, the implication is that minimally symptomatic or asymptomatic patients with os odontoideum without C1-C2 instability can be managed non-operatively with little or no morbidity over time. While patients with os odontoideum and myelopathy or C1-C2 instability have been managed conservatively, most patients with myelopathy or instability are treated surgically.

Clements et al, in 1995 reported a patient who had a documented os without instability who at five years follow-up developed symptomatic frank C1-2 instability which required surgical stabilization and fusion.(4) It appears that a lack of C1-C2 instability at initial diagnosis does not guarantee that instability will not develop in these patients. It is recommended,

therefore, that clinical and radiographic follow-up be provided to patients with os odontoideum who are found to have radiographic C1-C2 stability on initial assessment.

More recent series reported in the literature provide better descriptions of the operative procedures and postoperative immobilization techniques employed for patients with os odontoideum.(2,5-9,15,17,21-26) Smith et al, described 11 children with os who underwent posterior wiring and attempted fusion.(21) Autologous bone graft and halo immobilization were used in all children. Two children had fusion failure with non-union. One child incurred an intraoperative cord injury secondary to sublaminar wire passage. Lowry et al, also described eleven children with os odontoideum that were treated with C1-2 fusion and posterior wiring.(15) One child treated with a Gallie-type procedure had continued instability and fusion failure. The C1-C2 construct was revised successfully with a Brooks-type fusion procedure. The remaining ten children were successfully treated with Brooks C1-C2 wiring and fusion procedures. Coyne et al, in a review of posterior C1-2 fixation techniques described five patients with os odontoideum.(5) Three of these five had unsuccessful attempted posterior fusions despite halo immobilization. Two developed new neurological deficits after surgery. Dai et al, described 44 patients with os odontoideum with a mean follow-up of 6.5 years.(6) Seven patients were asymptomatic at presentation. Five of these seven refused surgery and were treated with a cervical collar only, and remained stable at last follow-up. The remaining 39 patients underwent successful fusion procedures following skeletal traction. The authors reported that nine patients underwent atlantoaxial fusion and 33 required occipitocervical fusion (42 operations in 39 patients). Symptoms and signs disappeared in 26 of their operative patients and improved in the remaining 13 at last follow-up. They employed occipital-cervical constructs

with fusion with or without C1 laminectomy in those patients with irreducible deformities because of the concern that sublaminar passage of wires or cables might result in neurological morbidity.

Wang et al, reported 16 children with C1-2 instability of which four had os odontoideum.(26) These four children were treated with C1-2 transarticular screw fixation with posterior C1-C2 wiring and fusion. The youngest child was four years old. All achieved stable fusion arthrodesis without complications. Halo immobilization devices were not used. Brockmeyer et al, as well, reported 31 children they treated with C1-2 transarticular screw fixation and fusion.(2) Twelve of these children had os odontoideum. Bilateral screws were placed successfully without complication in all children with os odontoideum. They did not comment on the type of postoperative immobilization devices they employed. In 1991 Dickman and colleagues reviewed their experience with fusion plus twelve weeks of halo immobilization in the treatment of C1-C2 instability.(7) They described 36 patients with C1-2 instability, four of whom had os odontoideum. Three of four os patients they treated in this way developed osseous union. One had a stable fibrous union at last follow-up. In a subsequent report in 1998, Dickman et al, compared their series of patients undergoing C1-2 transarticular screw fixation with posterior wiring and fusion to those patients who were treated with posterior wiring and fusion alone. The fusion rates in the two groups were 98% and 86%, respectively.(8) No patient with os treated with C1-C2 transarticular fusion techniques failed to fuse. Only one of eight patients with os odontoideum in the posterior wiring and fusion group developed a nonunion (previously described). In contrast to the posterior wiring and fusion only patients, no patient treated with transarticular screw fixation required postoperative halo immobilization. Menezes and Ryken described four children with os odontoideum and Down's syndrome that they

successfully treated with posterior wiring and fusion, utilizing full thickness autograft rib, and at least three months of postoperative halo immobilization.(17) Dyck reported eight children with os odontoideum, six of whom were treated with posterior C1-3 wiring and fusion techniques.(9) All were externally immobilized in a four-poster brace “usually” for three to four months postoperatively. Two of six children required reoperation for nonunions.

Apfelbaum et al, described their experience in treating recent and remote ( $\geq 18$  months after injury) odontoid injuries with anterior screw fixation.(1) They reported a fusion rate of 25% in 16 “remote” odontoid injuries. If an os odontoideum were considered anatomically similar to a “remote” odontoid fracture, then the rate of fusion for os odontoideum treated with an odontoid screw fixation would likewise expected to be poor. Anterior C1-C2 trans-facet fixation techniques may have merit in the surgical treatment of os odontoideum, but there are no descriptions of its application for os odontoideum in the literature.

The surgical treatment of patients with C1-C2 instability in association with os odontoideum has been demonstrated to be successful when combined fusion and internal fixation techniques are employed, usually in conjunction with postoperative halo immobilization. Fusion success rates and reports of operative morbidity varied considerably among the clinical case series reported in the literature. While the numbers are small, transarticular C1-2 screw fixation and fusion has been associated with higher rates of fusion compared to posterior wiring and fusion techniques alone. Of note is that patients treated with transarticular screw fixation have been managed in hard collars postoperatively obviating the need for halo immobilization devices. If transarticular screw fixation is not utilized in the treatment of unstable os odontoideum, postoperative halo immobilization as an adjunct to dorsal internal fixation and fusion is recommended.

Ventral or transoral decompression for irreducible ventral cervicomedullary compression in association with os odontoideum has been suggested.(24) Reports of the management of ventral compression and os odontoideum are scant. In a review of 36 patients with Down's syndrome and craniovertebral junction abnormalities, twelve patients were described with os odontoideum.(24) Eleven patients of the 36 reported were noted to have basilar invagination. Five of these eleven patients with basilar invagination had irreducible ventral spinal cord compression and were treated with transoral decompression. The authors reported stable to excellent outcomes without complications following transoral decompression in all five patients; however, the total number of patients who had basilar invagination due to os odontoideum was not described. The report implies however, that selected patients with atlantoaxial instability and irreducible symptomatic ventral cervicomedullary compression may benefit from ventral decompression. On the other hand, Dai and colleagues reported the successful use of occipital cervical fusion with or without C1 laminectomy in cases of irreducible deformity with cervicomedullary neural compression in 33 patients with os odontoideum. They described improvement in all patients and no complications related to their dorsal only approach.(6) While it may seem intuitive to remove ventral neural compression in association with os odontoideum, the literature suggests that dorsal stabilization and fusion without ventral decompression is an effective management option.

## **SUMMARY**

Plain cervical spine radiographs appear adequate to make a diagnosis of os odontoideum in the vast majority of patients with this disorder. Lateral flexion and extension radiographs can provide useful information regarding C1-2 instability. Tomography (computerized or plain)

may be helpful to define the osseous relationships at the skull base, C1 and C2 in patients where the craniovertebral junction is not well visualized on plain radiographs. The degree of C1-C2 instability identified on cervical x-rays does not correlate with the presence of myelopathy. A sagittal diameter of the spinal canal at the C1-2 level of less than 13 millimeters does correlate with myelopathy detected on clinical examination. MR can depict spinal cord compression and signal changes within the cord that correlates with the presence of myelopathy.

Surgical treatment is not required for every patient in whom os odontoideum is identified. Patients who have no neurological deficit and have no instability at C1-2 on flexion and extension studies can be managed without operative intervention. Even patients with documented C1-C2 instability and neurological deficit have been managed non-operatively without clinical consequence during finite follow-up periods. Most investigators of this disorder favor operative stabilization and fusion of C1-C2 instability associated with os odontoideum. The concern exists that patients with os odontoideum with C1-C2 instability have an increased likelihood of future spinal cord injury. While not supported by Class I or Class II evidence from the literature, multiple case series (Class III evidence) suggest that stabilization and fusion of C1-C2 is meritorious in this circumstance.(6,15,24,26) Because a patient with an initially stable os odontoideum has been reported to develop delayed C1-C2 instability, and because there are rare examples of patients with stable os odontoideum who have developed neurological deficits following minor trauma, longitudinal clinical and radiographic surveillance of patients with os odontoideum without instability is recommended. (4,10)

Posterior C1-2 arthrodesis in the treatment of os odontoideum provides effective stabilization of the atlantoaxial joint in the majority of patients. Posterior wiring and fusion techniques supplemented with postoperative halo immobilization provided successful fusion in

40% to 100% of cases reported.(3,5,6,22,26) Atlantoaxial transarticular screw fixation and fusion appears to have merit in the treatment of C1-2 instability in association with os odontoideum, and appears to obviate the need for postoperative halo immobilization. Neural compression in association with os odontoideum has been treated with reduction of deformity, dorsal decompression of irreducible deformity, and ventral decompression of irreducible deformity, each in conjunction with C1-C2 or occipital cervical fusion and internal fixation. Each of these combined approaches has provided satisfactory results. Odontoid screw fixation has no role in the treatment of this disorder.

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

A cooperative multi-institutional natural history study of patients with os odontoideum without C1-C2 instability would provide demographic and clinical information that may provide predictive factors for the development of subsequent instability. In a related study, the prevalence of os odontoideum as an incidental finding should be established.

The literature supports essentially no treatment for os odontoideum, even with C1-2 sUBLuxation. Whether activity restriction is called for in these patients would be helpful and should be studied.

A cooperative multi-institutional prospective randomized trial of posterior wiring and fusion techniques with and without C1-2 transarticular screw fixation for patients with os odontoideum and C1-2 instability would help to definitely identify the risks and merits of each of the two procedures in this patient population.

## EVIDENTIARY TABLES

First Author Reference	Description of Study	Data Class	Conclusions
Apfelbaum RI, et al, 2000 <i>J Neurosurg</i>	18 patients with odontoid fractures incurred $\geq$ 18 months prior to treatment, who were treated with anterior odontoid screw fixation.	III	16 patients with follow-up. 25% fusion rate. Three with screw fracture and one with screw pull-out.
Brockmeyer DL et al, 2000 <i>J Neurosurg</i>	Review of transarticular screw placement in 31 children. 12 children with os odontoideum (ages four to 16 years).	III	55 of 62 possible sites deemed suitable for transarticular screws. All children with os odontoideum were able to have two screws placed.
Dai L et al, 2000 <i>Surg Neurol</i>	A review of 44 patients ages seven to 56 years with os odontoideum. Mean follow-up of 6.5 years.	III	7 patients were asymptomatic. five of these seven were treated with a cervical collar only and have remained stable. 39 underwent fusion successfully (9 atlantoaxial and 33 occipitocervical). Symptoms and signs disappeared in 26 and improved in 13.
Taggard DA et al, 2000 <i>J Neurosurg</i>	A review of craniovertebral junction abnormalities in 36 Down's Syndrome patients. Os odontoideum present in 12.	III	Twenty-seven underwent surgical procedures. Of 11 with basilar invagination, it was irreducible in five and transoral decompression was performed.
Dickman CA et al, 1998 <i>Neurosurgery</i>	Review of 121 patients treated with posterior C1-2 transarticular screws and wired posterior bone struts. Os odontoideum was present in 9. This group was compared to 74 patients treated with posterior wiring techniques alone.	III	2 failures in the transarticular group. The etiology of the C1-2 instability was not stated for these two failures. One of eight patients with os odontoideum in the posterior wiring group had a nonunion. Overall fusion rate for transarticular was 98% versus 86% for posterior wiring techniques.
Wang J, et al, 1999 <i>Pediatr Neurosurg</i>	16 children treated for atlanto-axial instability. Four of which had os odontoideum who were treated with C1-2 transarticular screws and posterior wiring and fusion techniques.	III	All fused. No halo immobilization. Transarticular screws were successfully used in children as young as 4-years
Kuhns LR, et al, 1998 <i>J Pediatr Ortho</i>	4 children with os odontoideum underwent MR examinations.	III	All four children had changes in the nuchal cord consistent with injury.
Lowry DW et al, 1997 <i>J Neurosurg</i>	A review of 25 children requiring upper cervical fusions. 11 children had os odontoideum.	III	10 underwent a Brooks type C1-2 fusion. two of these children did not fuse. one underwent a Gallie type fusion. This child remained unstable and was revised to a Brooks type fusion which was successful.
Matsui H, et al, 1997 <i>Spine</i>	Review of the plain radiographic morphology of C2 was evaluated in 12 patients (15 to 71 years-old) with os odontoideum unrelated to any syndrome.	III	Three configurations described from an anteroposterior view: Round, cone, and blunt-tooth. Myelopathy was more severe in the group with a round configuration.
Verska JM 1997 <i>Spine</i>	Report of a pair of identical twins, one with os odontoideum, and one without an os odontoideum	III	History of trauma in the twin with an os odontoideum. Fell at age three years, had torticollis and neck pain for several months.

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
Watanabe M et al, 1996 <i>Spine</i>	Review of 34 patients with os odontoideum (5 to 76 years-old). Divided into groups by Rowland Classification (1=local symptoms, two =post-traumatic transient myelopathy, 3,4=progressive myelopathy or intracranial symptoms). Lateral neutral and dynamic radiographs obtained. Sagittal plane rotation angle, minimum distance, and instability index were measured.	III	Low correlation between sagittal plane rotation angle and instability index. Sagittal plane rotation angle of > 20 degrees or instability index of > 40% correlates with myelopathy.
Clements WD et al, 1995 <i>Injury</i>	Report of nonoperative treatment of an incidentally discovered os odontoideum without C1-2 instability at diagnosis	III	After five years profound C1-2 instability and symptoms had developed necessitating posterior instrumentation and fusion.
Coyne TJ et al, 1995 <i>Neurosurg</i>	Review of posterior C1-2 fusion and instrumentation techniques. Five of 32 patients had os odontoideum.	III	3 of five with os odontoideum failed with posterior wiring techniques. All were immobilized in halos. two of five developed new neurological deficits as operative complications.
Stevens JM et al, 1994 <i>Brain</i>	Review of abnormal odontoids and C1-2 instability. 24 of 62 patients with os odontoideum. nine children and 15 adults.	III	Periodontoid soft tissue thickening was only present in those with Morquio's disease. Following fusion the odontoid was noted to partially or completely regenerate in cases of Morquio's disease.
Menezes AH et al, 1992 <i>Neurosurg</i>	Review of 18 Down's syndrome patients with symptomatic cervicomedullary compromise. Four had os odontoideum.	III	All four had gross instability on dynamic radiographs. Successful fusion with posterior wiring techniques and full thickness rib grafts. Immobilized for a "minimum of three months".
Dickman CA et al, 1991 <i>J Neurosurg</i>	Review of 36 patients treated with C1-2 posterior wiring and fusion for various reasons. Four patients had os odontoideum (ages 16,25,38,43). All patients were maintained in a halo for 12 weeks after surgery.	III	Of the four with os odontoideum, three developed osseous unions and one had a stable fibrous union (follow-up of 15 to 44 months). No complications for these four patients.
Hosono N et al, 1991 <i>Spine</i>	Cineradiographic evaluation of six patients with os odontoideum	III	2 types of C1 posterior arch translation: straight (vertical)(n=4) and sigmoid (n=2). Correlated abnormal motion with biomechanics of posterior wiring techniques.
Smith MD et al, 1991 <i>Spine</i>	Review of 17 children operated on for C1-2 instability. 11 had os odontoideum. Posterior wiring techniques, autologous bone, and halo used in all.	III	2 of the 11 with os odontoideum had non-unions. One cord injury thought secondary to sublaminar wire passage.

First Author Reference	Description of Study	Data Class	Conclusions
Shirasaki N et al, 1991 <i>Spine</i>	9 patients with os odontoideum and posterior instability had three radiographic parameters measured. Distance between the os and C2 spinous process in extension (Dext), distance between the os and posterior C1 arch (Dat1), and “degree of instability” (Inst). These findings were compared to their neurological status.	III	Those without history or evidence of myelopathy had a Dext of > 16 mm. Dext was ≤ 16 mm in those with myelopathy. The presence or absence of myelopathy was not related to the Inst. In those with myelopathy and a Dat1 > 13 mm there was reversible cord compression in extension, in those with a Dat1 of ≤ 13 mm the cord remained compressed in flexion and extension.
Morgan MK et al, 1989 <i>J Neurosurg</i>	Report of three family members with C2-3 Klippel-Feil abnormalities and os odontoideum	III	Ages 16 (index case), 39 (father), and 64 (paternal grandmother). None with neurological signs or symptoms.
Yamashita Y et al, 1989 <i>Acta Radiologica</i>	Correlation of clinical status, MR, and radiographs in 29 patients with C1-2 instability. four had os odontoideum.	III	The atlanto-dens interval did not correlate with the degree of myelopathy but MR degree of cord compression did correlate with degree of myelopathy.
French HG, et al, 1987 <i>J Pediatr Ortho</i>	Review of dynamic cervical spine radiographs in 185 patients with Down’s Syndrome	III	Six had abnormal odontoids consistent with os odontoideum for an incidence of 3%. Three had prior radiographs showing no abnormality. One had an exaggerated ADI of six mm.
Spierings EL et al 1982 <i>J Bone Joint Surg (Br)</i>	37 patients with os odontoideum. 20 treated conservatively.	III	Of 20 managed conservatively, one was lost to follow-up. 15 had no myelopathy (median f/u of five years) and none developed myelopathy. Of four with myelopathy (f/u of 0.5, 1, 7, and 14 years) one is dead from cancer, one has neck pain, one has neck pain and paresthesias, and one has headaches.
Fielding HG et al, 1980 <i>J Bone Joint Surg (Am)</i>	35 patients (3 to 65 years-old) with os odontoideum. 25 patients were symptomatic.	III	22 patients had anterior instability with a mean ADI of 10.3 mm. five had posterior instability. three had no detectable motion. three had less than three mm of C1-2 motion. 26 underwent posterior fusion successfully. Five were not operated, three were asymptomatic with no instability. They remained well with no instability at 1,2, &3 years, respectively. One patient with instability refused surgery but was well at two years follow-up. One patient died of renal failure.
Brooks AL et al, 1978 <i>J Bone Joint Surg (Am)</i>	3 children (8,11,12 years-old) with os odontoideum treated with sublaminar C1-2 wires and autologous iliac crest graft. Minerva cast immobilization	III	All fused. Spontaneous extension of fusion to C3 in one child.

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
Dyck P, 1978 <i>Neurosurg</i>	Review of eight children (ages seven to 17 years) with os odontoideum. six were treated with posterior wiring and fusion of C1-3. External immobilization for “usually” three to four months.	III	6 children underwent posterior fusion by the author. Two required reoperation.
Griswold DM et al, 1978 <i>J Bone Joint Surg (Am)</i>	4 patients with os odontoideum treated with sublaminar C1-2 wires and autologous iliac crest.	III	3 fused. One did not fuse after three attempts.

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## TREATMENT OF SUBAXIAL CERVICAL SPINAL INJURIES

### RECOMMENDATIONS

#### **Subaxial Cervical Facet Dislocation Injuries:**

Standards: There is insufficient evidence to recommend treatment standards.

Guidelines: There is insufficient evidence to recommend treatment guidelines.

Options:

- Closed or open reduction of subaxial cervical facet dislocation injuries is recommended.
- Treatment of subaxial cervical facet dislocation injuries with rigid external immobilization, anterior arthrodesis with plate fixation, or posterior arthrodesis with plate fixation is recommended.
- Treatment of subaxial cervical facet dislocation injuries with prolonged bedrest in traction is recommended if more contemporary treatment options are not available.

#### **Subaxial Cervical Injuries Excluding Facet Dislocation Injuries:**

Standards: There is insufficient evidence to recommend treatment standards.

Guidelines: There is insufficient evidence to recommend treatment guidelines.

Options:

- Closed or open reduction of subluxations or displaced subaxial cervical spinal fractures is recommended.
- Treatment of subaxial cervical spinal injuries with external immobilization, anterior arthrodesis with plate fixation, or posterior arthrodesis with plate fixation is recommended.

## **RATIONALE**

Subaxial cervical vertebral fracture dislocation injuries are common following non-penetrating cervical trauma and are often associated with neurological injury. Prior to the advent of spinal instrumentation, many of these injuries were managed with traction, postural reduction, or external orthoses with frequent success. However, the morbidity and mortality associated with prolonged immobilization for three months or more prompted surgeons to investigate the utility of internal fixation in the management of these injuries. In order to develop treatment recommendations for closed subaxial cervical spinal injuries, an analysis of the articles examining their management is undertaken in this report. In particular, this focused review assessed the utility of closed reduction with or without external immobilization compared to arthrodesis with or without internal fixation.

## **SEARCH CRITERIA**

A National Library of Medicine computerized literature search of publications from 1966 to 2001 was performed using the following headings limited to the English language: cervical vertebrae, spinal fractures, and dislocations. An exploded search of the headings led to 15,124, 3,010 and 17,811 citations respectively. The first heading was combined with the second two headings, leading to a subset of 688 and 1159 citations respectively. Another exploded search of therapeutics or treatment limited to the English language led to 1,566,596 citations. This was combined with each of the two prior subsets, leading to 198 and 287 citations respectively. The abstracts were reviewed and only those containing ten or more cases of subaxial cervical injury after non-penetrating cervical trauma were included. An exception was made for ankylosing spondylitis because of the paucity of reports including more than ten patients with this disorder.

Sixty-three articles met the selection criteria and provide the basis for this review. They are summarized in Evidentiary Table format.

## **SCIENTIFIC FOUNDATION**

The variety and heterogeneity of subaxial cervical spinal injuries requires accurate characterization of the mechanics and type of cervical spinal injury in order to compare the efficacy of operative and nonoperative treatment strategies. The absence of a uniformly accepted classification scheme for cervical vertebral injuries limits the ability to compare the effects of treatment reported in multiple clinical studies. In 26 articles describing series of patients with cervical injuries, subaxial cervical injuries are not differentiated. The Allen and Ferguson (3) classification system has been the most commonly used scheme to differentiate and characterize subaxial vertebral injuries. Although few authors reported injuries by subtype, many of the reports described cervical injuries that could be grouped into the following broad categories as described by Allen and Ferguson: distractive flexion (DF), compressive flexion/vertical compression (CF/VC), extension, and subluxation. The effectiveness of treatments of subaxial cervical spinal facet dislocation injuries, a subset of DF injuries, may be different from other subaxial cervical spinal fracture-dislocation injuries and are considered separately (53). Finally, four articles described unique characteristics of the management of subaxial cervical injuries in patients with ankylosing spondylitis and are included in this review.

Several general principles can be analyzed in the review of 26 articles that describe the treatment of subaxial cervical injuries without accurate differentiation into specific injury types. Although closed reduction was successful in 64% to 91% of patients with traumatic subaxial cervical malalignment (5,28,35,43), patients with delayed treatment of injuries had a higher

failure rate of closed reduction (22.5%) compared those treated early.(5) Halo vest application was employed successfully to immobilize patients with subaxial cervical injuries on arrival to the hospital to facilitate transport and workup; none had neurological worsening.(32) Orthoses failed to maintain reduction of subaxial cervical fracture-dislocation injuries in 7% to 56% of patients.(10,16,18,21,22,23,27,35,53,46) Overall, 30% of these injuries (222 of 752) had recurrent displacement or inadequate alignment during external immobilization (503 - halo vest, 249 - traction). Six of these patients were reported to heal with good ultimate alignment after readjustment of the halo device (three patients), or continued postural reduction (three patients).(27,46) Nineteen percent (140 of 752 patients) were maintained in external immobilization despite displaced injuries and healed in an unreduced, non-anatomic position. (10,16,18,21,23,27,35,53,46) Eleven percent (82 of 752 patients) underwent subsequent surgical treatment, typically for correcting cervical malalignment.(10,16,18,22,23,27,35,53,46) Several risk factors were identified in association with failure of nonoperative management of subaxial cervical injuries. Patients with more than 40% compression of a cervical vertebra, more than 15° of kyphotic angulation, or more than 20% subluxation of one vertebra on another were more likely to fail treatment with external immobilization (craniocervical traction alone or traction followed by external orthosis).(35)

In contrast, failure to maintain anatomic reduction of subaxial cervical fracture dislocation injuries after operative treatment ranged from 1% to 18%.(7,10,23,28,36,48,51,46,56) Anterior cervical fusion procedures (28,48,59,56) were associated with less frequent failure to maintain reduction (5% - ten of 213) when compared with posterior cervical fusion procedures (14% - 38 of 280) among all patients with subaxial cervical injuries treated operatively.(7,36,51) Overall, nine percent of patients (61 of 704) had recurrent angulation or subluxation despite

surgical management. (7,10,23,28,33,35,36,46,48,51,56,59) A second operation in treatment of progressive deformity was rare in these patients. Successful arthrodesis occurred in nearly every patient reported.(7,15,33,36,43,46,48,56) Surgical complications were relatively common in these series, ranging from 9% to 25%.(7,20,27,33,46,48,56,59) In particular, graft extrusion after anterior cervical surgery without plate fixation was observed in as many as 10% of patients managed in this way.(20,27) Overall four of 104 patients experienced graft displacement (4%) (20,23,27,35,52,59) after anterior fusion without plate fixation, compared to none of 291 patients treated with anterior fusion with plate fixation.(5,28,33,48,56) Complications have been reported utilizing posterior plate fixation as well; radiculopathy occurred in 25% of patients in one report describing these techniques.(7,36,51)

### **Subaxial cervical facet dislocation injuries:**

Twenty-eight articles provided sufficient information to evaluate patients with subaxial cervical distractive flexion (DF) injuries. Most reports were retrospective series of patients with subaxial cervical spinal facet dislocation injuries, unilateral, bilateral, or both. (8,11-13,16,19,27,30,37,39,41,45,47,49,50,53,54,55,57,61) Overall, 26% of patients (181 of 701) with cervical spinal facet dislocation injuries had failure to achieve closed reduction with craniocervical traction. (8,11,12,17,19,30,39,41,42,45,47,50,53-55,57,61) Reduction, when accomplished, could not be maintained in 28% of patients (112 of 393) treated with subsequent external immobilization. (8,11-13,16,19,27,30,37,39,41,45,47,49,50,53-55,57,61) Mortality associated with closed treatment of facet dislocation injuries was 7% (28 of 392 patients) in series reporting this complication. (8,11-13,19,30,37,39,41,45,50,53,57,61) Prolonged bedrest and cervical traction alone for 12 weeks to 16 weeks duration was associated with the highest

mortality of all treatment strategies reported for these injuries, 27% in one series of 41 patients managed in this way.(13)

Vertebral subluxation, facet injury (ligamentous or fracture) or a locked/perched facet on the initial radiographs or subsequent CT or MRI studies have been cited as factors associated with failure of nonoperative treatment.(8,11,30,31,57) Facet fractures associated with cervical spinal facet dislocation injuries may preclude successful closed reduction. (30,57) They have also been associated with a high rate of arthrodesis with external immobilization alone (halo device) if closed reduction can be accomplished, 97% in one report on this issue. (30) Ligamentous disruption without facet fracture is associated with an increased likelihood of failure of external immobilization (halo device, Minerva cast) in the treatment of these injuries. (30,31) Laminar fractures have been associated with an increased risk of late kyphosis after surgical treatment of cervical spinal facet dislocation injuries.(40) Although patients with unreduced facet dislocations treated with external immobilization often achieve spinal stability once treatment has been completed, arthrodesis in a position of malalignment has been associated with persistent cervical pain.(8,50,55) No differences were observed in the success of achieving closed reduction and/or maintaining cervical spinal alignment in patients with unilateral facet dislocations compared to patients with bilateral facet dislocation injuries.

In contrast, open reduction was achieved in all but one of 24 patients treated with anterior fusion procedures (42,44,55) and in all but seven of 167 patients treated with posterior fusion procedures in series that reported this finding.(7,8,11,19,30,42,47,50,54,57,61) Delayed instability occurred in six of 101 patients (6%) treated with anterior fusion procedures (12,13,24,40,42,55,61), and six of 237 patients (3%) treated with posterior fusion procedures. (7,8,11,25,30,37,40,42,49,50,55,57,61) Not one of these six patients who failed to achieve

stability after anterior fusion was treated with plate fixation in addition to fusion. Seven of 85 patients (8%) treated with anterior fusion procedures developed kyphosis; none had been treated with anterior plate fixation.(7,8,11,25,30,37,40,42,49,50,55,57,61) Sixteen patients described by Shapiro also developed kyphotic angulation following anterior cervical fusion without internal fixation.(58) In contrast, 22 of 165 patients (13%) developed kyphosis after posterior cervical fusion with wiring, (25,40,54,55) while only one of 40 patients (3%) developed kyphosis after posterior fusion with lateral mass plate fixation.(25,55) Alternatively, Halifax interlaminar clamps were successfully used in five patients with facet dislocations treated with posterior arthrodesis.(2)

Graft displacement was the most common complication after attempted anterior arthrodesis without internal fixation (8%, seven of 85 patients).(6,13,24,40,42,44,55) Seven percent of patients (8 of 113) died after anterior fusion procedures (13,24,40,42,44,45,55,61); three percent of patients (7 of 268) died after posterior fusion procedures. (7,11,19,25,30,42,47,49,50,55,57,61) All but one of the 15 patients who died following surgery in an attempt to correct deformity and stabilize the spine in these reports had complete cervical spinal cord injuries.(7,42,57,61)

### **Subaxial Cervical Spinal Injuries Excluding Facet Dislocation Injuries:**

Fourteen articles provided sufficient information to evaluate patients with subaxial cervical spinal compression fracture injuries. (1,4,6,11,13,14,16,17,24-26,34,37,39) Although some authors differentiated compressive flexion (CF) injuries from vertical compression (VC) injuries, others considered these injuries together. Many nonoperative treatment strategies were described including traction and external immobilization in collar, plaster jacket, or halo vest.

Overall, 5% of patients (17 of 349) treated with immobilization for compressive injuries of the subaxial cervical spine had persistent instability after non-operative treatment employed for eight weeks to 12 weeks.(6,11,13,17,26,37,39) In contrast, nearly every patient with these injuries treated with anterior (22 of 22) or posterior (26 of 27) fusion procedures developed a stable union.(1,4,25,27) Subluxation or kyphosis developed in two of 18 patients who were treated with posterior fusion.(11,25) Operative complications were more common in patients treated with posterior fusion procedures (37%, ten of 27) when compared with anterior fusion procedures (9%, three of 33).(1,4,24,25) Graft displacement was the most common complication described in patients treated with anterior cervical fusion without internal fixation (9% - three of 33).(1,24)

Only seven articles reported sufficient data to analyze patients treated for extension injuries of the subaxial cervical spine.(4,11,13,24,37,38,43) Twenty-four percent of patients (19 of 79) failed treatment with external immobilization.(11,13,37,38,43) In contrast, not one of 19 patients failed treatment with anterior cervical fusion.(37,38) Two patients had irreducible vertebral displacements and three patients developed kyphotic deformities among eleven patients with cervical spinal extension injuries treated with attempted posterior cervical fusion.(38)

Eight articles reported sufficient data to analyze patients treated for vertebral subluxation injuries of the cervical spine.(4,6,11,13,17,18,25,49) Sixty-four percent of patients with these injuries had successful treatment with external immobilization; patients with greater than 50% subluxation were twice as likely to maintain anatomic cervical realignment after closed reduction (72% vs. 36%).(6) Thirty-six percent of patients (39 of 108) failed external immobilization following closed reduction (11,13,17,18,49) compared to seven percent of patients with these injuries managed surgically.(4,49) A kyphotic deformity developed in four percent of reported

patients (three of 74) treated with posterior cervical fusion and lateral mass plate fixation procedures.(4,25)

Several characteristics of subluxation injuries of the subaxial cervical spine were associated with failure of nonoperative treatment.(6,53) Patients with subluxation or kyphotic angulation frequently failed to achieve a good anatomical result after treatment with halo vest immobilization (45% - 46 of 103). Combined fractures to all parts of the cervical spinal column and the presence of facet fractures were not associated with a higher likelihood of failure of treatment with external immobilization.(53) Closed reduction was more successful with a subluxation greater than 50% of the vertebral body diameter.(6)

Comparatively few studies examined the specific difficulties associated with the management of patients with ankylosing spondylitis who sustain cervical spinal injuries.(9,17,29,60) In four articles reporting patients with this entity and injury, nine of 22 total patients died. Four patients managed non-operatively died. Two of nine survivors treated with external immobilization failed treatment. One worsened neurologically when placed in a halo and was subsequently successfully treated with laminectomy and fusion. The other patient had persistent cervical subaxial spinal instability but refused further therapy. In contrast, five of nine ankylosing spondylitis patients with cervical fracture injuries treated primarily with surgery died. One patient was neurologically worse after surgery. Three patients healed successfully without instability.

## **SUMMARY**

In conclusion, closed reduction is successful for most subaxial cervical spinal fracture-dislocation injuries. Failure of closed reduction is more common with facet dislocation injuries.

Similarly, treatment with external immobilization is frequently successful in the management of most subaxial cervical spinal injuries, although failure to maintain reduction is more frequent with facet dislocation injuries as well. Virtually all forms of external immobilization have been employed in the treatment of subaxial cervical spinal injuries. More rigid orthoses (halo, Minerva) appear to have better success rates than less rigid orthoses, (collars, traction only) for fracture-dislocation injuries once reduction has been accomplished. Treatment with traction and prolonged bedrest has been associated with increased morbidity and mortality.

Both anterior and posterior cervical fusion procedures are successful in achieving spinal stability for the majority of patients with subaxial cervical spinal injuries. Indications for surgical treatment offered in the literature include failure to achieve anatomic injury reduction (irreducible injury), persistent instability with failure to maintain reduction, ligamentous injury with facet instability, spinal kyphotic deformity greater than 15°, vertebral body fracture compression of 40% or greater, vertebral subluxation of 20% or greater, and irreducible spinal cord compression. Anterior fusion without plate fixation is associated with an increased likelihood of graft displacement and the development of late kyphosis, particularly in patients with distractive flexion injuries. Similarly, late displacement with kyphotic angulation is more common in patients treated for facet dislocation injuries with posterior fusion and wiring compared to those treated with posterior fusion and lateral mass plate fixation. Although patients with persistent or recurrent cervical spinal malalignment often achieve spinal stability with either external immobilization or surgical fusion with or without internal fixation, a greater proportion of these patients have residual cervical pain compared to similarly treated patients in whom anatomic spinal alignment was achieved and maintained.

## **KEY ISSUES FOR FUTURE INVESTIGATION**

To better compare the advantages and disadvantages of nonoperative versus operative treatment strategies for subaxial cervical injuries, future studies must differentiate between the mechanisms of injury which have resulted in subaxial injury. Although the Allen and Ferguson classification offers a commonly used framework for stratifying these patients, the number of subtypes in their scheme precludes many investigators from obtaining sufficient numbers of patients with specific injuries. A broader classification of patients into compressive flexion (CF), distractive flexion (DF), vertical compression (VC), and extension injuries would allow comparison of most patients who sustain subaxial cervical spinal injuries. A multicenter study would allow more rapid accumulation of patients with these various categories of subaxial cervical injuries. A prospective examination of the efficacy of rigid external immobilization compared to surgical arthrodesis with internal fixation (anterior and posterior approaches) may further refine the most effective treatment for patients with subaxial cervical spinal injuries.

## EVIDENTIARY TABLES: Injury Types Mixed

First Author Reference	Description of Study	Data Class	Conclusions
Kalff R et al, 1993, Neurosurg Rev	Retrospective study 97 cervical injuries (79 ant and 18 ant-post fusion w/ plate) 16 DF, 14 VC, 64 Fx-dislocation	Class III	9% operative complications related to fixation devices, but less than half require reoperation. All patients fuse.
Lemons VR & Wagner FC, 1993, Surg Neurol	Retrospective study 64 cervical fractures 14 VC, 12 CF, 12 UFD, 16 BFD 10 extension 38 halo 38 fusion (12 failed halo, 26 primary) 26 post w/ wire or plates, 4 ant w/o plate, 4 ant-post	Class III	12 of 38 halo treated injuries were unstable and were fused. 4 healed malaligned. None with extension injuries were unstable. 5 of 38 treated w/ fusion were unstable. Risk for orthosis failure: >40% compression, >15° angulation, >20% subluxation.
Cybulski GR et al, 1992, Spine	Retrospective study 21 cervical injuries failing posterior wiring txed with anterior fusion	Class III	2 of 21 had graft extrusions.
Della Torre F & Rinonapoli E, 1992, Inter Orthop	Retrospective study 28 cervical injuries 3 CE, 7 CF, 4 DF <b>treated</b> with halo	Class III	4 of 7 CF injuries were not reducible. All were stable with immobilization.
Heary RF et al, 1992, J Trauma	Retrospective study 78 cervical injuries Halo for transport 49 subaxial <b>fractures</b> , 45 subaxial subluxation	Class III	No patient worsened neurologically in halo before receiving definitive treatment.
Levine AM et al, 1992, Spine	Retrospective study 24 facet fractures Posterior fusion with plates	Class III	11 complications including 4 who lost correction and 6 with radiculopathy. All achieved fusion.
Roy-Camille et al, 1992, Spine	Retrospective study 221 cervical injuries 89% post fusion 11% ant fusion	Class III	15% develop kyphosis after surgery.
Nazarian SM & Louis RP, 1991, Spine	Retrospective study 23 cervical injuries Posterior fusion with plates 11 <b>unilateral facet dislocation (UFD)</b> , 4 <b>bilateral facet dislocation (BFD)</b> , 3 <b>subluxation</b> , 5 <b>facet fractures</b>	Class III	3 of 12 failed closed reduction and 3 were unstable in an orthosis. All achieved fusion.
Ripa et al, 1991, Spine	Retrospective study 92 cervical injuries Ant fusion w/ plate 48 multifix, 20 VC, 13 DF, 6 extension	Class III	No patient worsened neurologically. 12 of 15 complications were hardware related. 1 patient had pseudarthrosis.

First Author Reference	Description of Study	Data Class	Conclusions
Sears W & Fazl M, 1990, J Neurosurg	Retrospective study 173 cervical injuries 103 non-facet dislocation injuries Halo treatment Operative procedure unreported	Class III	31 of 103 patients failed nonoperative treatment (3 were irreducible, 10 were neurologically worse, 16 subluxed in halo, 2 had late instability). Sublux and angulation predicted failed treatment, while fracture did not.
Benzel E and Ketersen L, 1989, J Neurosurgery	Retrospective study 50 cervical injuries 25 fx-subluxation Post fusion w/ wire	Class III	1 complete patient of 25 patients died. Remainder healed.
Goffin et al, 1989, Neurosurg	Retrospective study 41 cervical injuries Anterior fusion with plate	Class III	2 of 41 subluxed, requiring surgery. 3 of 12 dislocations were irreducible. All 4 deaths were in quadriplegics.
Shoung H and Lee L, 1989, Acta Neurochir	Retrospective study 37 cervical injuries Ant fusion w/ plate	Class III	All 37 healed. No graft extrusion. 1 death, 1 infection, 2 screw loosening.
Argenson C et al, 1988, Spine	Retrospective study 47 cervical injuries 7 posterior fusion 40 anterior fusion	Class III	17 of 22 were reducible, but 5 old dislocations were irreducible. 1 died of vertebrobasilar thrombosis.
Bucci MN et al, 1988, J Trauma	Retrospective study 49 cervical injuries: 20 halo alone (1 refused) 28 fusion w/immob, proced unreported	Class III	12 of 20 with halo stable. 26 of 28 fused were stable (p<0.01). 2 in each group lost reduction. 1 in each group neuro worse.
Donovan WH et al, 1987, J Neurosurg	Retrospective study 61 cervical injuries: 17 fusion w/immob (4 ant, 13 post) 43 6wk tx to halo 1 lami w/o fusion	Class III	18 of 43 had alignment in halo . 3 of 9 DF unstable:2 surgery/1 asymp. All patients treated with fusion were stable but 3 developed kyphosis.
Savini R et al, 1987, Spine	Retrospective study 12 cervical injuries with late instability after closed <b>treatment</b>	Class III	No grafts dislodged when anterior fusion was performed before posterior reduction.
Ersmark H & Kalen R, 1986, Arch Orthop Trauma Surg	Retrospective study 64 cervical injuries with halo vest (36 subaxial)	Class III	29 dislocations and 5 VC injuries were stable after halo vest treatment.
Glaser et al, 1986, J Neurosurg	Retrospective study 245 cervical injuries 125 complex fxs Halo treatment Fusion posteriorly w/ wire or anteriorly w/o plate.	Class III	17 of 86 lost alignment in the halo. 2 interbody grafts displaced after surgery w/o plate.
De Smet L et al, 1984, Acta Orthop Belgica	Retrospective study 28 cervical injuries with traction	Class III	4 of 28 failed early reduction. 2 of 24 had late instability.
Cahill DW et al, 1983, Neurosurg	Retrospective study of 25 DF or CF injuries txed with posterior fusion with wiring	Class III	18 of 18 with 3 month F/U were stable and none had complications.

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
Chan RC et al, 1983, J Neurosurg	Retrospective study 188 cervical injuries 150 subaxial w F/U Halo treatment	Class III	4 of 55 fx-disloc or complex fx, 13 of 53 had UFD/BFD and 2 of 41 VC unstable
Cooper PR et al, 1979, J Neurosurg	Retrospective study 33 cervical injuries txed halo	Class III	2 of 11 "complex" fxs. 1 of 3 subluxations unstable
Verbeist H, 1969, J Bone Joint Surg Am	Retrospective study 47 cervical injuries Ant fusion w/o plate	Class III	5 patients died, 4 with complete spinal cord injuries. 6 had residual malalignment and 1 other had reop for lost alignment.
Paeslack et al, 1967, Proceedings Vet Admin Spinal Cord Injury Conf	Retrospective study 221 cervical injuries 68 CF, 114 DF Postural reduction, Traction 31 cervical injuries Ant or post fusion	Class III	75 aligned, 67 wedged, 43 partially reduced, 36 failed reduction. Four of 221 had late instability, 3 stable with further treatment and 1 with surgery. 2 of 31 were unstable after surgery.
Koskinen EVS & Nieminen R, 1967, Inter Surg	Retrospective study 159 cervical injuries Various treatments	Class III	No difference in pain, neck mobility, radiculopathy, or mortality when comparing operative and nonoperative treatments.

**EVIDENTIARY TABLE: Distractive Flexion (DF)**

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
Ordonez et al, 2000, J Neurosurg	Retrospective study 6 UFD, 4 BFD 9 anterior reduction and fusion, 1 A-P-A	Class III	10 with stable fusions, although 1 was incompletely reviewed.
Shapiro S et al, 1999, J Neurosurg	Retrospective study 51 UFD 24 SP wiring 22 SP wire + plates 5 Ant-Post-Ant	Class III	1 of 24 with wire failed and 13 of 24 had late kyphosis. All patients with plate fixation had stable fusions.
Fehlings et al, 1994, J Neurosurg	Retrospective study 44 cervical injuries 19 facet dislocations Post. fusion w/ plate	Class III	5 of 19 patients had complications including 2 late failed reductions.
Lieberman IH & Webb JK, 1994, J Bone Joint Surg Br	Retrospective study 41 cervical injuries 9 facet dislocations Patients >65 years old	Class III	5 patients died, one treated with traction and 4 with halo. 3 of 4 survivors treated with traction healed. All 4 survivors with halo treatment healed.
Lukhele M, 1994, S Afr J Surg	Retrospective study 43 facet dislocations 12 with laminar fx Post fusion w/ wire	Class III	5 of 12 patients developed kyphosis.
Pasciak M & Doniec J 1993, Arch Orthop Trauma Surg	Retrospective study 32 UFD 23 nonoperative (tx + halo or plaster vest) 9 operative	Class III	All 9 treated with surgery healed. 8 of 23 who failed closed reduction were fused. 7 of 15 who failed to maintain reduction were fused.
Shapiro SA, 1993, Neurosurgery	Retrospective study 24 UFD Post fusion w/ wire	Class III	23 of 24 patients with surgery healed. 1 with re-subluxation healed with ACF. Nine of 17 who failed closed reduction had fractures of laminae.
Hadley et al, 1992, Neurosurgery	Retrospective study 31 UFD 37 BFD	Class III	18 of 29 UFD and 20 of 37 BFD successful closed reduction. 16 UFD and 15 BFD healed in halo. 7 of 31 failed halo treatment. (5 of 7 without assoc. facet fractures). When facet fractures present, once reduced, 97% success rate in halo. UFD/BFD results similar.
Mahale YJ & Silver JR, 1992, J Bone Joint Surg Br	Retrospective study 13 missed BFD with neurologically worse and late treatment	Class III	All 13 reduced (10 completely). 12 of 13 healed with traction, 1 needed surgery.
Beyer et al, 1991, J Bone Joint Surg Br	Retrospective study 36 UFD +/- fx 24 tx, halo or orthosis 10 posterior ORIF	Class III	15 of 24 reduced in halo. 8 of 10 reduced with surgery. 11 of 24 failed halo. All 10 healed with surgery. Pain was more frequent despite healing if unreduced.

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
Wolf A et al, 1991, J Neurosurg	Retrospective study 52 BFD 44 post fusion w/wire 3 ant fus+plate, 2 both	Class III	12 of 52 failed closed reduction. All 3 deaths had complete quadriplegia.
Cotler HB et al, 1990, Spine	Retrospective study 23 UFD (10 nonop) 12 BFD (4 nonop) 30 fused (21 primary)	Class III	1 of 2 failed halo. 8 of 12 failed traction. Complications were not reported.
Rockswold et al, 1990, J Trauma	Retrospective study 140 cervical injuries 8 facet dislocations (6 UFD, 2 BFD) txed halo or surgery	Class III	1 of 6 UFD failed halo. 0 of 4 failed surgery. 1 of 2 BFD failed halo. 2 of 9 failed surgery.
Sears W & Fazl M, 1990, J Neurosurg	Retrospective study 173 cervical injuries 70 dislocation injuries (38 UFD, 32 BFD)	Class III	19 healed with halo, 16 in good alignment. 16 failed reduction and 23 subluxed in halo required surgery. Subluxation and angulation were not associated with failure of halo. UFD/BFD results similar.
Benzel E and Kesterson L, 1989, J Neurosurg	Retrospective study 50 cervical injuries 19 UFD, 6 BFD Post fusion w/ wiring	Class III	All healed with fusion 1 BFD neuro worse required ACF, 1 UFD incomplete pt died
Bucholz R and Cheung K, 1989, J Neurosurg	Retrospective study 124 cervical injuries 20 DF injuries tx halo or surgery	Class III	9 of 20 failed halo. 1 neuro worse postop, unreported if DF or subluxation patient
Osti OL et al, 1989, J Bone Joint Surg Br	Retrospective study 167 dislocations 82 nonoperative (Traction) 85 operative (Ant fusion w/o plate)	Class III	6 of 82 who failed reduction were fused. 14 of 76 with late instability were fused 7 operatively treated within 24 hr died (all ASIA A).
Lind B et al, 1988, Spine	Retrospective study 83 injuries treated with halo	Class III	4 of 31 failed halo. Loose pins common.
Rorabeck CH et al, 1987, Spine	Retrospective study 26 UFD	Class III	20 of 26 failed closed reduction. 10 healed in halo. 8 of 10 remaining reduced with surgery. Pain common with failed reduction.
Glaser et al, 1986, J Neurosurg	Retrospective study 245 cervical injuries 17 dislocations	Class III	3 of 12 UFD failed halo. 1 of 5 BFD failed halo.
Maiman DJ et al, 1986, Neurosurgery	Retrospective study 26 BFD with data 14 post fus w/ wire 12 ant/AP fusion	Class III	10 of 18 reduced with closed reduction. 3 died, all complete injuries.
Chan RC et al, 1983, J Neurosurg	Retrospective study 188 cervical injuries 150 subaxial w F/U 40 halo alone 20 halo, post fusion	Class III	27 of 40 healed with halo. All 20 with primary surgery healed.

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
Dorr LD et al, 1982, Spine	Retrospective study 117 cervical injuries 25 flex-rot injuries	Class III	2 of 3 ACF had complications (1 graft displaced, 1 kyphosis).
Sonntag VKH, 1981, Neurosurgery	Retrospective study 15 BFD Halo or surgery	Class III	10 of 15 reduced with closed reduction. 4 halo heal (2 no FU) 8 post fusion with wire healed, 1 died (complete).
Stauffer ES & Kelly EG, 1977, J Bone Joint Surg Am	Retrospective review 10 dislocations 5 fractures 1 fracture subluxation Anterior fusion	Class III	16 of 16 had recurrent angular deformity after ACF without plate. 3 of 16 fused angulated.
Burke DC and Tiong TS, 1975, Paraplegia	Retrospective review 175 cervical injuries txed traction, traction-manipulation, collar	Class III	2 of 14 UFD and 0 of 13 BFD failed nonoperative treatment.
Burke DC & Berryman D, 1971, J Bone Joint Surg Br	Retrospective review 76 facet dislocations 41 UFD, 35 BFD 41 manipulation 35 traction (3/35 failed manipulation) 3 fusion primarily	Class III	4 of 41 failed manipulation and 4 of remaining 37 had late instability. 0 of 32 failed traction.
Cheshire DJE, 1969, Paraplegia	Retrospective review 257 cervical injuries treated with traction or collar (33 excluded)	Class III	3 of 40 UFD and 2 of 35 BFD failed nonoperative treatment.

**EVIDENTIARY TABLE: Compressive flexion or vertical compression**

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
Fehlings et al, 1994, J Neurosurg	Retrospective study 44 cervical injuries Posterior fusion with plates	Class III	Complications in 6 of 17 including 1 residual kyphosis and 1 new kyphosis (reop).
Lieberman IH & Webb JK, 1994, J Bone Joint Surg Br	Retrospective study 41 cervical injuries Patients >65 years old	Class III	1 of 4 died. 2 in collar and 1 fused patient were stable.
Kiwerski JE, 1993, Inter Orthop	Retrospective "cross-over" study: 273 VC 1 <sup>st</sup> 70 traction 2 <sup>nd</sup> 203 anterior fusion	Class III	Fewer died and more recovered neurologic function when treated with surgery.
Aebi M et al, 1991, Spine	Retrospective study 22 cervical injuries Anterior corpectomy with plate	Class III	All 22 achieved stable fusion. 2 screw complications occurred.
Anderson PA et al, 1991, Spine	Prospective study 30 cervical injuries Post fusion with plate	Class III	All 9 achieved stable fusion, though 1 had late kyphosis.
Bucholz R and Cheung K, 1989, J Neurosurgery	Retrospective study 32 cervical injuries 19 VC, CF injuries	Class III	1 of 19 failed halo treatment. Pt failed post fusion w/ wire
Cabanela M & Ebersold MJ, 1988, Spine	Retrospective study 8 tear drop fractures Ant fusion with plate	Class III	All 8 achieved stable fusion with none developing kyphosis.
Lind B et al, 1988, Spine	Retrospective study 83 cervical injuries Halo treatment	Class III	2 of 19 were unstable. Drainage and loose pins were common.
Chan RC et al, 1983, J Neurosurg	Retrospective study 188 cervical injuries 150 subaxial w F/U Halo treatment	Class III	All 22 burst fxs and 17 of teardrop fxs achieved stable fusion.
Dorr LD et al, 1982, Spine	Retrospective study 117 cervical injuries 32 VC injuries	Class III	1 of 11 had graft displacement after ant fusion w/o plate.
Burke DC and Tiong TS, 1975, Paraplegia	Retrospective study 175 cervical injuries treated with traction, traction-manipulation, collar	Class III	1 of 46 failed nonoperative treatment.
Frankel H et al, 1973, Proceed Vet Admin Spinal Cord Injury Conf	Retrospective study 218 cervical injuries 45 Burst, 97 Teardrop Closed treatment	Class III	7 of 142 failed postural reduction. 103 had residual deformities.
Cheshire DJE, 1969, Paraplegia	Retrospective review 257 cervical injuries treated with traction or collar (33 excluded)	Class III	3 of 63 failed nonoperative treatment.
Beatson TR, 1963, J Bone Joint Surg Br	Retrospective study 59 cervical injuries All immobilized	Class III	All 16 were stable with immobilization.

## EVIDENTIARY TABLES: Extension

First Author Reference	Description of Study	Data Class	Conclusions
Lifeso RM & Colucci MA, 2000, Spine	Retrospective and prospective study 32 CE1 injuries (3 lost to F/U)	Class III	All 18 treated with brace failed (17 were unreduced). 9 of 11 healed with PCF, but 3 had stable kyphosis. 2 of 11 healed w/o reduction.
Lieberman IH & Webb JK, 1994, J Bone Joint Surg Br	Retrospective study 41 cervical injuries Patients >65 years old	Class III	1 of 3 healed with collar and 1 of 3 healed with surgery.
Anderson PA et al, 1991, Spine	Prospective study 30 cervical injuries Posterior fusion with plates	Class III	All 30 healed but 1 had screw loosening.
Rockswold et al, 1990, J Trauma	Retrospective study 140 cervical injuries txed halo or surgery	Class III	All 3 treated with halo healed. All 3 treated with surgery healed.
Bucholz R and Cheung K, 1989, J Neurosurgery	Retrospective study 32 cervical injuries 12 extension injuries	Class III	1 of 12 failed halo treatment. 1 pt stable post fusion w/ wire
Dorr LD et al, 1982, Spine	Retrospective study 117 cervical injuries 45 extension injuries	Class III	40 of 45 were treated with brace. Of all cervical injuries treated with brace, 5 of 86 failed.
Burke DC and Tiong TS, 1975, Paraplegia	Retrospective review 175 cervical injuries txed traction, traction-manipulation, collar (30 excluded)	Class III	All 45 healed without surgery.

**EVIDENTIARY TABLE: Subluxation**

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
Fehlings et al, 1994, J Neurosurg	Retrospective study 44 cervical injuries Posterior fusion with plates	Class III	2 of 5 lost reduction, including 1 who died.
Anderson PA et al, 1991, Spine	Prospective study 30 cervical injuries Posterior fusion with plates	Class III	19 of 19 healed with fusion. 8 of 19 had complications, including 2 with increase kyphosis & 3 requiring additional levels to be fused.
Rockswold et al, 1990, J Trauma	Retrospective study 140 cervical injuries treated with halo or surgery	Class III	12 of 26 failed halo treatment. 2 of 10 failed surgical treatment.
Bucholz R and Cheung K, 1989, J Neurosurgery	Retrospective study 32 cervical injuries 6 subluxation injuries	Class III	2 of 6 failed halo treatment. 1 worse postop, unreported if DF or sublux patient
Cooper PR et al, 1979, J Neurosurg	Retrospective study 33 cervical injuries treated with halo	Class III	1 of 3 failed halo treatment.
Burke DC and Tiong TS, 1975, Paraplegia	Retrospective study 175 cervical injuries treated with traction, traction-manipulation, collar (30 excluded)	Class III	1 of 14 failed nonoperative treatment.
Cheshire DJE, 1969, Paraplegia	Retrospective study 257 cervical injuries treated with traction or collar (33 excluded)	Class III	4 of 19 failed nonoperative treatment.
Beatson TR, 1963, J Bone Joint Surg Br	Retrospective study 59 cervical injuries (3 excluded)	Class III	8 of 22 with <50% subluxation reduced. 2 of 14 remaining had surgery. 13 of 18 with >50% subluxation reduced. 5 of 5 remaining had surgery.

**EVIDENTIARY TABLE: Ankylosing Spondylitis (AS)**

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
Weinstein et al, 1982, J Neurosurg	Retrospective study 13 AS 7 traumatic cervical 6 quadriplegic 2 central cords w/o fx	Class III	2 treated with traction died of pneumonia. 2 treated with traction/brace healed. 1 worse halo treated surgically. 1 lami/fusion worse, 1 lami/fusion had pseudoarthrosis.
Bohlman HH, 1979, J Bone Joint Surg	Retrospective study 300 cervical injuries 8 AS	Class III	5 of 8 patients died. 2 healed after brace treatment and 1 after laminectomy.
Cheshire DJE, 1969, Paraplegia	Retrospective study 257 cervical injuries 1 AS	Class III	1 C5-C6 extension injury healed with surgical fusion
Grisolia et al, 1967, J Bone Joint Surg Am	Retrospective study 6 AS	Class III	3 of 4 healed with brace +/- traction. 2 with laminectomy and PCF died of PE.

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## MANAGEMENT OF ACUTE CENTRAL CERVICAL SPINAL CORD INJURIES

### RECOMMENDATIONS

Standards: There is insufficient evidence to support treatment standards.

Guidelines: There is insufficient evidence to support treatment standards.

Options:

- Intensive care unit (or other monitored setting) management of patients with acute central cervical spinal cord injuries (ACCSCI), particularly patients with severe neurological deficits, is recommended.
- Medical management including cardiac, hemodynamic and respiratory monitoring, and maintenance of mean arterial blood pressure at 85 – 90 mm Hg for the first week after injury to improve spinal cord perfusion, is recommended.
- Early reduction of fracture dislocation injuries is recommended.
- Surgical decompression of the compressed spinal cord, particularly if the compression is focal and anterior, is recommended.

### RATIONALE

Central spinal cord injuries are among the most common, well-recognized spinal cord injury patterns identified in neurologically injured patients after acute trauma. Originally described by Richard Schneider in 1954, this pattern of neurologically incomplete spinal cord injury is characterized by “disproportionately more motor impairment of the upper than of the lower extremities, bladder dysfunction and varying degrees of sensory loss below the level of the lesion”.(19) It has been associated with hyperextension injuries of the cervical spine, even without apparent damage to the bony spine, but has also been described in association with vertebral body fractures and fracture-dislocation injuries. The natural history of acute central

cervical spinal cord injuries indicates gradual recovery of neurological function for most patients, albeit usually incomplete and related to the severity of the original injury and the age of the patient.(4,12,15,17-19,21) The role of surgery and its timing for patients with acute central spinal cord injuries without fracture compression or dislocation injuries are the subjects of considerable debate.(3,5-8,18,19) The optimal management of patients who have sustained acute central cervical spinal cord injuries is the subject of these recommendations.

## **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”: “central cord syndrome” and “incomplete cervical spinal cord injury”. Approximately 1450 citations were acquired. Non English-language citations were excluded. Titles and abstracts of the remaining publications were reviewed and relevant articles were selected to develop the guidelines. We focused on the specific issues of the natural history, medical management and surgical treatment of acute human central cervical spinal cord injuries. These efforts resulted in 13 articles (all Class III studies) specifically describing management and outcomes of patients with central cervical spinal cord injuries. The bibliography includes several articles on Magnetic Resonance (MR) imaging of central cervical spinal cord injuries, multiple articles (all Class III studies) describing series of acute SCI patients, the majority of whom had incomplete cervical spinal cord injuries and several general review articles that address issues of acute spinal cord injuries, including pathophysiology and treatment. The 13 case series describing the management of patients with acute central cervical spinal cord injuries are summarized in Evidentiary Table format.

## SCIENTIFIC FOUNDATION

In 1951, Schneider described two patients with acute neurologically incomplete cervical spinal cord injuries for whom he suggested that early operation was indicated.(20) Both patients presented with sudden loss of motor function in the distal upper extremities, in the torso and lower extremities, but had preservation of touch and vibration sense following trauma. Both patients had anterior spinal cord compression from acute traumatic cervical disc herniations (one had an associated vertebral endplate fracture). The diagnosis and anatomic localization were based on the clinical examination. Both patients made incomplete but significant neurological recoveries following delayed surgical decompression via laminectomy, dentate ligament sectioning and transdural discectomy. Three years later Schneider, Cherry and Pantek described eight patients they managed along with six other patients culled from the available literature.(19) All but two of these patients presented with disproportionately more motor impairment in the upper extremities than in the lower extremities, bladder dysfunction with retention, and varying degrees of sensory loss below the level of the lesion. Two of the six patients identified in the literature review had complete motor injuries in both the upper and lower extremities, with some preservation of sensory function below the level of injury. These incomplete neurological deficits were related to acute traumatic central cervical spinal cord injuries, usually, but not exclusively, as a result of hyperextension of the head and neck relative to the torso. In several patients there was no damage to the bony spine. In these instances it was presumed that hypertrophic changes (spurs, ridges, thickened ligaments) within the spinal canal caused anterior and posterior cord compression in the position of hyperextension, resulting in injury to the central substance of the cervical spinal cord. Other patients had cervical compression fractures

or fracture-dislocation injuries of the cervical spine that contributed to the central spinal cord injury.

The authors operated on the first two of the eight patients they treated with this disorder. Both had central cervical spinal cord injuries without bony damage or displacement. Both were treated in delayed fashion via laminectomy with sectioning of the dentate ligaments followed by transdural exploration anterior to the cervical spinal cord. In both cases anterior bony osteophytes were identified but were not removed. Patient #1 was quadriplegic postoperatively. Patient #2 was neither better nor worse following surgery. Six additional consecutive patients were managed without surgical decompression, (#7 underwent delayed dorsal cervical fusion in treatment of fracture instability). Five of six patients they managed expectantly, (patient #4 had progression of neurological deficits despite immobilization and ultimately died three weeks after injury), and three of six patients from the literature improved without surgery, (75%). The majority experienced permanent loss of hand function and strength. One of six patients from their series and three of six from the literature died without neurological recovery (25%). Conversely, one of two patients treated surgically was immediately neurologically worse (50%), the second made a progressive albeit incomplete recovery over time, (50%), much like that observed in comparative patients managed without surgery. It was on the basis of this early experience that Schneider and colleagues determined that the prognosis following acute central cervical spinal cord injury was reasonably good. Surgery for these patients they concluded was “contraindicated” and “in fact known to harm... rather than improve them”.

In 1958 Schneider, Thompson and Bebin added observations on 12 additional patients they managed with acute central cervical spinal cord injuries.(18) One patient died of pneumonia without neurological recovery, one patient had a full neurological recovery, the

remaining ten improved compared to their initial post-injury neurological status, but were profoundly impaired at last follow-up. They noted two distinct age groups with acute central cervical spinal cord injuries. They described an older group of patients (mean age 59 years) without bony vertebral damage but with hypertrophic changes compromising the cervical spinal canal, and a younger group (mean age 31 years) with fracture or fracture dislocation injuries of the cervical spine. They reported that central cord edema, venous congestion and ischemia were components of the pathophysiology of this unique injury type. They advocated expectant management, including closed reduction with skeletal traction (four patients), for all patients with this syndrome, despite important, near-complete neurological recovery in a 17-year-old patient after operative reduction and decompression of a unilateral facet dislocation injury within 13 hours of injury.

Schneider's collective reported personal experience with the management of patients with acute central cervical cord injuries to date numbered 20 patients.(18) Of the 20, 17 were managed medically: two died without improvement, 14 improved but had profound residual deficits and one patient regained normal function. Three patients were treated with surgical decompression: one early (hours) and two late (weeks). The patient with early decompression improved dramatically. One late decompression patient neither improved nor worsened immediately after surgery but showed progressive long-term improvement, the other was quadriplegic after surgery. From this experience they concluded, "accurate diagnosis is stressed, with emphasis placed on the fact that operation is contraindicated, that the prognosis may be good and that should recovery occur it will follow a definite pattern". These suggestions have guided the care of acute central cervical spinal cord injury patients ever since.

In 1971 Bosch et al described observations made during their management of 42 patients with subacute central cervical spinal cord injuries they treated at a rehabilitation hospital and provided four month to 26 year follow-up.(2) At admission 19% were independent ambulators, 14% were partial ambulators and 67% could not ambulate. Twenty six per cent had functional hands upon admission. Upon discharge 57% had functional ambulation skills, 20% were partial ambulators and 42% had functional hands. Bladder control improved from 17% upon admission to 53% upon discharge. A similar improvement in bowel control was documented. Importantly, these authors noted late deterioration in 24% of patients who showed initial improvements in neurological function after central cervical spinal cord injury. Ten of 42 patients (24%) experienced the late neurological sequelae of “chronic central cord syndrome” and lost ambulation, hand and bladder control skills in long-term follow-up. The authors concluded that at least some return of neurological function in the immediate post-injury period could be expected in about 75% of cases, with 56% of patients regaining functional hands. Long term, only 59% of patients with central cervical spinal cord injuries they followed retained functional skills with conventional medical management.

In the same year, Turnbull presented his studies on the microvasculature of the human spinal cord and postulated as to the mechanisms of vascular insufficiency associated with various types of spinal cord displacement.(27) His work speaks to the anatomical basis of the pathophysiology of acute cervical central spinal cord injuries, particularly those which occur in older patients with underlying cervical spondylosis who sustain acute central SCI without bony vertebral injury. He found that as the cord becomes compressed, whether due to a mass lesion or progressive cervical spondylosis, it becomes flattened and widened. The vasculature of the cord is affected by cord distortion. Pial vessels become more tortuous. Arteries of the lateral columns

are elongated, narrowed and flattened. Branches from the central arteries that reach the gray matter run laterally and are similarly stretched lengthwise and are compressed from side to side. Turnbull reported that vessels chronically deformed by cervical spondylosis cannot respond to additional anteroposterior flattening of the cord as would normal arteries in a younger patient. “A little additional compression would pinch off side branches at their origins”. He concluded that mechanical distortion of the cord and its blood supply plays a major role in the pathophysiology of spinal disease and spinal cord injury. (26,27,28)

In 1977, Shrosbree reported 99 patients with acute central cervical spinal cord injuries managed at a South African Spinal Cord Injury Centre, the majority of whom were admitted within 72 hours of injury.(21) All patients were treated conservatively. Fracture/injury reduction was accomplished via closed means in 92% of patients with dislocation, either by traction or by reduction under anesthesia, within 72 hours of admission. Two age groups of patients were identified. Younger patients (21 to 50 years) had flexion rotation injuries and a higher incidence of dislocation injuries. Older patients (50 years and above) were more likely to have hyperextension injuries superimposed on preexisting cervical spondylosis. Outcome was related to the severity of the initial neurological deficit. Only five of 23 patients (22%) with severe motor deficits became independent ambulators. All of these patients had residual deficits in the hands. The author summarized by noting that “early reduction may well be a factor in promoting more favorable neurological recovery” among patients with facet dislocation injuries, but gave no data to support his claim.

In 1977, Maroon reported that “burning hands”, severe dysesthesias in the hands and fingers following trauma despite normal motor function may indicate acute central spinal cord injury.(13) He described two football players with dysesthetic symptoms in the hands referable

to modest injury to the central cervical spinal cord and warned physicians, trainers and coaches of the importance of this syndrome.

In 1980, Brodkey and associates revisited the management of the acute central cervical spinal cord injury syndrome.<sup>(5)</sup> They provided operative treatment to seven patients with traumatic central cervical spinal cord injuries within 18 to 45 days after acute injury that had profound residual neurological deficits after attentive medical treatment. These patients all had significant defects on myelography. Four patients underwent anterior cervical discectomy with fusion (ACDF), one was treated with multilevel laminectomy, one had multilevel ACDF and one received multilevel laminectomy followed by delayed (four years) multilevel ACDF. All patients had accelerated neurological recoveries following each surgical procedure, even the patient who deteriorated years after laminectomy and required late multilevel ACDF. Three patients returned to normal after severe injuries that persisted until surgical decompression. The authors concluded that cord compression does play an important role in the pathophysiology of central cord syndrome and that when present, in the setting of a stable poor neurological condition following injury, that decompression of the spinal cord may be of benefit.

Bose et al retrospectively analyzed their management of 28 patients with acute central cervical spinal cord injuries.<sup>(3)</sup> In their 1984 report, they noted significantly improved motor scores at time of discharge in patients managed with combined medical therapy and surgery, compared to those managed medically alone. All were treated aggressively in the ICU setting. Surgical patients had myelographic evidence of cord compression or evidence of cervical spinal instability. While selection bias (surgical patients had cord compression and/or instability or subluxation), and several other study flaws precluded direct comparison between the groups, the authors noted that no surgical patient worsened as a result of surgery and all improved

neurologically, several substantially. They argued that decompression of the compressed spinal cord in patients with acute central cervical spinal cord injury syndrome may be of benefit in selected patients.

Merriam et al, Roth et al, Bridle et al and Newey, Sen and Fraser each described the late outcomes of individual series of selected groups of patients following central cervical spinal cord injury.(4,12,15,17) All four groups of investigators noted marked heterogeneity among patients in the central cervical spinal cord injury population. All patients were managed medically. Most patients improved somewhat over time, with more recovery in the lower extremities than in the upper extremities. All concluded that outcome was in general good for patients less than 70 years of age; the final neurological result was influenced by patient age, particularly age over 70 years, and the degree of initial neurological impairment. Hand function impairment was the most common long-term disability, even among patients with a “good” outcome. Only Merriam et al made reference to surgical treatment, involving 30 of 77 patients in their series, presumably to provide spinal stabilization and fusion.(12) No association between surgical management and outcome was discerned.

Chen and colleagues reported their experience with 114 patients with acute and chronic traumatic central cervical cord syndrome.(7) Twenty-eight patients were managed with surgical treatment, 86 were managed medically. The authors did not randomize patients to one treatment group or another. Selection criteria for surgical decompression included failure to improve with medical therapy or deterioration in neurological function despite medical treatment with radiographic (MRI or CT/myelography) evidence of focal cord compression, or gross instability of the spine. They operated on three patients late (8, 12 and 24 months post-injury) for “chronic” central cord syndrome. Their 1997 retrospective review found that younger patients had better

long-term results than did older patients (in both management groups), and that surgical decompression was associated with more rapid and complete motor improvement compared to patients managed medically, even if operated late after injury. Both management groups had similar outcomes over time with respect to lower extremity and bladder function. Patients selected for surgery had more rapid and more complete recovery of function, particularly in the upper extremities. The authors noted that patients with stenosis at multiple levels managed conservatively had a poorer prognosis and a relatively higher chance to develop late myelopathy. The authors did not describe the outcome of similar patients with multi-level stenosis managed with operative decompression.

In 1998 Chen et al described the management of 37 patients with pre-existing cervical spondylosis who sustained acute incomplete neurological cervical spinal cord injuries after trauma.<sup>(6)</sup> Many of these patients had acute central spinal cord injuries. No patient sustained a bony vertebral column injury. In their retrospective review, patients were treated with surgical decompression if they did not improve more than one motor grade within nine days of injury (range three to 14 days). Patients were studied with MRI to document cord compression and/or signal change within the spinal cord. In total, 16 patients underwent surgical decompression, nine anteriorly and seven posteriorly. Twenty-one patients were managed medically. Thirteen of 16 surgical patients (81%) improved “remarkably” immediately after surgery. Thirteen of 21 patients managed medically (62%) improved to the same degree over time. As with surgical patients, patients with cervical stenosis over more than three vertebral levels fared less well than did patients with focal compression or with stenosis over three vertebral levels or fewer. There were no reported differences in outcome between patients in the two groups at two-year follow-up. The authors concluded that surgical decompression might be associated with more rapid

neurological improvement, early mobilization and shorter periods of hospitalization and rehabilitation. They consider MRI as the imaging modality of choice to assess the spinal cord in patients with acute central cervical spinal cord injuries, a conclusion consistent with those of other investigators of the role of MRI in the assessment of patients with spinal cord injuries. (9,14,16,25)

In 2000, Dai and Jia described their experience with 24 patients with acute traumatic disc herniation as the cause of acute central cervical spinal cord injuries.(8) Acute disc herniation was confirmed with pre-operative MRI imaging. The authors provided a retrospective assessment of patients operated upon anteriorly (ACDF without internal fixation) for cord decompression and spinal stabilization. The timing of surgery relative to injury was not described. They noted an inverse correlation between rate of recovery and age and found that patients with fracture dislocation injuries with acute disc herniation were more impaired pre-operatively and fared less well than patients without fracture dislocation injuries at late follow-up. They reported that surgical decompression, stabilization and fusion was successful in all patients and described marked improvement in neurological function in most patients treated.

Contemporary reviews confirm early reports that most patients with incomplete cervical spinal cord injuries meeting the clinical neurological criteria of acute central spinal cord injury will show neurological improvement over time.(2,12,15,17-19,21) Some patients with these injuries will die and many will remain profoundly impaired at late follow-up. These patients in general are older, have spinal cord injuries without bony vertebral injury and have medical comorbidity, or are younger but have fracture dislocation injuries as a cause of their neurological deficits. A large portion of patients will regain ambulation skills over time but will not have useful hands. A smaller portion of patients will demonstrate significant neurological recovery

and regain hand function. These patients are typically younger, do not have fracture dislocation injuries and have less severe neurological deficits at the outset. Up to 24 percent of patients managed non-operatively will improve early but decline again years later (“chronic central cord injury syndrome”).(2)

Surgery for decompression of the spinal cord in patients with acute central cervical spinal cord injuries has been denounced based on Schneider’s early poor experience with a single operated patient.(18,19) That patient, quadriplegic after dorsal cervical exploration and decompression, experienced significant manipulation of the injured cord during the process of dentate ligament sectioning and transdural anterior cord exploration, a procedure unlikely to be performed in similar fashion today.(19) The same group had a rewarding experience with early, (13 hours after injury), surgical decompression and facet fracture reduction in a 17-year-old male with profound early central cord neurological deficits.(18) Multiple other authors, including three contemporary series of patients with this disorder, have described good to excellent outcomes without neurological complications for surgical decompression of patients with spinal cord compression, particularly focal anterior cord compression.(3,5-8) However, no study to date has provided a randomized direct comparison of surgical patients to similar patients managed without surgery. Nor has any study adequately assessed the potential role of dorsal spinal decompression for multi-level cervical cord compression in patients with this disorder, particularly those with acute central cervical cord syndrome without bony vertebral damage. Surgery may have a role in the management of patients with acute central cervical spinal cord injury, but as yet that role has not been accurately defined by scientific study.

Schneider’s conclusion that central cord edema, venous congestion and ischemia were important components of the pathophysiology of these injuries, combined with Turnbull’s

hypothesis that vascular compression and distortion due to antero-posterior flattening of the cord plays a major role in the pathophysiology of cord injury, suggest several potential opportunities for treatment.(18,26-28) The compression of the cord and the distortion and compression of its blood supply might be relieved by surgical decompression. Ischemia of the cord, either due to the primary injury or secondary events might be improved with augmentation of spinal cord perfusion. While Turnbull did not offer specific strategies, he did offer anatomical and pathophysiological rationale for the potential of maintenance of spinal cord perfusion pressures and cervical cord decompression for patients who sustain an acute central cervical cord injury, particularly those with pre-existing cervical spondylosis.(26-28) Maintenance or increases in systemic blood pressure may improve perfusion to the injured, distorted spinal cord.(1,3,10,11,22,23,29) Several contemporary series of spinal cord injured patients treated with aggressive medical management with maintenance of mean arterial blood pressure to high normal ranges (85 mm Hg to 90 mm Hg) have suggested improved neurological outcomes with this management plan.(10,11,24,29-31) Decompression of the cord has the potential to eliminate both cord compression and vascular compression and distortion.(3,5-8,30) Either or both of these treatment strategies may improve spinal cord blood flow in the acute central cervical spinal cord injury setting, which could translate into preservation of neurological tissue and recovery of neurological function. The benefit may or may not be realized at the site of primary injury, but rather at vulnerable adjacent spinal cord levels fed by sulcal and collateral arteries that pass through the injury site but supply the cord rostral and caudal to the site of injury. (1,22,23,26,28).

## **SUMMARY**

The ideal management strategy for patients with acute central cervical spinal cord injuries appears to be multifaceted. As Schneider insisted years ago, a rapid, accurate diagnosis is essential. A detailed clinical examination, cervical spinal radiographs to assess vertebral column injury (see Radiographic Clearance in Symptomatic Patient recommendations) and MR assessment of the cervical spinal cord for intrinsic injury and/or compression will accomplish this goal. Many of these patients may require management in the ICU setting (see ICU Monitored Setting recommendations) for monitoring and treatment of cardiac, pulmonary and blood pressure disturbances. Blood pressure augmentation to MAP levels of 85 mm Hg to 90 mm Hg may be of benefit (see Blood Pressure Management recommendations). Early reduction of fracture or fracture dislocation injuries should be accomplished (see Subaxial Cervical Spinal Injuries recommendations). Administration of pharmacological agents may be of benefit according to specific parameters (see Pharmacological Therapy recommendations). Surgical decompression of the compressed spinal cord, particularly if the compression is focal, anterior and is approached anteriorly, appears to be of benefit in selected patients.

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

A prospective, controlled, randomized investigation of patients with acute central cervical spinal cord injuries treated with aggressive medical therapy alone (ICU management, blood pressure augmentation, closed fracture dislocation reduction), compared to those managed with aggressive medical therapy and early surgical decompression of the spinal cord is needed.

**EVIDENTIARY TABLES:**

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
Dai L and Lianshun J, 2000, <i>Spine</i>	Retrospective review of 24 patients with acute disc herniation as cause of ACCSCI treated with ACDF	Class III	Disc herniation common cause. Surgery successful in all patients, more rapid improvement. Poor outcome with fracture dislocation injuries.
Newey ML et al, 2000, <i>J Bone and Joint Surg (Br)</i>	Retrospective review of 32 patients with ACCSCI managed conservatively.	Class III	Improvement seen in most patients over time. Older patients had worse outcome.
Chen TY et al, 1998, <i>Spine</i>	Retrospective review of 37 patients with ACSI with pre-existing spondylosis. Many with central cord injury pattern. MR assessment of compression, cord injury. 16 managed with surgical decompression, 21 medically.	Class III	MR modality of choice to image cord compression/injury. Surgical decompression associated with more rapid improvement, shorter hospital and rehab.stay. No difference in outcome at two year follow-up.
Chen TY et al, 1997, <i>Surg Neurol</i>	Retrospective review of 114 patients with acute or chronic CCSCI. 28 patients managed with surgery (3 chronic patients), 86 medically. No randomization.	Class III	Surgery associated with more rapid and complete recovery, particularly in upper extremities, compared to similar patients managed medically. Patients with long segment stenosis had poor prognosis.
Bridle MJ, et al, 1990, <i>Paraplegia</i>	Random late assessment of 18 patients with ACCSCI.	Class III	Most patients improve over time, although most with long-term deficits, pain and dysfunction.
Roth EJ et al, 1990, <i>Arch Phys Med Rehabil</i>	Retrospective review of 81 rehab patients after ACCSCI.	Class III	Two age groups of patients, marked heterogeneity. In general most patients will improve over time. Outcome related to age, severity of initial injury.
Merriam WF et al, 1986, <i>J Bone and Joint Surg (Br)</i>	Retrospective review of 77 patients with ACCSCI. No patient with surgical decompression, 30 underwent late stabilization and fusion.	Class III	Marked variation among patients and injury patterns. Most improve. Outcome related to age, severity of initial injury.

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
Bose B et al, 1984, <i>Neurosurg</i>	Retrospective review of 28 patients with ACCSCI, 14 managed with aggressive medical therapy, 14 with medical therapy and surgical treatment. No randomization. Follow-up at time of discharge.	Class III	No patient worse with treatment, medical or surgical. Surgery provided more rapid, more complete recovery at time of discharge.
Brodkey JS et al, 1980, <i>Surg Neurol</i>	Retrospective review of seven patients with ACCSCI operated upon late after injury. All had stable, profound deficits and myelographic evidence of cord compression.	Class III	All had accelerated neurological improvement after surgery. Three normal at late follow-up. Surgery of benefit in select patients with persistent deficits and evidence of cord compression.
Shrosbree RD, 1977, <i>Paraplegia</i>	Retrospective review with late follow-up of 99 patients with ACCSCI managed conservatively.	Class III	Two groups identified. Younger patients with flexion rotation injuries. Older patients with hyperextension injuries. Outcome related to severity of initial injury.
Bosch A et al, 1971, <i>JAMA</i>	Retrospective review and long-term follow-up of 42 patients with ACCSCI managed conservatively.	Class III	Most patients improve over time. 75% will regain ambulatory skills, 56% regain functional hands. ten of 42 patients had late deterioration after initial gains ("chronic central cord syndrome").
Schneider RC et al, 1958, <i>J Neurol Neurosurg Psychiat</i>	Retrospective review of 12 additional patients with ACCSCI. Eleven managed expectantly, one managed with surgical decompression 13 hours after injury.	Class III	Two age groups of patients. Young patients with fracture dislocation injuries. Older patients with hyperextension injuries often without bony vertebral damage. Most patients improve. Expectant management is ideal treatment.
Schneider et al, 1954, <i>J Neurosurg</i>	Retrospective review (and first description) of eight patients with ACCSCI they managed (6 expectantly, two surgically) and discussion of six cases from literature.	Class III	Most patients with acute central cervical spinal cord injuries will improve with time and expectant management. Injury and its recovery follows specific pattern. Surgery contraindicated for this injury.

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# MANAGEMENT OF VERTEBRAL ARTERY INJURIES FOLLOWING NON-PENETRATING CERVICAL TRAUMA

## RECOMMENDATIONS

### Diagnostic

Standards: There is insufficient evidence to recommend diagnostic standards.

Guidelines: There is insufficient evidence to recommend diagnostic guidelines.

Options: Conventional angiography or magnetic resonance angiography (MRA) is recommended for the diagnosis of vertebral artery injury (VAI) after nonpenetrating cervical trauma in patients who have complete cervical spinal cord injuries, fracture through the foramen transversarium, facet dislocation, and/or vertebral subluxation.

### Treatment

Standards: There is insufficient evidence to recommend treatment standards.

Guidelines: There is insufficient evidence to recommend treatment guidelines.

Options:

- Anticoagulation with intravenous heparin is recommended for patients with vertebral artery injury who have evidence of posterior circulation stroke.
- Either observation or treatment with anticoagulation in patients with vertebral artery injuries and evidence of posterior circulation ischemia is recommended.
- Observation in patients with vertebral artery injuries and no evidence of posterior circulation ischemia is recommended.

## RATIONALE

The association of cerebrovascular insufficiency and cervical fracture was first described by Suechting et al in a patient with Wallenburg's syndrome occurring four days after a C5-C6

fracture-dislocation injury.(13) Although Schneider et al implicated vertebral artery injury at the site of cervical fracture dislocation as a cause of posterior circulation cerebral ischemia (9), Gurdjian et al suggested that unilateral vertebral artery occlusions might be asymptomatic.(4) Subsequent articles described larger series of patients with asymptomatic vertebral artery injuries (VAI) after blunt cervical spinal trauma.(2,18) However, Biffl et al, in the largest prospective series consisting of 38 patients with VAI diagnosed by angiography, reported more frequent strokes in patients not treated initially with intravenous heparin anticoagulation despite an asymptomatic VAI.(1) Fractures through the foramen transversarium, facet fracture-dislocation, or vertebral subluxation are almost always seen in patients with VAI.(1,2,3,5,17,18,19) A cadaveric study demonstrated progressive vertebral occlusion with greater degrees of flexion-distraction injury, confirming this clinical observation.(11) In order to develop diagnostic and treatment recommendations for VAI after blunt cervical trauma, an analysis of the articles examining its management is undertaken in this report. Specific questions that were addressed include: the clinical and radiographic criteria used to prompt diagnostic evaluation, appropriate diagnostic tests for identifying VAI, and the management of VAI (observation versus anticoagulation with heparin).

## **SEARCH CRITERIA**

A National Library of Medicine computerized literature search of publications from 1966 to 2001 was performed using the following headings: vertebral artery, cervical vertebrae, dislocation, and wounds and injuries. An exploded search of the headings led to 6,447, 15,667, 24,174 and 459,759 citations respectively. The first two headings were combined, and a subset of 61 citations was also contained in the third heading. A subset of 239 citations was also

contained in the fourth heading. Abstracts were reviewed and only articles in English containing three or more human subjects with vertebral artery injury after blunt cervical trauma were included. Fourteen articles, including eight articles that were prospective studies (1,2,3,5,10,16,17,18), met these selection criteria, and provided data on 122 patients for this report. These articles are summarized in Evidentiary Table format.

## **SCIENTIFIC FOUNDATION**

### **Diagnosis**

The diagnosis of VAI can be made with a variety of radiographic studies. Angiography is the traditional imaging technique utilized to diagnose VAI and was used for all patients in seven of the studies reviewed (1,5,7,14,15,18,19) and in combination with other modalities in two additional studies.(6,8) Three studies prospectively applied conventional angiography to patients sustaining nonpenetrating cervical trauma that met certain inclusion criteria.(1,5,18) Similarly, three studies prospectively applied magnetic resonance angiography to patients sustaining nonpenetrating cervical trauma that met certain inclusion criteria.(2,16,17)

Biffel et al reported the largest prospective study using angiography, selecting patients from 7205 blunt trauma victims using clinical and radiographic criteria.(1) “Symptomatic” patients were selected for angiography if they had facial hemorrhage (bleeding from mouth, nose, ears), cervical bruit (in those younger than 50 years of age), expanding cervical hematoma, cerebral infarction by computed tomography (CT), or lateralizing neurological deficit. “Asymptomatic” patients were selected for angiography if they had cervical hyperextension/rotation or hyperflexion injuries, closed head injury with diffuse axonal injury, near hanging, seat belt or other soft tissue injuries to the neck, basilar skull fractures extending

into the carotid canal, and cervical vertebral body fractures or distraction injuries. Between 350 and 400 angiograms were performed, identifying 38 patients with VAI. However, neither the exact number of angiograms performed nor the number of patients meeting the various criteria without VAI were reported. As a result, sensitivity, specificity, positive predictive value, and negative predictive value of the selection criteria could not be determined. Cervical spine injuries were observed in 27 of 38 patients with VAI, including fractures through the foramen transversarium in four, dislocations in six, vertebral subluxations in two, and more than one of these injuries in two. Twenty-nine patients had unilateral VAI (18 left, 11 right); nine had bilateral VAI. A vascular injury scale was used to stratify patients into five categories:

- Grade I - arterial dissections less than 25% luminal narrowing,
- Grade II - arterial dissections with more than 25% luminal narrowing,
- Grade III - pseudoaneurysm of the vertebral artery,
- Grade IV - occlusion of the vertebral artery,
- Grade V - vertebral artery transection.

Seven patients died; five had bilateral VAI (Grade I), while two had unilateral VAI (one Grade I, one Grade IV). Three patients with either no neurological deficit or mild deficit had bilateral VAI. The authors concluded that stroke incidence and neurological outcome appeared independent of the grade of vertebral artery injury.

Another prospective study by Willis et al identified 30 patients with midcervical fractures and/or dislocation for angiography.(18) However, only 26 patients meeting the criteria agreed to proceed with angiography. Twelve patients sustained VAI demonstrated by angiography (six left occlusion, three right occlusion, one left intimal flap, one left pseudoaneurysm, one left dissection). The authors provided sufficient data regarding the presence of foramen

transversarium fracture, facet dislocation, and subluxation to determine the utility of these radiographic findings in identifying patients with VAI. The calculated sensitivity of foramen transversarium fracture as a criterion for identifying VAI in this study was 58%. The specificity was 36%. The positive predictive value of foramen transversarium fracture was 44%. The negative predictive value was 50%. The calculated sensitivity of facet dislocation as a criterion for identifying VAI was 42%. The specificity was 57%. The positive predictive value of facet dislocation was 45%; the negative predictive value was 53%. The calculated sensitivity of subluxation as a criterion for identifying VAI was 67%; the specificity was 29%. The positive predictive value of subluxation was 80%; the negative predictive value was 50%. Any combination of foramen transversarium fracture, facet dislocation, and/or vertebral subluxation revealed a calculated sensitivity for identifying VAI of 92% and a specificity of 0%. The positive predictive value of the presence of any of the three criteria and VAI was 44%. The negative predictive value was 0%.

A third prospective study by Louw et al examined 12 consecutive patients with cervical spine facet dislocations with digital subtraction angiography.(5) Five of seven patients with bilateral facet dislocations had vertebral artery occlusion (one bilaterally), and four of five patients with unilateral facet dislocations had unilateral vertebral artery occlusion. Angiography was not performed in blunt cervical trauma patients without facet dislocation. In a retrospective study by Woodring et al (19), seven of eight patients with transverse process fractures undergoing angiography had VAI (two right occlusions, two left occlusions, two right dissections, one left dissection). Seventy-eight percent of transverse process fractures extended into the foramen transversarium. Angiography was not performed in 44 other patients with transverse process fractures. Alternatively, magnetic resonance angiography (MRA) has been

used to diagnose VAI noninvasively. Weller et al prospectively examined twelve patients with nonpenetrating cervical trauma who sustained fractures through the foramen transversarium.(17) Three patients had unilateral vertebral artery occlusion and one had focal narrowing, all at the site of fracture. MRA was not performed on the twenty-six patients without these fractures. Giacobetti et al prospectively evaluated all patients admitted with cervical spine injuries with MRA.(3) Twelve of 61 patients had vertebral artery occlusion demonstrated by MRA and all injuries were unilateral (six left, six right). Although seven of twelve patients with VAI had flexion-distraction injuries with facet dislocations, the types of cervical spinal injuries sustained by the 49 patients with normal MRA were not reported. Since none of these four articles provided sufficient information regarding the types of injury and results of vertebral artery imaging in the entire population of patients studied, sensitivity, specificity, positive predictive value, and negative predictive value of the injury types could not be determined. (3,5,17,19)

Friedman et al prospectively examined 37 patients admitted with “major” blunt cervical spine injuries using MRA and compared these patients with a size-matched control group of patients without a history of cervical trauma.(2) Nine patients had VAI (six unilateral occlusion, two narrow, one bilateral injury). Both vertebral arteries were visualized in all 37 control subjects. Complete spinal cord injuries were observed in 12 of 37 patients with cervical trauma, six of whom had VAI ( $p < 0.02$ ; chi-square test). More than three millimeters of subluxation was observed in 13 of 37 patients, five of whom had VAI ( $p < 0.14$ ; chi-square test). The calculated sensitivity of complete spinal cord injury as a criterion for identifying VAI in this study was 67%, whereas the specificity was 79%. The positive predictive value of complete spinal cord injury was 50%. The negative predictive value was 88%. The calculated sensitivity of

subluxation as a criterion for identifying VAI was 56%. The specificity was 71%. The positive predictive value of subluxation was 38%, whereas the negative predictive value was 83%.

Other diagnostic modalities have also been used to identify of VAI. CT with intravenous contrast demonstrated a unilateral vertebral artery occlusion in one patient with a Jefferson fracture, which was subsequently confirmed by angiography.(12) Duplex sonography has also been used to diagnose of VAI.(8,10,14) Angiography has occasionally been used to confirm the results of MRA or ultrasonography, but there has not been a study comparing MRA or ultrasonography with angiography in the diagnosis of VAI.

## **Treatment**

After diagnosis of VAI, treatment options examined by the various studies have included observation alone or anticoagulation with either intravenous heparin or oral antiplatelet agents. Some authors treated asymptomatic patients (1,2,18,19); others did not treat symptomatic patients. (5,7)

Several articles retrospectively identified patients with neurological complications of VAI. Schellinger et al described four patients with VAI who had delayed onset of neurological dysfunction.(8) Two patients awoke after surgery with altered consciousness and posterior circulation stroke. One of these patients with vertebrobasilar occlusion diagnosed by ultrasound died; the other with vertebral artery dissection and pseudoaneurysm improved. Neither patient was anticoagulated. Two other patients developed delayed symptoms; one 12 hours after injury, the other three weeks after injury. The first patient died from basilar artery thrombosis confirmed by CT. The second patient developed vertigo and nystagmus due to a dissection diagnosed by ultrasound and angiography. This patient was successfully treated with

intravenous heparin anticoagulation and recovered within several weeks. Thibodeaux et al reported one patient who incurred immediate blindness after left vertebral artery dissection.(14) No infarction was seen on CT. Treatment included three months of anticoagulation with sustained improvement at four-year follow-up. A second patient with ataxia and dizziness two days after VAI had occipital infarction by CT and bilateral vertebral artery dissections, recovered with six months of anticoagulation therapy. One patient with dissection remained asymptomatic without anticoagulation. Prahbu et al reported three patients of five patients with VAI who experienced sudden loss of consciousness three hours after injury.(6) All were anticoagulated. One patient with vertebrobasilar thrombosis died; one patient with bilateral vertebral occlusion improved. The third patient with vertebral artery stenosis improved, although anticoagulation was stopped several days later secondary to a rectus sheath hematoma. Two patients with asymptomatic vertebral artery occlusion were not treated and remained asymptomatic. Tulyapronchote et al reported three patients with delayed onset of symptoms two weeks to three months after VAI, including syncope, visual disturbance, dysarthria, dysphagia, and vertigo.(15) Two patients had vertebral artery occlusions and one had a dissection. No treatment was reported for any of these patients. Woodring et al reported two strokes in nine patients with transverse process fractures after blunt cervical trauma.(19) All nine were studied with vertebral angiography; seven studies were abnormal. One patient with vertebral artery occlusion was treated with anticoagulation and improved. One with dissection improved without treatment. Three of five asymptomatic patients were anticoagulated (intravenous heparin converted to Coumadin, total treatment of six weeks); two patients were not treated. All five remained asymptomatic. Schwarz et al reported four patients with ischemic vertebrobasilar symptoms after non-penetrating cervical trauma.(7) Two patients with atlantoaxial injuries recovered

following atlantoaxial stabilization (one treated with halo immobilization, one operatively). Treatment of one patient with delayed symptoms after unrecognized facet dislocation was not reported. Streptokinase infusion was used in a patient with vertebral artery occlusion who became comatose shortly after reduction of a facet dislocation injury. This strategy failed to achieve complete thrombolysis and the patient died days later.

Six prospective studies examining the diagnosis of VAI provided the incidence of neurological complications related to VAI. Biffi et al (1) reported the highest frequency of posterior circulation stroke, occurring in nine of 38 patients (24%). Stroke occurred eight hours to twelve days after injury in these patients, with 78% occurring more than 48 hours after injury. Three of 21 asymptomatic patients treated with intravenous heparin subsequently developed stroke; one died and two had mild residual deficits. In contrast, six of 17 asymptomatic patients not initially treated with intravenous heparin developed stroke; two died, three had mild deficits, and one had a severe deficit. Neither of the two patients who died were treated with anticoagulation when stroke developed nine to twelve days after VAI (one Grade IV, one bilateral Grade I which progressed to right Grade II and left Grade III). All three asymptomatic patients who subsequently developed stroke and were then treated with heparin had mild residual deficits (one Grade I, one Grade II, one Grade IV). A final patient who developed stroke and was treated with antiplatelet agents had severe residual deficits (Grade I). Two of nine patients with stroke who were treated with heparin developed hemorrhagic infarction. Two of the 38 patients had dominant vertebral arteries; both had injuries to the dominant artery and neither developed a stroke.

Weller et al (17) treated all four patients in their report with vertebral artery injuries with aspirin. Three patients with vertebral artery occlusion remained asymptomatic; one with

vertebral artery narrowing developed syncope 17 days after injury. This patient was treated with intravenous heparin followed by aspirin without recurrent symptoms. Giacobetti et al (3) described three of twelve patients with vertebral artery occlusion who developed transient blurred vision; all three were treated with three months of anticoagulation (intravenous heparin followed by Coumadin), and none had recurrent symptoms. Friedman et al (2) reported one patient with bilateral vertebral artery injuries who died after a massive right cerebellar infarct. One patient with vertebral occlusion and concurrent carotid occlusion remained asymptomatic on heparin. The remaining seven patients remained asymptomatic without treatment (five occlusions, two stenosis). Willis et al (18) reported no symptoms in twelve patients with VAI, none of whom were treated with anticoagulation. Louw et al (5) reported two of nine patients with vertebral artery occlusion with symptoms including blurring of vision which spontaneously resolved without anticoagulation.

The management and outcome of 106 patients with VAI after nonpenetrating cervical injury could be determined from the articles reviewed. Twelve patients had radiographic evidence of a posterior circulation stroke as their first symptom. Four patients were treated with intravenous heparin; one died and three improved.(1,19) The remaining eight patients were not treated with intravenous heparin; five died, two improved, and one had a severe neurological deficit.(1,2,8,19) Fifteen patients developed symptoms of posterior circulation ischemia without stroke before treatment was instituted.(3,5,6,7,8,14,17) Eleven of 15 patients were treated with intravenous heparin; two died (both had strokes) and nine improved (one developed a stroke). The remaining four patients were not treated with intravenous heparin; all improved without developing a posterior circulation stroke. Twenty-seven asymptomatic patients were prophylactically treated with intravenous heparin.(1,2,18,19) Three patients developed posterior

circulation stroke; twenty-seven patients remained asymptomatic. Finally, fifty-two asymptomatic patients were not prophylactically treated with heparin (eight were treated with aspirin and one with embolization). Three patients died from injuries unrelated to the VAI. (1,18) Four patients had severe deficits (three were treated with aspirin and one with embolization) and three had mild deficits (one was treated with aspirin); all were related to the associated spinal cord injury.(1) The remaining 42 patients (three of whom were treated with aspirin) remained asymptomatic.(1,2,3,5,6,14,17,18,19)

The articles reviewed did not specifically address the risk of progressive spinal cord hemorrhage worsening in patients with VAI and an associated spinal cord injury treated with heparin. One patient with a complete cervical spinal cord injury and hematomyelia was placed on intravenous heparin prophylactically for left carotid and vertebral artery occlusion; no neurological changes occurred with treatment.(2)

At least 13 of 42 patients treated with intravenous heparin had complications during their treatment, six of which were significant (14%). Six patients (two of whom died) developed posterior circulation strokes after treatment with intravenous heparin was initiated.(1,6,7,14) Two patients developed hemorrhagic posterior circulation strokes; the timing of intravenous heparin relative to the development of the posterior circulation stroke was not reported.(1) Intravenous heparin was discontinued in three patients. One symptomatic patient developed a rectus sheath hematoma (6); the patient's symptoms stabilized after intravenous heparin treatment was discontinued. Two asymptomatic patients had progression in the grade of vertebral artery injury; both remained asymptomatic after intravenous heparin was discontinued. (18) Four other patients had progression in the grade of vertebral artery injury; intravenous

heparin was continued and none of the patients developed deficits related to the progression of injury grade.(1)

In several studies patients were re-imaged to determine whether disease progression or resolution occurred after vertebral artery injury. Biffel et al reported follow-up angiography on 21 patients.(1) Of 16 patients treated with heparin, two improved to a lesser grade of vascular injury and four worsened to a poorer grade. Of five patients not receiving heparin, one improved and three had worse vascular injury grades. Vaccaro et al found reconstitution in one of six vertebral artery injuries by MRA twelve days after the original diagnosis; this patient was not treated with anticoagulation.(16) The remaining five still had vertebral artery occlusion more than one year later, including two treated with anticoagulation. Willis et al described the results of follow-up angiography in three patients with VAI.(18) One patient with a pseudoaneurysm received one week of intravenous heparin followed by aspirin; the pseudoaneurysm had slightly enlarged seven days after treatment was begun, but had disappeared on angiography performed six weeks later. One patient treated with intravenous heparin for a vertebral artery dissection had an asymptomatic occlusion of the artery demonstrated by angiography two days later; the heparin was discontinued. The third patient was treated with intravenous heparin for a vertebral artery intimal flap; the patient had a normal vertebral angiogram ten days later. Thibodeaux et al found a patent vertebral artery six months after dissection was diagnosed; this patient was not anticoagulated.(14) Sim et al reported delayed Duplex sonography in eleven patients with a history of facet dislocation, but unknown vertebral artery status at the time of the original cervical spine injury.(10) Two of these studies demonstrated VAI: one with persistent dislocation had vertebral occlusion, and one patient with a reduced injury had vertebral artery stenosis.

## SUMMARY

The incidence of vertebral artery injury may be as high as 11% after non-penetrating cervical spinal trauma in patients with specific clinical criteria, including facial hemorrhage (bleeding from mouth, nose, ears), cervical bruit in those younger than 50 years of age, expanding cervical hematoma, cerebral infarction by CT, lateralizing neurological deficit, cervical hyper-extension/rotation or hyperflexion injuries, closed head injury with diffuse axonal injury, near hanging, seat belt or other soft tissue injuries to the neck, basilar skull fractures extending into the carotid canal, and cervical vertebral body fractures or distraction injuries. Many patients with VAI have complete spinal cord injuries, fracture through the foramen transversarium, facet dislocation, and/or vertebral subluxation, but many patients with these injuries have normal vertebral arteries when imaged, thus compromising specificity of these injury criteria. Many patients with VAI are asymptomatic, including those with vertebral artery occlusion or dissection. The literature reviewed indicates that patients with posterior circulation stroke and VAI have a better outcome when treated with intravenous heparin compared to those who are not. However, others have reported improvement among patients without anticoagulation.(8,19) The outcome of patients who develop symptoms of posterior circulation ischemia without stroke treated with intravenous heparin (3,6,8,14,17) is similar to those patients receiving no treatment.(5,7) Although the largest prospective study suggested a trend toward less frequent stroke in asymptomatic patients treated with heparin (1), others have not reported similar observations.(2,3,5,6,14,18,19) Since the risk of significant complications related to anticoagulation is approximately 14% in these studies, there is insufficient evidence to recommend anticoagulation in asymptomatic patients.

## **KEY ISSUES FOR FUTURE INVESTIGATION**

Although several prospective studies have been reported examining patients at risk for VAI, most articles did not report sufficient data about the characteristics of the patients with normal vertebral arteries to examine clinical or radiographic characteristics to predict which patients may have VAI. A prospective study comparing MRA with conventional angiography in nonpenetrating cervical spine trauma may define the role of non-invasive imaging studies in these patients. A multicenter, randomized, prospective study comparing anticoagulation with intravenous heparin with observation in asymptomatic patients and in symptomatic patients with posterior circulation ischemia but without stroke is recommended to determine whether anticoagulation of these patients is justified.

## EVIDENTIARY TABLES

First Author Reference	Description of Study	Data Class	Conclusions
Schellinger PD et al, 2001, <i>Spine</i>	Retrospective review of 4 patients with cervical spine injury of 27 patients with vertebral artery dissection diagnosed with various imaging studies	Class III	2 with motor complete cord injuries awoke after cervical surgery with stupor and posterior circulation infarcts; one with basilar occlusion by Doppler died and one with dissection by DSA partially recovered. One with C5 foramen trans. fx became comatose 12 hrs after admission and had fatal basilar thrombosis by CT hrs later. One with treated C2 fx had vertigo & nystagmus 3 wks later; dissection by angio; sx resolved with Coumadin followed by ASA.
Biffi WL et al, 2000, <i>Ann Surg</i>	Prospective angiography screening in blunt craniovertebral injury identified 38 pts with vertebral injuries	Class III	38 pts with 47 VI out of approx 350 angios done. 27 of 38 had cervical fxs. 6 of 27 had fxs thru foramen transv. 9 of 38 post circ stroke. Stroke not related to occlusion v. stenosis 8hr-12 day delay, most beyond 48 hr 3 of 21 asymptomatic patients treated with heparin had stroke vs. 6 of 17 w/o heparin had stroke (p=0.13) 1 of 6 died w/ heparin. 2 of 3 died w/o heparin. 2 treated with heparin had hemorrhagic strokes.
Weller SJ et al, 1999, <i>J Trauma</i>	Prospective MRA in 12 pts with for. transv. fx	Class III	3 of 12 had VA occlusion; all remained asymptomatic on ASA. 1 of 12 with stenosis had delayed syncope on ASA, resolved with brief IV heparin followed by ASA.
Vaccaro AR et al, 1998, <i>Spine</i>	Prospective study with F/U MRA in 6/12 pts (1 excluded) previously reported by Giacobetti FB et al (3)	Class III	1 of 6 treated w/o heparin reconstituted by 12 days. 5 of 6 remained occluded (2 of 5 w/ heparin) >1 yr later.
Giacobetti FB et al, 1997, <i>Spine</i>	Prospective study with MRA in 61 pts with cervical injuries found 12 pts with vert occlusion	Class III	1 of 4 with transv fx had occl, 6 of 15 with facet disloc had occl. 3 of 12 with transient blurred vision resolved with 3mo anticoagulation.
Thibodeaux et al, 1997, <i>Br J Surg</i>	Retrospective review of 3 pts with vertebral dissection on angiography	Class III	1 with blindness but no infarct improved with anticoagulation. 1 with C4-5 facet fx was asymptomatic w/o treatment. 1 with ataxia/dizziness 2 days later with occipital infarct recovered with 6 mo anticoagulation.

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
Prahbu V et al, 1996, <i>J Trauma</i>	Retrospective review of 5 symptomatic pts with 4 vertebral artery occlusion and 1 stenosis on MRA and/or angiography	Class III	3 with fxs delayed coma at 3 hrs, 2 occl/1 narrow, 1 died with stroke, 2 improved with anticoag but 1 d/ced 2° rectus hematoma. 1 with fx delayed confusion/aphasia with multiple MCA strokes improved w/o anticoag 1 with fx asymptomatic w/o anticoagulation.
Friedman D et al, 1995, <i>Am J Roentgen</i>	Prospective study of 37 pts with nonpenetrating cervical trauma found 9 vertebral injuries by MRA	Class II	50% patients w complete cord injuries had vert injury vs 12% pts w incomplete cord injuries (p<0.02). 5 of 13 patients w >3mm subluc had vertebral injuries vs 4 of 24 patients w <3mm subluc. 1 w bilat vert injuries died of large cerebellar infarct (bilat for. trans. fx) 8 asymptomatic (1/8 anticoag also had carotid occlusion).
Tulyapronchote R et al, 1994, <i>Neurology</i>	Retrospective study of 3 pts with VB ischemia >2 wks after occult C2 fractures diagnosed by angiography	Class III	2 occluded, 1 narrow. Sxs included syncope, vertigo, dysphagia, dysarthria, facial numbness, blurred vision. Treatment not reported.
Willis BK et al, 1994, <i>Neurosurgery</i>	Prospective study of 26 pts with cervical facet dislocation or facet fx thru for transv. fx revealed 12 with vertebral injuries on angiography	Class II	9 of 14 with normal angio had for. transv. fx vs. 7 of 12 with abnormal angio. 1 of 9 with occlusion w/o anticoag died from unrelated injuries. 1 dissection became an occlusion on heparin. 1 intimal flap/1 pseudoaneurysm healed with heparin in 7-10 days.
Sim E et al, 1993, <i>Acta Orthop Scand</i>	Prospective delayed Duplex sonography of 11 pts with previously locked facets	Class III	1 of 11 had occlusion (persistent locked facet). 1 of 11 had a narrow vertebral artery.
Woodring JH et al, 1993, <i>J Trauma</i>	Retrospective study of 216 pts with cervical fxs showed 52 with TP fxs, 8 had angiography	Class III	78% of TP fxs extended into for. transv. 4 of 8 had occlusion, 3 of 8 had dissection, 1 of each had stroke that improved with anticoag. 3 asymptomatic treated with anticoagulation.
Schwarz N et al, 1991, <i>J Trauma</i>	Retrospective review of 4 pts with symptomatic vertebral injuries by angiography	Class III	4 delayed sxs (3 occlusions, 1 pseudoaneurysm). 1 with coma and stroke 20 min after reduction of C4-5 dislocation received Streptokinase/heparin for occlusion but died.

<b>First Author Reference</b>	<b>Description of Study</b>	<b>Data Class</b>	<b>Conclusions</b>
Louw JA et al, 1990, <i>J Bone Joint Surg</i>	Prospective study of 12 pts with facet dislocations with digital subtraction angiography	Class III	4 of 5 unilateral and 5 of 7 bilateral dislocations had occluded vert (1 bilat). 3 occluded at level, 5 within 2 cm. 2 of 9 symptomatic patients had bilat C5-6 facet disloc and improved w/o treatment.

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