Trauma and Emergency Care

Research Article

Cervical spine immobilization in penetrating cervical trauma is associated with an increased risk of indirect central neurological injury

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Abstract

This study hypothesized that cervical spine immobilization (CSI) in penetrating cervical trauma is associated with increased central neurologic injury rather than prevention. Data abstraction proceeded from a previously constructed patient database formed via retrospective chart analysis of the trauma registries of two independent American College of Surgeons verified Level 1 Trauma centers. Neurologic injuries were categorized as peripheral or central. Central neurologic injuries were further subdivided into spinal cord and brain injuries. Patients were grouped according to the presence and type of neurologic injury, the presence and type of cervical spine fracture, death, and the presence or absence of respiratory and vascular injury. Vascular injury was further subdivided into major and minor categories. Groups were compared statistically. Significance was accepted for $p<0.05$. Cervical spine fracture (CSFx) was a significant risk factor for cervical spinal cord injury (CSCI) ($p<0.00001$; RR 20.56; 95% CI 8.44-26.47) but all patients with unstable CSFx presented with complete spinal neurologic devastation. Major vascular injury was associated with brain injury ($p=0.01$; RR 10.21; 95% CI 6.67-15.65) but was not associated with CSCI ($p=0.99$) or CSFx ($p=0.67$). Hypoperfusion was a strong independent risk cervical cord and brain injury ($p<0.00001$; RR 38.4; 95% CI 16.17-91.2). CSI was a significant risk factor for indirect central neurologic injury ($p<0.001$; RR 1.63; 95% CI 1.23-1.95). Brain injury was not associated with CSFx ($p=0.35$) or CSCI ($p=0.08$). No benefit of CSI in penetrating cervical trauma could be determined from this study. CSI entailed an absolute risk increase for central neurologic injury of 18.69% with a 5.3 number needed to harm (NNH).

Introduction

Current recommendations for CSI were developed for blunt trauma patients and empirically extended to include patients with penetrating injury [1-4]. CSI was designed to prevent progression of direct neurologic injury from cervical spine instability caused by CSFx and/or ligamentous injury. Cervical spine instability resulting from isolated ligamentous instability does not occur following penetrating cervical trauma making CSI unindicated in the absence of unstable CSFx. Unstable CSFx in association with neurologic salvageability has not been clearly shown to exist in the setting of penetrating cervical trauma. Unstable CSFx resulting from penetrating cervical trauma has been repeatedly associated with complete neurologic devastation in the surveyed literature. CSI has no demonstrated benefit in neurologically devastated patients [2].

Empiric extension of CSI to penetrating cervical trauma ocurred without rigorous examination of efficacy or cost-benefit analysis and inappropriate CSI is not without adverse effects. CSI can impede medical care of vascular and respiratory injuries in cases of penetrating cervical trauma [3,5,6] which raises concern given that penetrating cervical trauma causes major vascular injury in over 25% of patient with an incurred 50% mortality [1,6-9]. Central neurologic injury frequently results from common and internal carotid artery (ICA) injuries which are the most commonly injured vessels [1]. 50% of patients with ICA injuries and 77% of patients with common carotid injuries with neurologic deficits due to hypoperfusion and resultant brain ischemia [10]. Expedited definitive care is needed not only for prevention of neurologic injury [11-13] but also because exsanguination is the cause of over 50% of deaths with penetrating cervical trauma [8]. Delayed operative repair o carotid injuries results in significant progression of cerebral neurologic sequelae [10,11]. CSI can cause respiratory compromise through direct mechanical compromise (rapidly expanding hematomas) as well as impeding successful endotracheal intubation [6,14-16] potentially leading to hypoxia and further neurologic injury.

The rapid mortality associated with vascular and airway injuries emphasizes the importance of immediately treating these life-threatening injuries before addressing concerns about cervical spine instability [5,17-22]. Transport delays for CSI vary from an average of 8-30 mins [23,24]. Average total field times are reduced 34% from 46-20 mins by initiation of a “scoop and run” policy [24]. The effect of time on patient outcome is critical as a 10% increase in mortality accompanies every incremental 10 min delay in reaching definitive

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care [25]. The time required for appropriate CSI may contribute to delay iatrogenia.

The purpose of this study is to examine the relationship between penetrating cervical trauma and the development of central neurologic injury with prophylactic CSI. The lack of applicability of CSI in penetrating cervical trauma and the host of iatrogenia associated with CSI, surmises the hypothesis that central neurologic injury in penetrating cervical trauma is more likely associated with, rather than prevented by, CSI.

Materials and methods

Multi-center, retrospective chart analysis performed using the data housed in the trauma registries of two independent American College of Surgeons verified, Level 1 Trauma centers was used to construct a patient database [26,27]. The trauma registries of the Louisiana State University Health Sciences Center, New Orleans (New Orleans, LA-Charity) and the Hurley Medical Center, Flint, Michigan, were queried for all cases of penetrating cervical trauma. The Charity Hospital trauma registry was searched from 01/01/1994 to 04/17/2003, The Hurley Medical Center trauma registry was searched from 01/01/2000 to 12/31/2005. Charts were excluded for lack of penetrating cervical trauma, incompleteness and patient elopements prior to evaluation and patient discharges "against medical advice". Patients were grouped according to the presence or absence of neurologic injury, CSFx, vascular injury, respiratory injury and CSI. Abstracted data included age, sex, race, mechanism of injury, admission Glasgow Coma Scale (GCS), admission neurologic findings, zone of injury, associated injuries, the types and findings on radiographic imaging, surgical intervention, neurologic outcome, disposition from the hospital and mortality. Institutional review board approval was obtained from Tulane University (New Orleans, LA), Louisiana State University Health Sciences Center, New Orleans, and Hurley Medical Center.

Hospital systems

Trauma surgery, orthopedic spine and neurological coverage at Charity were the responsibility of both Tulane University (Tulane) and Louisiana State University, New Orleans (LSU) and alternated daily between these two institutions. Senior radiology residents and staff radiologists of the LSU department of radiology provided radiology services. Emergency medicine coverage was continuously provided by LSU emergency medicine staff and residents. Hurley has teaching affiliations with Henry Ford Hospital (Detroit, MI), Michigan State University (Lansing, MI) and University of Michigan (Ann Arbor, MI). Radiology services were provided by Hurley radiology residents and staff radiologists. Emergency medicine staff and residents of University of Michigan provided continuous emergency medicine coverage.

Treatment of cervical spine injuries was similar between the treating institutions in that cervical spine injury associated with neurologic injury was care for by neurosurgery while all other cervical spine injury was cared for by orthopedics. Cervical instability was designed based on the three-column system [28]. Two of the three columns had to be disrupted to meet criteria for instability. Radiology provided initial reports of instability, which was then subject to confirmation by the consulting spine service. Peripheral nerve injury was defined as any neurologic injury attributable to the cervical distribution that was not related to a spinal cord injury. Spinal cord injury was defined as any injury of the spinal cord. CSFx induced any bony disruption of the cervical spinal column. Brain injury was defined as penetrating cervical trauma causing any indirect injury of the cerebral hemispheres, basal ganglia, cerebellum or brain stem structures. Direct penetrating brain injury was excluded from causal analysis.

Major vascular injury was defined as involving the internal jugular vein (IJV), common carotid artery (CCA), ICA, external carotid artery (ECA) and vertebral artery (VA). Minor vascular injuries were defined as any cervical vascular injury not considered a major vascular injury and included the external jugular vein (EJV), anterior jugular vein (AJV), occipital artery, lingual artery, thyroid artery, facial vein and unnamed veins.

Data management and statistical analysis

Patient data were recorded, described statistically, and presented in frequency and proportion using inherent database computational software (Microsoft Excel 2000, Redmond, WA). Tests for comparing the proportion difference between two conditions were conducted by Chi-squared (χ^2) statistic using SAS v9.1 (SAS Institute Inc., Cary, NC). Significance was accepted for $p<0.05$.

Results

The study cohort consisted of 231 patients. 35 of these patients died. All patient deaths were in the Charity group. Survivors numbered 153 patients in the Charity group and 43 in the Hurley group. Patient characteristics were summarized for both groups and statistically compared (Table 1). Demographics, mechanism of injury and neurologic deficits were similar between the two groups, but mortality and the incidence of CSI in the Charity group was significantly higher than the Hurley group.

Of 94 non-DOA patients with CSI, 15 (16%) had CSFx. All of these patients received CSI. Cervical spinal cord injury (CSCI) occurred in 8 (8.5%) non-DOA patients and 88% (7) had an associated CSFx. Two patients incurred unstable CSFx and both had attendant complete spinal cord neurologic devastation. CSFx was a significant risk factor for CSCI ($p<0.00001$; RR 20.56; 95% CI 5.21-32.33). However, unstable CSFx was associated with non-neurologic salvageability ($p<0.0001$; OR 0.001; 95% 0.00-0.05). 21 patients incurred 26 major vascular injuries (Table 2a). Multiple major vascular injuries occurred in five patients with accompanied minor vascular injury in three patients (Table 2b). The IJV was the most commonly injured major vessel (N=10), followed

Table 1. Patient characteristics were summarized and compared statistically.

Demographic Characteristic	Charity	Hurley	Significance (p)
Male	130 (84.96%)	40 (93.02%)	NS
Female	23 (15.03%)	3 (6.98%)	NS
Age, mean ± SD, yrs	28.97 ± 11.53	29.61 ± 11.64	NS
African American	111 (72.55%)	31 (72.09%)	NS
Caucasian	37 (24.18%)	10 (23.26%)	NS
Other Race	5 (3.27%)	2 (4.65%)	NS
Other Race	**35**	0	0.001
Gunshot Wound	59 (38.56%)	21 (48.84%)	NS
Stab Wound	94 (61.44%)	22 (51.16%)	NS
Cervical Spine Fracture	10 (6.54%)	5 (11.63%)	NS
Brain Injury	1 (0.65%)	1 (2.33%)	NS
Respiratory Injury	9 (5.88%)	5 (11.63%)	NS
Peripheral Nerve Injury	9 (5.88%)	3 (6.98%)	NS
Cervical Spinal Cord Injury	5 (3.27%)	3 (6.98%)	NS
C-Spine Immobilization	**84 (54.90%)**	**11 (25.58%)**	**< 0.001**

The Charity and Hurley patient cohorts differed significantly only in the incidence of death and cervical spine immobilization (c-spine immobilization)

Significance was accepted for $p < 0.05$

Table 2a. Major vascular injuries indicating respective treating institutions and associated injuries.

Major Vascular Injury	Charity	Hurley	PNI	CSCI	Brain Injury	CSF	CSI
Internal Jugular Vein (IJV)	1 (+ICA)¥		YES (XII)	No	No	No	No
	1		No	No	No	No	YES
	1		No	No	No	No	YES
	2		No	No	No	No x 2	No
		2	No	No	No	No x 2	No
	1 (+VA)£		YES (Brachial Plexus)	No	No	No	YES
		1 (+ CCA)Φ	No	YES (C6-7)	R MCA CVA	STABLE	YES
		1	No	No	No	No	YES
Common Carotid Artery (CCA)	1		No	No	R MCA CVA	No	YES
	1 (+ICA)μ		YES (VII)	No	No	No	YES
	1		No	No	No	No	YES
	1 (+FV)¶		No	No	No	No	YES
		1	No	No	No	No	No
		1 (+ ECA)€	No	No	No	No	No
		1 (+ IJV)Φ	No	YES (C6-7)	R MCA CVA	STABLE	YES
Internal Carotid Artery (ICA)	1 (+CCA)μ		YES (VII)	No	No	No	YES
	1 (+IJV)¥		YES (XII)	No	No	No	No
	1		No	No	No	STABLE	YES
	1 (+ ECA)α		YES (VII/XII)	No	No	No	No
External Carotid Artery (ECA)	1		No	No	No	No	No
	1		No	No	No	No	No
		1 (+CCA)€	No	No	No	No	No
	1 (+VA)π		No	No	No	No	No
	1 (+ICA)α		YES (VII/XII)	No	No	No	No
Vertebral Artery (VA)	B		No	QUADRIPLEGIA	No	UNSTABLE C6	YES
	1 (+ECA)π		No	No	No	No	No
		1	No	No	No	No	No
	1 (+IJV)£		YES (Brachial Plexus)	No	No	No	YES

PNI: Peripheral nerve; CSCI: Cervical spinal cord; CSFx: Brain and cervical spine fracture; CSI: Cervical spine immobilization, indicating presence on admission (YES) or absence on admission (No); VA: Vertebral Artery; FV: Facial Vein; XII: Cranial Nerve 12; VII: Cranial Nerve 7; C6–7: Spinal cord level at cervical vertebrae six and seven; R MCA: Right middle cerebral artery; CVA: Cerebrovascular Accident.
Same superscripts indicate same patient

Table 2b. Minor vascular injuries indicating respective treating institutions and associated injuries.

Minor Vascular Injury	Charity	Hurley	PNI	CSCI	Brain Injury	CSFx	CSI
Vertebral Artery (VA)	BILATERAL		No	QUADRIPLEGIA	No	UNSTABLE C6	YES
	1 (+ ECA)ð		No	No	No	No	No
		1	No	No	No	No	No
	1 (+ IJV)£		YES (Brachial Plexus)	No	No	No	YES
Anterior Jugular Vein (AJV)	3		No x 3	No x 3	No x 3	No x 3	YES x 3
	3		No x 3	No x 3	No x 3	No x 3	No x 3
		1 (+ RV)ë	No	No	No	No	No
External Jugular Vein (EJV)	2		No x 2	No x 2	No x 2	No x 2	YES x 2
		1	No	No	No	No	No
	1		No	No	No	No	No
Minor Arteries (Occipital, Lingual, Thyroid)	1 (Occipital)		No	No	No	No	YES
	1 (Lingual)		No	No	No	No	No
		1 Superior Thyroid	No	No	No	No	No
		1 Superior Thyroid	No	No	No	No	No
		1 Lingual	No	No	No	No	No
Minor Veins (Facial, Unnamed)	1 FV (+ CCA)¶		No	No	No	No	YES
	4 Unnamed		No x 4	No x 4	No x 4	No x 4	No x 4
		1 FV (+TCT)ñ	No	No	No	No	No
Non-Cervical Vascular Injury							
Thyrocervical Trunk (TCT)		1 (+ FV)ñ	No	No	No	No	No
Left Ventricle		1 (+ AJV)ë	No	No	No	No	No
Right Ventricle (RV)		1	No	No	No	No	No

PNI: Peripheral nerve, CSCI: Cervical spinal cord, CSFx: Brain and cervical spine fracture; CSI: Cervical spine immobilization, indicating presence on admission (YES) or absence on admission (No); ECA: External Carotid Artery; IJV: Internal jugular vein; FV: Facial Vein; CCA: Common carotid artery; AJV: Anterior jugular vein; C6: Cervical vertebra six
Same superscripts indicate same patient

by the CCA (N=7) and the ECA (N=5). Four cervical ICA injuries were noted. Seven of the twenty-one patients with major vascular injury had an associated neurologic deficit, four with peripheral neurologic injuries, two with brain injury and one with a CSCI. Major vascular injury was a significant risk for brain injury (p=0.01; RR 10.21; 95% CI 6.67-15.65) but not CSCI (p=0.99). CSFx occurred in three patients but was not associated with major vascular injury (p=0.99). Two of these patients had stable CSFx and one of these had two major vascular injuries (Table 2a). One patient with bilateral VA injuries had an unstable CSFx with quadriplegia. 10 patients with major vascular injury received CSI.

Indirect neurologic injury occurred in four patients surviving to discharge (Tables 3 and 4). Two of these patients incurred indirect brain injury secondary to arterial flow disruption. One of these two patients developed a right sided cerebrovascular infarction secondary to stab wound (SW) disruption of right ICA blood flow. The other of these two patients also developed a right cerebrovascular infarction that was secondary to a gunshot wound (GSW) disruption of right CCA blood flow. The third patient with indirect central neurologic injury involved CSCI secondary to shock with central cord ischemia noted on magnetic resonance imaging (MRI) from cervical vertebral level three to five (C3-C5). The fourth patient incurred an indirect C5 central cord syndrome. CSI was performed in three of the patients with indirect neurologic injury: both carotid injuries and the C5 central cord syndrome. CSI was associated with an increased risk for indirect central neurologic injury (p<0.001; RR 1.635; 95% CI 1.23-1.95).

Prehospital mortality was defined as death prior to arrival in the emergency department and also termed dead on arrival (DOA). Early death was defined as death following arrival in the emergency department but prior to being admitted to the hospital. Late date was defined as in hospital death excluding early and prehospital deaths.

Table 3. Cervical Spinal Cord Injuries indicating the Number of Patients at Each Institution, the Mechanism of Injury (Direct versus Indirect) and the Presence or Absence of Cervical Spine Immobilization.

CERVICAL SPINAL CORD INJURIES	CHARITY	HURLEY	INJURY	CSI
Complete	2	1		
C2 Quadriplegia	1		Direct	Yes
C5 Quadriplegia		1	Direct	Yes
C6 Quadriplegia	1		Direct	Yes
Incomplete	4	2		
C8 Paraplegia	1		Direct	Yes
C6 Temporary Quadriplegia	1		Direct	Yes
C6 Contusion		1	Direct	Yes
C3 Central Cord Syndrome		1	**Indirect**	No
C5 Central Cord Syndrome	1		**Indirect**	Yes
TOTAL	5	3	6 Direct 2 Indirect	7 Yes 1 No

Table 4. Classification of Indirect Central Neurologic Injuries as Brain or Cervical Spinal Cord with Mechanism of Injury.

Indirect Central Neurologic Injuries	Number of Patients	Mechanism	CSI
Brain Injuries	**2**		
R CVA	1	SW R ICA	YES
R CVA	1	GSW R CCA	YES
Cervical Spinal Cord Injury	**2**		
Central Cord Syndrome	1	Shock	YES
Central Cord Syndrome	1	Shock	NO

GSW: Gunshot Wound; SW: Stab Wound; R: Right; ICA: Internal Carotid Artery; CCA: Common Carotid Artery; CVA: Cerebrovascular Accident

Prehospital death, or DOA, was pronounced in seven patients. CSI was performed in six of the DOA patients. CSI was performed in 13 of the 18 early decedents, and in eight of the ten late decedents (Table 5). Cardiac arrest was the most common cause of death in the early group indicating exsanguination and /or hypoxia as the cause of death. Death from traumatic cardiac arrest was considered penultimate indirect neurologic injury. Direct CSCI occurred in six patients (Table 3). All of these patients received CSI. Patients with direct brain injury were excluded.

14 patients presented with 16 airway injuries (Table 4). Six of these patients received CSI of which two had compound airway injuries. Two patients underwent emergent cricothyroidotomy upon arrival and one of these patients arrived in CSI. 12 patients (6%) presented with 17 peripheral nerve injuries (PNI) (Table 6). Respiratory injury was associated with PNI (p=0.04; RR 4.33; 95% CI 1.32-14.22) but not CSFx (0.61), CSI (0.79), major vascular injury (0.22) or central neurologic injury (0.99).

Brain injury occurred in two patients, one with a CSFx and one without. Brain injury was neither associated with CSFx (p=0.35) nor CSCI (p=0.08). Hypoperfusion occurred in four patients and was a significant risk factor for indirect central neurologic injury (p<0.00001; RR 38.4; 95% CI 16.17-91.2). Statistical comparisons were summarized on Table 7.

Discussion

Central neurologic injury with penetrating cervical trauma was more likely associated with, rather than prevented by, CSI in this study. This study constituted the first known neurologic risk/benefit analysis of CSI. CSI in this study demonstrated a significant increase in indirect neurologic injury without any possibility of prophylaxis against progression of indirect neurologic injury from unstable CSFx. The juxtaposed non-incidence of salvageable direct CSCI amenable to CSI prophylaxis with the marked incidence of treatable vascular trauma and shock (combined incidence vascular trauma and shock %). Appeared a likely source of this increased risk of indirect central neurologic injury. CSI iatrogenia may have facilitated indirect neurologic injury by negatively impacting the "ischemic penumbra" [2-6,15,22-24,27,29,30].

Two cases of unstable CSFx were found in this study which was 1% of all patients. Both of these patients had associated complete neurologic devastation which made prophylactic CSI inappropriate. The true incidence of indicated CSI in this study then was 0%. Four patients in this study developed indirect central neurologic injury

Table 5. Patient fatalities categorized according to time position of post – injury occurrence with noted presence (+ CSI) or absence (- CSI) of cervical spine immobilization (CSI).

PATIENT FATALITIES		
TIME OF DEATH	**+ CSI**	**-CSI**
Dead on Arrival (Prehospital)	**6**	**1**
Emergency Department Deaths (Early)	**13**	**5**
Cardiac Arrest	11	4
Exsanguination	1	1
Traumatic Brain Injury	1	0
Died in Hospital (Late)	**8**	**2**
Cardiac Arrest in OR	3	1
Anoxic Brain Injury	2	0
Multi-System Organ Failure	2	0
Right Cerebral Infarct	1	0
Traumatic Brain Injury	0	1

Table 6. Respiratory tract injuries indicating respective treatment institutions and associated injuries.

Respiratory Tract Injury	Charity	Hurley	PNI	CSCI	CSFx	Vascular Injury	CSI
Trachea		1	No	No	No	No	No
		1	No	No	No	No	No
		1	YES (Laryngeal)	No	No	No	YES
	1		YES (Laryngeal)	No	No	No	YES
	1		No	No	No	No	No
Pharynx	1 (+ Hypopharynx)[£]		No	No	No	No	YES
	1		No	No	No	YES (ECA)	No
	1		No	No	No	No	No
	1		No	No	No	YES (ECA)	YES
		1	No	No	No	No	No
Hypopharynx	1 (+ Pharynx)[£]		No	No	No	No	YES
	1 (+ Larynx)[¥]		No	No	No	No	YES
		1	No	No	No	No	No
Larynx	1		YES (Brachial Plexus)	No	No	No	YES
	1 (+ Hypopharynx)[¥]		No	No	No	No	YES
	1		No	No	No	Yes (IJV)	No

PNI: Peripheral nerve; CSCI: Cervical spinal cord; CSFx: Brain and cervical spine fracture; CSI: Cervical spine immobilization, indicating presence on admission (YES) or absence on admission (No); ECA: External Carotid Artery; CCA: Common Carotid Artery; IJV; Internal Jugular Vein
Same superscript indicates same patient

Table 7. Conditional Risks. CSCI – Cervical Spinal Cord Injury.

Clinical Condition	*p*	Relative Risk	95% Confidence Interval
Brain Injury v. CSCI	NS	N/A	N/A
Brain Injury v. CSFx	NS	N/A	N/A
Indirect Cent Neuro Inj v. Hypoperfusion	**< 0.00001**	**38.40**	**16.17 – 91.20**
CSCI v. CSFx	**< 0.00001**	**20.56**	**8.44 – 26.47**
CSCI v. Unstable CSFx	**0.001**	**32.33**	**5.21 – 32.33**
Major VI v. Brain Injury	**0.01**	**10.21**	**6.67 – 15.65**
Major VI v. CSCI	NS	N/A	N/A
Major VI v. PNI	NS	N/A	N/A
Major VI v. CSFx	NS	N/A	N/A
Major VI v. CSI	NS	N/A	N/A
CSI v. Indirect Cent Neuro Inj	**< 0.001**	**1.635**	**1.23 – 1.95**
Respiratory Injury v. Brain Injury	NS	N/A	N/A
Respiratory Injury v. CSI	NS	N/A	N/A
Respiratory Injury v. CSFx	NS	N/A	N/A
Respiratory Injury v. PNI	**0.04**	**4.33**	**1.32 – 14.22**
Respiratory Injury v. Major VI	NS	N/A	N/A

VI: Vascular Injury; CSFx: Cervical Spine Fracture; CSI; Cervical Spine Immobilization; Indirect Cent Neuro Ing: Indirect Central Neurologic Injury; PNI: Peripheral Nerve Injury
Significance was accepted

which equaled an incidence of 2.1%. Two of these four patients incurred indirect neurologic injury from disruption of carotid perfusion while the other two injuries resulted from shock. Early repair of carotid vascular disruption and resuscitation has marked efficacy in preventing consequential indirect neurologic injury [11-13,29-35]. None of the patients with indirect central neurologic injury had unstable CSFx, making the use of CSI in 75% of these patients inappropriate. The 2.1% incidence of indirect central neurologic injury amenable to resuscitation and surgical intervention then exceeded the 0% incidence of direct central neurologic injury amenable to CSI prophylaxis.

CSI has demonstrated iatrogenia. CSI has been shown to cause treatment delay, obscure injury, impair care delivery, respiration, increase intracranial pressure, effect negative outcomes and exacerbate and impede control of hemorrhage in penetrating cervical trauma [2-6,15,22-24,27,29,30]. Both patients with indirect central neurologic injuries secondary to carotid arterial disruption received CSI. One patient with an indirect C3 central cord syndrome secondary to shock also received CSI. Iatrogenic consequence may have contributed to this study's finding that CASI was strongly associated with an increased risk for indirect neurologic injury (p<0.001; RR 1.635; 95% CI 1.23-1.95).

Direct CSCI occurred in 3.1% of all surviving patients. Major vascular injury occurred in 11% of surviving patients which constituted a nearly 3.5-fold greater incidence than direct CSCI. Prior studies reported major vascular injury complicating over 25% of penetrating cervical trauma [1,7-9,17]. The source of discrepancy between the incidence of major vascular injury in this study and other reported studies was not clear but may represent statistical variance. Alternatively, this percentage difference may reflect inclusion loss secondary to death in the study cohort. All deaths in this study occurred in Charity patients wherein CSI was regularly practiced as opposed to Hurley patients where in CSI was restricted. Prior analysis of the Charity cohort (Vanderlan) reported vascular and respiratory compromise as the likely source of fatality. The incidence of vascular injury with penetrating cervical trauma when all 35 fatalities were added to the number of survivors with known vascular injury (21+35=56/196+35). The incidence also more closely approximated the prior reported incidence if patients that were excluded as early and late deaths were included with the known vascular injury survivors

(21+28/196+28). This study's increased risk of CSI associated indirect central neurologic injury have proved more pronounced had the study incidence of major vascular injury approximated a 25% noting that central neurologic injury occurs in approximately 30% of patients with penetrating cervical penetrating trauma [29,35].

Brain injury occurred in close association with major vascular injury (p=0.01). Hypoperfusion also proved a significant risk factor (p<0.00001) for central neurologic injury. Both brain injury and hypoperfusion occurred independent of CSFx and CSCI. While the relative risk of brain injury associated with major vascular injury (RR 10.21) was 1/3 the relative risk of central neurologic injury associated with an unstable CSFx (RR 32.33), primary concern for prevention of brain injury superseded concern for prophylactic CSCI because all patients with unstable CSFx were neurologically devastated. The relative risk of central neurologic injury from hypoperfusion (RR 38.4) was nearly twice the risk of central neurologic injury associated with CSFx (RR 20.56). Correction of hypoperfusion in this study should have superseded concern for CSI prophylaxis because of the markedly higher relative risk of injury combined with CSI prophylactic inefficacy. CSI was provided to 47.62% of all patients in this study with major vascular injury, none of whom had unstable CSFx. CSI was provided to three of the four patients in this study who developed ischemic central neurologic injury. CSI associated risk for indirect neurologic injury may have resulted from exacerbation of ischemic complications by increasing the intracerebral pressure and causing venous outflow obstruction [36-39].

"Ischemic penumbra" is a term that developed to describe an area of nonviable neurologic tissue surrounded by variably viable ischemic neurologic tissue (Figure 1). Resuscitation and surgical strategies to expeditiously restore perfusion to salvageable tissue within the "penumbra" are keys to neurologic salvage as hemodynamic stabilization of neurologically injured patients has been reported as critical for neurologic outcome [11,12]. Aggressive fluid resuscitation and selective use of vasopressors demonstrated improved neurologic outcomes in both intracranial and extracranial central neurologic tissues, emphasizing the need for rapid definitive intervention [11,12]. Early repair of cervical vascular injury has also been shown to improve neurologic outcomes secondary to restoration of intracranial blood flow which again emphasizes emergent transport of these patients to definitive care [13,29,31-35].

The reported incidence of direct neurologic injury in penetrating cervical trauma ranges from <1%-7% [1,3,9,40]. Direct CSCI was noted in 3.06% of surviving patients in this study and found agreement with the prior reported incidence range. The incidence of direct CSCI with unstable CSFx in this study was 1%. Rhee, et al., reported in a large meta-analysis that concurrent CSFx and CSCI with penetrating cervical trauma was an extremely rare event with an incidence of less than 0.6% [3]. Lustenberger, *et al.,* recently reported a similar incidence of concurrent CSFx and CSCI at 0.4% for penetrating cervical trauma and specifically noted this concurrence is commensurate with severe neurologic impairment with no benefit recognized from surgical stabilization [41]. The slightly greater incidence of CSCI and unstable CSFx in this study compared to those cited was unclear but may be isolated statistical variance. The incidence of CSCI and unstable CSFx in this study however, agrees with prior studies that no neurologic salvageability noted in the study cohort [42,43].

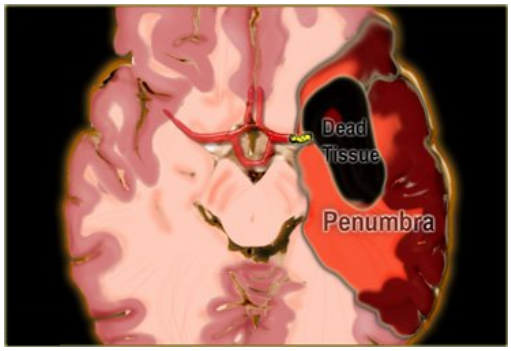

Figure 1. Graphic representation of ischemic penumbra depicting central necrosis surrounded by potentially salvageable ischemia [42].

Possible benefit from deferring CSI in this study greatly exceeded the less than 1% possibility of benefit since none of the 94 (48% of all patients) of surviving patients who received CSI with an associated unstable CSFx were neurologically salvageable. While none of these 94 patients may have benefitted from CSI, 100% of them were exposed to the risk of CSI iatrogenia. Deferring CSI might have especially benefitted the 15% of patients in this study that were critically injured for whom undelayed transport to definitive care was most important35. The importance of deferring CSI in these severely injured patients became clear when recognizing that CSI under ideal conditions, and excluding errors, required approximately 6 mins while a 10% increase in mortality was associated with each ten-min delay in reaching definitive car [23,25]. These patients were then viewed as unsalvageable. Early and late deaths were included in the analysis of indirect neurologic injury because the cause of death in this cohort was previously determined as exsanguination and/or hypoxia [27]. Justification for inclusion of early and late deaths rested on the theory that within the ischemic penumbra there is some point on the continuum of injury where irreversible indirect neurologic injury occurs and the ischemic penumbra loses all salvageability.

DOA patients were excluded from analysis because cardiopulmonary resuscitation was not associated with CSI in this patient cohort, indicating possible rapid prehospital death [27]. Prehospital death from penetrating cervical trauma may have resulted from severe hemorrhage, respiratory injury, neurologic injury or a combination of these. Autopsy results were not available for patients in the DOA category so these patients were excluded from the analysis.

Respiratory tract injuries have been reported in up to 20% of penetrating neck injuries with a maximum mortality of 33% [1,7,17]. Respiratory physiology and obtainment of a definitive airway had both been previously shown to be impaired by CSI [2,6,15,16]. Consideration was then given to examining the relationship between respiratory tract injuries and indirect neurologic injury in this study. This incidence of respiratory tract injuries in this study was 7.1%. CSI was performed in six patients with respiratory injuries of which two had commensurate major vascular injuries. The association between respiratory injury, major vascular injury and CSI was insignificant. CSFx did not occur in any patient with respiratory tract injuries. Respiratory tract injury was also not associated with central neurologic injury. Peripheral neurologic injury was a significant risk for respiratory tract injury (p=0.04; RR 4.33; 95% CI 1.32-14.22) which seemed consistent with cervical anatomy.

Calculating the effectiveness of CSI in penetrating cervical trauma could not be determined in this study because unstable CSFx did not occur in any patients with neurologic salvageability. The absence of cervical instability occurring with neurologic salvageability in this

study was predictable from the surveyed literature and may reflect an incidence so remote as to be undiscoverable. Only one case of cervical spine instability resulting from penetrating cervical trauma in a neurologically intact patient has been reported and the accuracy of this report has been challenged (Vanderlan) [20]. Interestingly, the patient's cervical collar was removed in transit for control of hemorrhage without resultant central neurologic injury [20]. With only a single case report of a neurologically intact patient with cervical spine instability secondary to penetrating cervical trauma, the true number needed to treat (NNT) was hypothesized to lie between zero and negative infinity (0>NNT>-∞). The probability of the NNT being negative seemed even more likely given that this sole case entailed discontinuation of CSI without subsequent neurologic consequence. The absolute risk increase for central neurologic injury associated with CSI was 18.69% in this study, paired with a NNH of 5.3.

Haut et al. [30] retrospectively reviewed the National Trauma Data Bank and calculated the NNT and NNH for CSI in penetrating cervical trauma [30]. Possible benefit was defined "as patients with incomplete spinal cord injury that underwent operative repair". This definition permitted NNT and NNH calculations but provided an inaccurate assessment of CSI effectiveness because (1) CSI only benefits neurologically intact patients with unstable CSFx, (2) operative repair in penetrating cervical trauma does not constitute cervical spine instability, (3) cervical spine instability in penetrating cervical trauma usually results from necessary surgical iatrogenia, and (4) incomplete spinal cord injury had not been definitively proven to occur commensurately with cervical spine instability from penetrating cervical trauma.

The absence of cervical spine instability with commensurate neurologic salvageability and the resultant inability to accurately determine NNT were limitations of this study. This study was also limited by a relatively small sample size and by all the inherent problems of a retrospective study.

Conflicts of interest

The authors have no conflicts of interest.

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