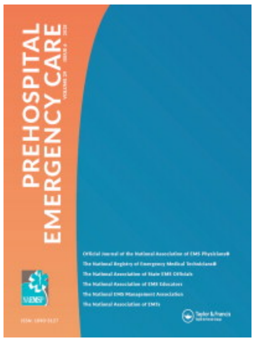

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Prehospital Trauma Compendium: Prehospital Management of Spinal Cord Injuries – A NAEMSP Comprehensive Review and Analysis of the Literature

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ABSTRACT

Objectives: Spinal motion restriction (SMR), requiring the use of a cervical collar and allowing for use of a vacuum splint or ambulance cot, and spinal immobilization, requiring the use of a backboard and a cervical collar, have long been established as the standard of care in the prehospital management of trauma. Both techniques are based on the hypothesis that post-injury movement of the spinal column may lead to the development of delayed neurological deficits. However, these techniques, which have the potential for significant patient harm, are without definitive evidence of clinical benefit. The objective of this review is to evaluate the potential pathophysiology to delayed neurological injury, and examine the potential harms and benefits of spinal immobilization and SMR.

Methods: A structured review of the literature was performed within the National Association of EMS Physicians (NAEMSP) Trauma Compendium Series. Searches were performed in PubMed, Embase, CINAHL, and Web of Science dating back to 1900 looking for manuscripts that addressed the pathophysiology of delayed neurological injury as well as the harms, and benefits, to spinal immobilization and SMR.

Results: Out of 3944 manuscripts screened, 115 manuscripts were identified. Noting that some manuscripts answered multiple study questions – 14 studies addressed the pathophysiology of disease to the phenomenon of delayed neurological injury, 55 studies examined the harms of immobilization procedures, 58 studies addressed the effectiveness of immobilization procedures, and 7 studies addressed other factors. Two case series were identified hypothesizing post-injury movement as the cause of delayed neurological injury; and 8 retrospective studies, including two case control studies and three retrospective cohort studies, were identified showing an association between hypoperfusion and worsening neurological injury. There were 55 studies showing harms, and no studies showing a definitive benefit to spinal immobilization.

Conclusions: There are no data in the published literature to support spinal immobilization and spinal motion restriction as standard of care. Efforts aimed to reduce the use of cervical collars should be considered, and the use of backboards and full body vacuum splints should be limited to the point in time of active patient extrication.

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Introduction

The historical basis to the practice of restricting movement of a patient with concern for spinal cord injury began during the Second World War (1). Howlett described the concern that excessive movement of patients with vertebral fractures could result in hyperflexion or hyperextension of the neck and back (1). Hypothesizing that post-injury movement of an unstable spinal column may lead to irreversible injury to the spinal cord (1), it was recommended to transport patients with spinal fractures immobilized in anatomic position with padding to account for anatomy (1).

During the 1950s and 1960s two case series asserting the hypothesis that post-injury movement can lead to paralysis (2,3), and three educational papers demonstrating how to remove a patient from a car onto a hard board with pullies and winches (4–6), became the foundation to the standard practice of spinal immobilization. In 1957, Rogers published a retrospective case series of 87 patients treated for cervical spine injury with eight reporting delayed onset of neurological deficits (2). The author concluded that movement of the patients' cervical spinal column between time of injury and "…definitive treatment…" directly lead to paralysis (2).

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Notably, the article by Rogers includes pictures of his specially designed collar that he argued would have prevented neurological deficit if it had been applied at the time of injury.

Despite a level of evidence limited to case reports (2,3), which include premature conclusions made by retrospective review and without comparison groups, the American Academy of Orthopedic Surgeons (AAOS) established spinal immobilization as a standard of care with the publication of one of the first emergency medical services (EMS) textbooks, *Emergency Care and Transportation of the Sick and Injured*, in 1971 (7). The authors write, "Carefully splint the injured spine, avoiding abnormal or excessive motion," and "Be sure that the injured person is properly splinted and transported on a long backboard… without bending or twisting the spine in any direction." Further, the authors write that "If the head of an individual with a broken neck is allowed to move, the motion may contribute to paralysis or death" (7).

At the time of the AAOS text, the adoption of spinal immobilization as standard of care was consistent with the common practice of splinting other bony injuries. Importantly, only the bones of the spinal canal could be visualized with advanced radiology at the time, since magnetic resonance imaging (MRI) was not introduced to characterize spinal cord injury until the late 1980s (8–10).

Noting demonstrated harms of immobilization on a hard board that include pain (11), respiratory suppression (12,13), and formation of decubitus ulcers (14–16), balanced against what has been assumed to be a theoretical benefit (7) that is supported by an unproven hypothesis (2,3), the National Association of EMS Physicians (NAEMSP) and the American College of Surgeons Committee on Trauma (ACS-COT) published a statement in 2013 (17), with an accompanying resource document in 2014 (18), which stated, "Utilization of backboards for spinal immobilization during transport should be judicious, so that potential benefits outweigh risks" (17). With the publication of this statement, there was clear recognition that the procedure causes inherent harm that may not be outweighed by benefit.

In 2018 ACS-COT, the American College of Emergency Physicians (ACEP), and NAEMSP published updated guidance identifying spinal immobilization, and spinal motion restriction (SMR), as referring to the "… same concept," while noting that spinal immobilization requires the use of a backboard and SMR "… may be achieved by use of a scoop stretcher, vacuum splint, ambulance cot, or other similar device…" (19). Thought to be less harmful, while allowing for protection of the spinal cord in the advent that movement is indeed the cause of delayed neurological injury, Fischer et al. wrote that, "The goal of… SMR… is to minimize unwanted movement of the potentially injured spine," and continued to advocate for the use of cervical collars as a, "… critical component of SMR [that] should be used to limit movement of the cervical spine…" (19).

Focusing specifically on cervical collars, the 2013 practice guideline published by the American Association of Neurological Surgeons and the Congress of Neurological Surgeons noted that "… a rigid cervical collar… is effective in limiting motion of the cervical spine," and, therefore, preventing the onset of delayed neurological injury (20). Supporting their statement with a case series (21), the authors continued: "Pathologic motion of the injured cervical spine may create or exacerbate cervical spinal cord… injury" (20).

Since the practice of immobilization was first proposed in the 1940s (1) to becoming standard of care in the 1970s (7), authors have written that the cause of delayed neurological deficits is movement of an unstable spinal column (19,20); and the use of spinal immobilization, or SMR, has been implemented as a modality to mitigate against the potential for movement to result in further injury (19). Yet, it seems that there is a dearth of literature supporting the conclusion that movement is the primary cause of delayed neurological injury, or that SMR, or immobilization procedures, have a clinical benefit.

With knowledge that immobilization, and its conceptual equal SMR (19), causes patient harms (18), we sought to identify the literature that sheds light on the underlying pathophysiology of delayed neurological injury in the setting of trauma, and to examine the scientific basis to the effectiveness of these procedures as well as the balance between demonstrated harms and potential clinical benefit.

Methods

We performed a structured review of the literature within the NAEMSP Trauma Compendium Series (22), and we developed a single search strategy with the assistance of a trained medical librarian to answer four research questions:

1. What are the underlying pathophysiological causes to the phenomenon of delayed neurological injury in the setting of trauma with a focus on movement, hypoxia, and hypoperfusion?
2. Does the use of a backboard or cervical collar result in patient oriented harms, with a focus on the formation of decubitus ulcers, developing respiratory depression, causing increased intra-cranial pressure, or direct harm to the nervous system?
3. Are backboards and cervical collars effective at preventing delayed neurologic injury or effective at immobilizing the spinal column as intended?
4. Are there other factors that may affect the utility of backboards and cervical collars such as patient anxiety, patient anatomy, patient age, or environmental conditions?

Once the basic search strategy was developed, we refined it in accordance with the strategy that was developed for all the projects in the Trauma Compendium Series (22), and to account for the unique requirements of four databases. We then ran the searches on April 19, 2023 in PubMed, Embase, CINAHL, and Web of Science using the Covidence systematic review software (see supplemental Table 1 for search strategies) (23).

For the purpose of this project, we defined a backboard as any object that is used to immobilize the entire spinal column including the rigid spine board, Kendrick extrication device (KED), boards used with head blocks, any type of strapping material, and use of a vacuum splint. We defined a cervical collar as any hard, or soft, collar that is used to prevent movement of the neck regardless of the manufacturer or type of collar. As such, we defined both backboards and cervical collars by their intended purpose of immobilization. Reviewers considered both immobilization with a backboard and cervical collar as a single unit and immobilization with a cervical collar alone.

We used the following inclusion criteria to consider a manuscript in the final analysis: peer-reviewed literature dating back to 1900, human and animal studies, addressed one of the four study questions, included patient data, and the final publication was available in English. We reviewed manuscripts dating back to 1900 to be as inclusive as possible in identifying the scientific basis to the pathophysiology of delayed neurological deficits. While we excluded the following from the final analysis, manuscripts in these categories could be flagged to review references for hand search and to be included in the introduction to our manuscript: simulation studies involving manikins and not involving human subjects, abstracts that did not reach full publication in a peer-reviewed journal, editorial letters, other reviews or meta-analysis, clinical practice guidelines, book chapters, and non-English published full manuscripts.

Single reviewers first screened titles and abstracts of manuscripts in the database. Two reviewers then performed full manuscript reviews to determine if the manuscript should be included in the final analysis. The first author (MM) resolved any discrepancies, and provided a secondary review for all manuscripts chosen for full analysis. We abstracted data from the manuscripts chosen for analysis using a standardized data abstraction form in Covidence that was developed by the first author (MM).

In abstracting the data the reviewers summarized the manuscript, and developed a conclusion from their review of the data presented, to answer the four study questions. A master database of the abstracted data was created exporting the data from Covidence into an Excel spreadsheet. This was then grouped into four additional spreadsheets by one of the study authors (GK) based on the manuscripts that answered each of the four study questions. The first and second authors (MM and JI) then reviewed these spreadsheets for accuracy and made edits as needed to ensure that all data abstractions in the four Excel spreadsheets were complete and followed a standardized format for reporting and discussion.

Once the final four spreadsheets were completed these were sent to all the reviewers for independent analysis. We then performed hand searches of additional manuscripts through review of references from clinical practice guidelines, and references provided by the reviewers, and these were then also reviewed using Covidence by consensus of the first and second authors (MM and JI). The abstracted data from these additional manuscripts were added to the final four spreadsheets. Finally, we performed the search strategies two additional times, on June 1, 2024 and February 1, 2025, to account for any articles that may have been published since the initial search; and the first and second authors (MM and JI) then screened, reviewed, and abstracted data from these additional articles in a similar manner as the original search.

Results

After removal of duplicate entries, the reviewers screened 3944 manuscripts (3500 from the search performed in 2023, 300 from the search performed in 2024, and 144 from the search performed in 2025). After title and abstract screening, 769 full manuscripts were reviewed, and 99 manuscripts were identified for full analysis (93 from the initial search, 2 from the 2024 search, and 4 from the 2025 search). An additional 18 studies were considered for inclusion by hand search with 16 included in the final analysis. In total there were 115 manuscripts identified for this scientific review. Some manuscripts answered multiple study questions – 14 studies addressed the pathophysiology of disease to the phenomenon of delayed neurological injury (3,21,24–35), 55 studies examined the harms of immobilization procedures (12,15,16,25,36–86), 58 studies addressed the effectiveness of immobilization procedures (3,25,35,36,41,51,59,63,65,70,77,79,84–129), and 7 studies addressed other factors (38,55,82,110,130–132). See supplemental Figure 1 for literature search flow diagram.

Tables 1–4 provide a summary review of the literature. Table 1 outlines the literature examining potential pathophysiological processes of delayed neurological injury organized by year relative to the introduction of MRI technology. Table 2 outlines the harms of immobilization and SMR as grouped by year of publication – before the 2013 NAEMSP/ACS-COT position statement (17), in the interim years between the 2013 position statement (17) and the 2018 ACS-COT/ACEP/NAEMSP position statement (19), and after the publication of the 2018 position statement (19). Tables 3 & 4 respectively outline the effectiveness of immobilization and SMR in preventing movement and injury. Supplemental tables 2–5 provide a comprehensive summary of the 115 manuscripts in this review grouped by the study questions.

Review of the Literature Addressing Research Questions

What Are the Underlying Pathophysiological Causes to the Phenomenon of Delayed Neurological Injury in the Setting of Trauma with a Focus on Movement, Hypoxia, and Hypoperfusion?

In review of the manuscripts focusing on the pathophysiology of disease, there is greater strength in the evidence

Table 1. Literature pointing toward specific pathophysiological process.

	Pre-MRI (< 1990)	Post-MRI (> 1990)
Post-Injury Movement	Geisler 1966 Toscano 1988	
Hypoperfusion		Gregg 2005 Guly 2008 Mezue 2013 Haldrup 2020 Yin 2021 Clark 2023 Jung 2023
Cord Edema		Ackland 2011
Cord Contusion		Papadopoulos 1991 Hosalkar 2005 Kleweno 2008 Asha 2021
Vascular Injury		Yoshihara 2011

MRI refers to magnetic resonance imaging.

Table 2. Literature on the harms of immobilization and spinal motion restriction (SMR).

Harm	Pre-2013[a]	2013–2017[a]	2018–2025[a]
Airway	Dodd 1995		Jarvis 2023
Respiratory	Bauer 1988 Totten 1999		Jarvis 2023
Increased ICP	Herzenberg 1989 Craig 1991 Raphael 1994 Davis 1996 Mobbs 2002 Lemyze 2011		
Worse Neuro Outcome			Jung 2023
Separation of C1–C2	Ben-Galim 2010		
Decubitus Ulcer	Plaisier 1994 Cordell 1995 Main 1996 Sheerin 2007 Kosashvili 2009 Berg 2010 Hemmes 2010	Mok 2013 Hemmes 2014 Nemunaitis 2015 Ham 2016 Pernik 2016	Mitra 2024 Worsley 2018
Missed Injury	Barkana 2000	Klein 2016	Hussain 2019
Delays in Care	Brown 2009 Haut 2010	Bucher 2015	Taghavi 2021
Increase Radiology	Leonard 2012	 Tello 2014	Drain 2020 Clemency 2021
Pain	Cordell 1995 Hauswald 2000 Hemmes 2010 Leonard 2012	Bruijns 2013 Bucher 2015 Ham 2016 Oosterwold 2017	Bruton 2024 Mitchnik 2024

[a]Manuscripts organized by year in relation to the publication of position statements on spinal immobilization – before the 2013 NAEMSP/ACS-COT statement, in the interim years between the 2013 position statement and before the 2018 ACS-COT/ACEP/NAEMSP position statement, and after the 2018 position statement.

Table 3. Literature on the effect of immobilization or spinal motion restriction (SMR) on limiting movement.

Increases movement of spinal column	Has no effect on movement of spinal column	Limits movement of spinal column
Perry 1999	Johnson 1996	Graziano 1987
Chin 2006	Horodyski 2011	Howell 1989
Prasarn 2012	Conrad 2012	Joyce 1992
Meusch 2014	Bucher 2015	Hughes 1998
Wampler 2016	Uzun 2020	Hostler 2009
Rahmatalla 2018	McDonald 2021	Engsberg 2013
Haske 2020		Mahshidfar 2013
Nolte 2021		Pryce 2016
Usun 2024		Thezard 2019
		Jung 2021
		Eisner 2022

supporting the hypothesis that the underlying pathology is related to hypoperfusion of the spinal cord (26,29,58), which may have an associated systemic vascular injury (21,35), than the evidence supporting the hypothesis that post-injury movement of the spinal column is the leading cause of delayed neurological deficits. While we identified two studies in our review that make an association between movement and delayed neurological deficits (3,21), both of these studies were case series with confounding variables with hypoperfusion as a possible underlying pathological process.

In 1966, Geisler et al. published a retrospective case series including patients who developed neurological deficits after the initial point of injury (3). The study authors collected data from hospital nursing notes to document changes in

Table 4. Literature on the effect of immobilization or spinal motion restriction (SMR) on the development of delayed neurological injury.

Causes neurological injury	Has no effect on development of neurological injury	Prevents neurological injury
Yoshihara 2011	Arishita 1989	
Asha 2021	Barkana 2000	
Klein 2016	Burton 2005	
	Armstrong 2007	
	Jin 2007	
	Vanderlan 2009	
	Lin 2011	
	Tatum 2017	
	Misasi 2018	
	Swartz 2018	
	Tsutsumi 2018	
	Underbrink 2018	
	Castro-Marin 2020	
	Clemency 2021	
	Nilhas 2022	
	Chen 2022	
	Vaillancourt 2023	
	Burton 2024	
	Mitchnik 2024	
	Mitra 2024	

neurological exam during a patient's hospital course. While the authors stated that they examined 29 patients with delayed onset of symptoms, they only provided data for two patients in their manuscript. The first patient, with hypoperfusion, a depressed skull fracture, and thoracic spine tenderness, developed permanent paralysis at the T4 level after being in a car crash in 1955. The second patient, similarly in a car crash in 1949, developed weakness in his leg with standing that improved with physical therapy. With limited information on extent of these two patients' injuries, their vital signs, or the findings from a complete motor exam, the authors wrote that the onset of delayed paralysis could have been prevented "…had the spinal instability been recognized and precautions taken," and further asserted that "… [these] patients developed further paralysis through faulty handling" (3).

Also published before the widespread use of MRI technology (8–10), Toscano reported a retrospective case series with 9 patients who were admitted to a spinal injury unit that were noted to have onset of neurological deficits that developed while in the care of ambulance personnel (21). The author of this case series concluded, based entirely on interviews with the patients, ambulance personnel, and clinicians at the spinal injury unit, and without comparison groups, that, "… appropriate handling and immobilization… could make neurological deterioration a rare event," while also noting that it was difficult to determine if the deterioration was due to post-injury movement or another pathophysiological process (21).

Specific to hypoperfusion as a potential cause to the development of delayed neurological deficits, in eight studies neurological outcomes were associated with perfusion (26–29,31,32,34,58) with outcomes reported in three retrospective cohort studies directly related to reported mean arterial pressures (MAP) (26,29,58). In a study of 99 patients with spinal cord injury, Clark et al. showed that a 10 mmHg increase in prehospital MAP led to an average increase of 79% in the odds of a patient having an improvement in neurological outcome with an inflection point MAP of 85 mmHg (26). Further, Haldrup et al. studied hypotensive events, defined as a MAP < 80 mmHg, in three phases of a patients' care – prehospital, in the operating room, and in the neuro-intensive care unit – and demonstrated that less prehospital hypotensive events led to improved neurological recovery at one year ($p < 0.001$) (29).

While not specifically studying perfusion, in a questionnaire based study of 68 patients with cervical cord injury admitted to a neurosurgical center in Nigeria, patients transported lying down had better outcomes than those that were transported sitting, which could have been related to post-injury movement or relative hypoperfusion to the spinal cord (32).

In addition to specifically focusing on blood pressure, our review identified cord edema/cord contusions (24,30,31), epidural hematomas (25,33), and vascular injury (21,35) as other potentially related causes of delayed neurological deficits, all of which may still be associated with decreased perfusion. Of note, we identified no manuscripts that identified post-injury movement as a pathophysiological cause beyond the two previously mentioned case series.

Does the Use of a Backboard or Cervical Collar Result in Patient-Oriented Harms with a Focus on the Formation of Decubitus Ulcers, Developing Respiratory Depression, Causing Increased Intra-Cranial Pressure, or Direct Harm to the Nervous System?

With regards to harms caused by immobilization devices, our review demonstrated that the bulk of the literature focused on the risk of developing pressure ulcers from spinal immobilization, and specifically noted increased tissue pressures (16,49,53,54,60,64,67–69,72, 75,81) and decreased tissue perfusion (15,73). This was also noted in manuscripts studying cervical collars alone (49,73,81,86), and implicates alternative devices such as the vacuum splint which, while more comfortable, still result in tissue pressures that are similar to a padded long spine board (75). Importantly, our review demonstrated that the decreased tissue perfusion, and higher risk of developing decubitus ulcers, occurs within minutes of the application of the device and is not a phenomenon isolated to prolonged use of these devices (15,16,49,68,81).

The respiratory impact of spinal immobilization is also of concern, with several manuscripts describing respiratory compromise. In one study of anesthetized patients, use of cervical collars caused complete airway obstruction in five patients (46). Another indicated statistically significant reduction in pulmonary function indices including forced vital capacity and forced expiratory volume in one second in healthy volunteers (12), which would have a deleterious effect in a patient with an injury to the respiratory system. Once again, comparison with vacuum splint indicated that despite being more comfortable, the vacuum splint did not mitigate the risk of statistically significant reduction in pulmonary function indices (78).

Direct harm to the neurological system from the use of immobilization devices has also been documented. Increased intracranial pressure (ICP) as a result of cervical collar application has been examined by several authors through direct measurement of ICP in patients admitted to an intensive care unit with a cervical collar, with most suggesting a proposed mechanism of impaired venous or cerebral spinal fluid drainage (44,45, 56,66,74). A case study of a hanging patient supported this, noting neurological improvement in a patient with cerebral edema on CT after removal of the cervical collar (61).

While not specifically focused on increased ICP, Jung et al. showed worse neurological outcomes with cervical spine immobilization in patients with traumatic brain injury when there was a decreased MAP (58), further identifying the potential harm with the routine application of cervical collars in a multi-system trauma patient. Finally, with regards to mechanical worsening of injury, Ben-Galim et al. demonstrated a significant dissociative injury with the application of a cervical collar in nine cadavers with a surgically created fracture of C2 ranging from 3.23–9.23 millimeters (37).

Several authors have studied the potential to miss injuries that are masked by immobilization devices (36,57,59), as well as delays in care due to the use of these devices (39,41,52,76). Hussain and Corsar reported a case of a patient with a missed scalp injury requiring operative washout and repair due to placement of a cervical collar (57). Klein et al. reported on 151 out of 2,267 (6.7%) patients with missed potentially life threatening non-skeletal neck injuries, including tracheal lacerations and carotid artery dissection (59). From this cohort of patients with blast injuries, only 0.83% had cervical spine injuries (59). As such, immobilization exposed patients to the risk of missed neck injuries without a clinical benefit. Specific to harm among patients with penetrating trauma, Haut et al. demonstrated a two-fold increase in mortality of patients with penetrating trauma that were placed on a backboard (52), which the authors attributed to delays in patient care.

Spinal immobilization has been well documented to cause pain and discomfort (16,40,41,49,50,54,62,71,84,85), and to result in increased rates in the use of diagnostic radiology (47,62,77,93). Leonard et al. demonstrated that immobilized pediatric patients are more likely to incur cervical radiography than those that were not immobilized (56.6% vs. 13.4%, OR 8.2, 95% CI 4.5 – 15.4) (62). Although Ward et al. found no change in use of diagnostic radiology after implementation of a protocol change in the management of pediatric patients with concern for spine injury from compulsory use of backboards and cervical collars to the adoption of a modified version of SMR (20% vs 18%, OR 0.84, 95% CI 0.66 – 1.07) (80), this study should be taken in context of a new change to a statewide protocol that continued to require all trauma patients to be immobilized in a cervical collar, and directed clinicians to use full immobilization with a backboard for pediatric patients unable to ambulate on their own (133).

Are Backboards and Cervical Collars Effective at Preventing Delayed Neurological Injury or Effective at Immobilizing the Spinal Column as Intended?

In review of the manuscripts that focused on the question of effectiveness of immobilization, we identified one study suggesting a possible clinical benefit to immobilization. Chen et al., using data from the Pan-Asian trauma outcome study, found a statistically significant favorable outcome with spinal immobilization on a subgroup analysis of patients with cervical cord injury (aOR 3.14, 95% CI 1.04 – 9.50, $p = 0.043$) that was most pronounced in patients with an ISS > 9 (aOR 5.50, 95% CI 1.02 – 29.69, $p = 0.048$) (91). However, the authors found no improvement in neurological outcomes with the use of immobilization devices in a multivariable logistic regression of their entire cohort (aOR 1.06, 95% CI 0.62 – 1.81, $p = 0.826$).

It is important to note that the study by Chen et al. has multiple methodological errors. Most significantly, the authors defined a favorable outcome as a modified Rankin score (mRS) of 0–3 such that patients with a "favorable functional outcome" may have had moderate disability with a mRS of 3 (91). In addition, in the subset analysis of patients with cervical cord injury, the authors did not control for blood pressures, they excluded patients with traumatic brain injury, and in the overall database of 759 patients those with an ISS > 9 and those who received prehospital fluid management both had worse neurological outcomes with spinal immobilization (aOR 0.35, 95% CI 0.17 – 0.72, $p = 0.004$; and aOR 0.39, 95% CI 0.19 – 0.84, $p = 0.016$ respectfully) (91). Noting that 4.4% of patients with a favorable outcome compared to 19.8% of patients with an unfavorable outcome had prehospital fluid management (91), the authors' data are confounded by the potential that patients with a favorable outcome were more likely to be in the sub-group that did not have hypoperfusion regardless of immobilization.

Other than the study by Chen et al. (91), which has questionable clinical significance, we found no other manuscripts that clearly demonstrated that immobilization prevents the development of delayed neurological injury. In fact, we identified a number of manuscripts that specifically demonstrated either no change in neurological deficits (36,70,79,84–87,107,111,113,127), or worsening injury after application of the immobilization device (25,35,51,59).

In 1998, Hauswald et al. published a five-year retrospective chart review comparing patients with spinal injuries at two different academic medical centers (51). One hundred and twenty patients at the University of Malaya had no spinal immobilization compared to 334 patients at the University of New Mexico who did receive spinal immobilization. Hauswald et al. found less neurological disability in the un-immobilized patients than those who had been immobilized (OR 2.03; 95% CI 1.03 – 3.99; $p = 0.04$) (51). While this study has been met with criticism due to the vast differences between these two EMS systems and the authors not controlling for the severity of non-spinal injuries (20,134), the study by Hauswald et al. challenges the hypothesis that movement is the cause of delayed neurological injury and raises a hypothesis that perhaps there are other factors that cause this phenomenon (51).

While we did identify manuscripts that demonstrated that immobilization can minimize movement of the spinal column (63,98–100,104–106,109,110,119,123), we also identified manuscripts that demonstrated no change in the measurement of movement of the spinal column with immobilization (41,94,103,108,112,125). Of note, we further identified manuscripts that demonstrated a paradoxical effect with increased movement of the spinal column after the application of the immobilization device (65,92,102,114–116,120,128,129), with Meusch and Rahmatalla demonstrating an exaggeration of movement at the junction of the neck and sternum associated with fore-aft vibration from a transporting vehicle (65).

Finally, we identified manuscripts that demonstrated that withholding immobilization did not result in neurological deficit (88–90,93,121,122,124,126). Several manuscripts specifically examined the use of cervical collars (85,88,122,126) within the context of changes to cervical spine field clearance and immobilization protocols, and noted no increase in delayed neurologic injury in patients without cervical collars (85,88,122,126). This included patients found to have cervical spine injury who were transported without cervical immobilization (85,122,126). Manuscripts examining implementation of spinal motion restriction (SMR) protocols, which eliminated the use of backboards, also found no increase in delayed neurologic injury (90,93,124).

Are There Other Factors That May Affect the Utility of Backboards and Cervical Collars Such as Patient Anxiety, Patient Anatomy, Patient Age, or Environmental Conditions?

Examining literature that addressed other mitigating factors in the use of immobilization devices, it is worthwhile to note that cervical collars were found to distort the anatomy of pediatric patients (38,55). In addition, geriatric patients experienced prolonged time in these devices compared to a younger cohort (131) and they are at risk for developing dysphagia and respiratory failure (82). Our review did not identify literature that clearly demonstrated that other factors such as anxiety, or environmental conditions affected the utility of immobilization devices.

Discussion

Our findings challenge the historical precautionary principle (135) that immobilization is safer than no immobilization, which is based on the hypothesis that if an individual with a spinal column injury is allowed to move the "… motion may lead to paralysis or death" (7). With consideration to the historical precedent, balanced against known harms, we assumed that there was a scientific basis supporting the use of immobilization procedures, and we designed our study to find this literature. In addition to casting a wide net with our search strategy, we also included multiple opportunities to add to our database through hand searches.

Based on the compiled evidence, we believe that there is indisputable evidence that immobilization causes significant, and potentially life threatening, harms, which continue to mount (82–86), and we found no definitive evidence that there is a clinical benefit to immobilization, or procedures designed to restrict movement.

Further, and perhaps more important, in our review we found no definitive evidence that post-injury movement leads to the development of delayed neurological injury, which is a concept that has been perpetuated in the medical literature as a definitive pathology since the 1940s (1). While Rogers, Geisler, and Toscano all argue that post-injury movement caused the delayed neurological injury that they observed (2,3,21), and subsequent professional clinical practice guidelines echo these assertions (19,20), none of these authors are able to support their claims with a higher level of evidence than anecdotal observations from case reports. It is, thus, reasonable to question if the phenomenon of a patient developing delayed neurological injury after a traumatic event exists. Perhaps the injury is there at the point of impact, and the extent of the injury evolving over time is determined by the initial severity of the injury, regardless of the care provided.

On the other hand, if indeed the phenomenon of patients developing delayed neurological deficits does exist, while the pathophysiology may never be fully understood, our project identifies a greater level of evidence supporting the hypothesis that hypoperfusion of the spinal cord is the primary cause to this phenomenon than post-injury movement of the spinal column. In this context, it is also worthwhile to note that all of the manuscripts in our database pointing toward hypoperfusion, and other vascular insults, as the primary pathophysiology were published after the introduction of MRI technology (24–35), while the manuscripts suggesting post-injury movement were all published before MRI was in common use (3,21) (see Table 1). Interestingly, even in the editorial letter published in 1944 suggesting that patients should be carried on a board, the authors stress the importance of treating shock (1). As such, the authors of this review identify the potential for an iatrogenic distracting injury whereby field clinicians are at risk of focusing on limiting movement rather than focusing on limiting hypoperfusion.

Notwithstanding, even if post-injury movement can result in the development of delayed neurological deficits, our review demonstrates that the practice of restricting movement of the patient does not work as intended. In fact, in some cases this can actually result in increased, and unintended, movement of the cervical-thoracic junction of the spinal column (65,129). Common sense, clinical experience, and as reported in the literature in our review (16,50,136), patients with the ability to detect and react to painful stimuli may move themselves to get comfortable when there are attempts to restrict their movement.

Noting that we cannot rule out the possibility that movement contributes to the development of delayed neurological deficits, we believe that it is reasonable to engage in coaching and shared decision making (137–141) to minimize gross movement of the spinal column as much as possible without the use of immobilization devices as patients will inherently avoid movements that result in pain or injury (136). Further, the argument that a patient can be placed in a position with intent to restrict movement without causing skin breakdown is well refuted by the literature identified in our review and an understanding of the science of the formation of decubitus ulcers (142–145). Skin breakdown itself is partly driven by hypoperfusion (143,144), and this process begins as soon as blood supply is restricted to the area with increased tissue pressure (15,16).

Germane to the findings in our review are three fundamental principles of medical ethics – beneficence, nonmaleficence, and patient autonomy (146). Our project is further grounded in the Institute of Medicine's report Crossing the Quality Chasm – medical care should be safe, effective, and patient centric (147–149). Providing patients with informed consent about the potential harms and benefits to a medical procedure is fundamental to the objective of ensuring patient safety and reducing medical errors (147,150,151). In the interest of patient safety (152), we believe that the precautionary principle that is the foundation to immobilization procedures no longer applies, and the emphasis in patient care should be placed on resuscitation and maintenance of patient comfort.

There is no doubt that our findings reflect a major shift in the understanding of spinal cord trauma. Emergency medical services physicians should lead the development, and administration, of curricula and quality management programs (153), to ensure EMS clinicians are properly trained in the recognition of a patient with concern for spinal cord injury, and in the application of these findings into clinical practice. In addition, EMS system leaders should collaborate with local trauma system leadership in change management, and renewed focus should be placed on resuscitation.

Finally, EMS systems should ensure that protocols developed to protect patients with concern for spinal cord injury are based on techniques that have been proven to have a benefit through an evidence based approach (153–155). It is our hope that our review will open the door for future research on the EMS care of patients with concern for possible spinal cord injury.

Limitations

It is appropriate to acknowledge that most of the manuscripts identified in our review are retrospective studies complicated by sources of bias and uncontrolled confounding variables. Further, our assessment that the literature points toward hypoperfusion of the spinal cord as a more likely cause of delayed neurological deficits than post-injury movement is based on analysis of retrospective case control and cohort studies that show an association between hypoperfusion and poor neurological deficits. Yet, association does not prove causation. It is possible that hypotension is due to spinal cord injury, rather than worsening spinal cord injury being caused by hypotension. However, it should not be dismissed that the practice of restricting movement of the spinal column in the setting of trauma is entirely based on an association observed in a limited number of case reports.

As our review did not differentiate between hard and soft collars, we cannot fully comment if soft collars are any safer than hard collars. Nevertheless, it seems that soft collars can still cause the development of decubitus ulcers (81,86), while producing similar rates of patients having neurological deficits (84–86). Further research would be needed to determine if there is a benefit to soft collars without causing increased rates of decubitus ulcers, impaired vascular flow, or other harms.

We recognize that the studies that we reviewed on hypoperfusion were performed in an adult patient population.

We did not identify papers noting target MAPs for pediatric patients that correspond with neurological outcomes. In addition, it should be noted that this literature is primarily based on inpatient data, and there are no prospective studies examining optimal MAPs in the EMS environment. Emergency medical services systems should account for physiological differences when determining a threshold MAP for pediatric trauma patients (156,157), and prospective studies specific to the EMS environment should be performed.

Finally, we cannot comment on the best method to manage hypoperfusion in patients with concern for spinal cord injury. Other papers in the trauma compendium series (22) may address the concept of permissive hypotension (158), as well as the use of crystalloids (158), blood products, and vasopressor agents (159) to manage hypoperfusion in the setting of trauma. Further, our study focused on preventing the development of delayed neurological deficits in patients with concern for spinal cord injury, and it should be understood that trauma can be a multi-organ system disease. As there are likely greater benefits than harms to managing shock in trauma, EMS systems should consider a variety of treatment modalities, and may have to consider mobilization of advanced life support resources that may not otherwise be used in the care of these patients.

Conclusion

Despite historical precedent, there is no literature demonstrating a clinical benefit to spinal motion restriction. In fact, efforts to restrict movement cause harms and may have a paradoxical effect. The pathophysiology underlying the development of delayed neurological deficits in the setting of trauma, if this pathology exists, is likely multi-factorial. EMS clinicians should focus on management of shock and hypoperfusion. Efforts aimed to reduce the use of cervical collars should be considered, and the use of backboards and full body vacuum splints should be limited to the point in time of active patient extrication. Given the lack of data supporting clinical benefit, and the extent of data demonstrating the evidence of harm, spinal immobilization, and SMR, should not continue to be upheld as standard of care.

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MM and JG conceived the project with the oversight of the NAEMSP Trauma Compendium Project Editorial Board. MM, JI and GK wrote the initial draft of the manuscript with oversight by JG. MM, JI, and JG assume full responsibility for the entire content of the manuscript. All authors assume responsibility for the collection and integrity of the data; and participated fully in the data analysis, and editing of the manuscript.

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