Characterization of the spectrum of hemodynamic profiles in trauma patients with acute neurogenic shock

Richard L. Summers MD,*, Stephen D. Baker MD, Sarah A. Sterling MD, John M. Porter MD, Alan E. Jones MD

Department of Emergency Medicine, University of Mississippi Medical Center, Jackson, MS 39216, USA

Department of Surgery, University of Mississippi Medical Center, Jackson, MS 39216, USA

Keywords: Neurogenic; Shock; Trauma; Spinal injury; Hemodynamics; Profile

Abstract

Objective: Neurogenic shock considered a distributive type of shock secondary to loss of sympathetic outflow to the peripheral vasculature. In this study, we examine the hemodynamic profiles of a series of trauma patients with a diagnosis of neurogenic shock.

Methods: Hemodynamic data were collected on a series of trauma patients determined to have spinal cord injuries with neurogenic shock. A well-established integrated computer model of human physiology was used to analyze and categorize the hemodynamic profiles from a system analysis perspective. A differentiation between these categories was presented as the percent of total patients.

Results: Of the 9 patients with traumatic neurogenic shock, the etiology of shock was decrease in peripheral vascular resistance (PVR) in 3 (33%; 95% confidence interval, 12%-65%), loss of vascular capacitance in 2 (22%; 6%-55%) and mixed peripheral resistance and capacitance responsible in 3 (33%; 12%-65%), and purely cardiac in 1 (11%; 3%-48%). The markers of sympathetic outflow had no correlation to any of the elements in the patients’ hemodynamic profiles.

Conclusions: Results from this study suggest that hypotension of neurogenic shock can have multiple mechanistic etiologies and represents a spectrum of hemodynamic profiles. This understanding is important for the treatment decisions in managing these patients.

© 2013 Elsevier Inc. Open access under CC BY-NC-ND license.

1. Introduction

Approximately 7% to 10% of all patients with trauma spinal cord injuries develop a condition of neurogenic circulatory shock [1,2]. Traditionally, neurogenic shock has been thought of as a distributive type of shock secondary to a reduction of vascular tone and peripheral resistance because of the loss of sympathetic input [3]. In reality, the precise circulatory mechanisms involved have not been well characterized, and clinically, neurogenic shock is simply defined as hypotension and bradycardia with the exclusion of other causes of shock [1–3]. The study of neurogenic shock has been complicated by its association with conditions of trauma that often include other more likely causes for hypotension [4]. In addition, the sympathetic response to spinal cord injury is known to follow a 4-phase longitudinal evolution, which results in a varied hemodynamic responsiveness over time [5].
However, the early management of the neurogenic shock state has been shown to be critically determinative to the outcome of many of these patients [6,7]. Therefore, it is important that clinicians acquire an improved understanding of the physiologic etiology of the acute phase of neurogenic shock if effective and targeted management strategies are to be developed.

In this study, we collected some detailed early hemodynamic data from patients in whom a diagnosis of acute neurogenic shock had been made. This information was then examined using a system analysis approach to determine the likely circulatory physiologic etiology of their shock state and categorize their hemodynamic profiles into a spectrum of possible causative mechanisms.

2. Methods

A convenience sample of adult patients (> 18 years old) with a clinical diagnosis of acute neurogenic shock (acute spinal cord injury with hypotension not attributable to any other etiology) was studied in the early stages of their emergency department resuscitation at an academic medical center that see approximately 70 000 patients per year and serves as a level 1 trauma center for a large catchment area. Although all of these patients met the traditional criteria for the diagnosis of neurogenic shock (systolic blood pressure < 100 mm Hg and heart rate < 80 beats per minute), the goal of the study was to more extensively characterize the hemodynamics so that the mechanisms of circulatory shock could be better clarified. The hemodynamic variables collected included heart rate, systolic and diastolic blood pressure, and cardiac output that were obtained using impedance cardiography (Philips Medical ICG Monitor Model 2004; Philips Medical Systems, 3000 Minuteman Road, Andover, MA) and traditional emergency department vital sign determinations [8,9]. Additional hemodynamic variables such as systemic vascular resistance, pre-ejection period (PEP) (cardiac PEP), and left ventricular ejection time (LVET) were derived from these clinical measurements [9]. The reference range for the impedance measure PEP is between 0.08 and 0.15 seconds and is usually considered to be indicative of a peripheral sympathetic outflow. The reference range for impedance measure LVET is 0.25 to 0.55 second and is usually indicative of the cardiac sympathetic outflow. The measurements of cardiac output and systemic vascular resistance were indexed to the body surface area of the individual patient for more accurate comparisons. The data were collected under the auspices of a University of Mississippi Center Institutional Review Board–approved protocol.

2.1. Computational platform and system analysis protocol

The hemodynamic variables collected were used in a system analysis methodology to determine the most probable mechanistic etiology of the neurogenic circulatory shock for each individual patient. In this process, each individual patient’s clinically observed hemodynamic profile was matched with one of a variety of potential cardiovascular derangements as predicted by the model and based on the known levels of innervation of the individual circulatory elements (heart, peripheral vasculature, capacitance vessels).

The computational methodology used in the system analysis uses a well-established computer model of human physiology (Guyton/Coleman/Summers model) developed over the past 30 years that describes the integrative cardiovascular physiologic functioning of a virtual subject [10–13]. This model and methodology have previously been used in numerous studies that were intended to provide a more detailed understanding of the physiologic mechanisms involved in common clinical conditions [13–16]. In addition, several versions of this model have been previously demonstrated to accurately predict hemodynamic changes seen during hypotensive states [11,12]. This evidence suggests that the model can be used as a platform for the theoretical analysis of shock states. The model contains a variety of parameters that describe the detailed interactions of systemic, organ, and tissue cellular physiology and metabolism based upon basic physical principles and established biologic relationships. The structure of the model incorporates the cardiovascular and neurogenic physiologic responses to changes in pressures, flows, and hydraulics within the circulatory system as well as the utilization and mass balance fluctuations of metabolic substrates. The details of this model structure are beyond the scope of the current article and have been described in previous publications [11,13].

The system analytic procedure using the computational platform involves recreating the clinical scenario for a virtual subject with a spinal injury in an in silico environment [17,18]. We performed a series of simulation studies in which the efferent neurogenic input into the circulatory system was muted at varying degrees of severity for 1 of the 3 separate key controlling cardiovascular elements (heart, capacitance vessels, or peripheral arterial circulation). The computer simulation was allowed to run until the model system blood pressure reached a steady state that was consistent with neurogenic shock. The general circulatory profiles for each of these shock state neurogenic etiologies were described using clinically determinable parameters as listed in Table 1. The values for the measured patient parameters were then compared with those in the table to differentiate and categorize each individual patient as to the dominant etiology of their neurogenic shock.

3. Results

Over the period of this study, a total of 9 patients presented to the emergency department in which it was
thought clinically that traumatic neurogenic shock was the causation of their hypotension. The hemodynamic shock profiles and the associated mechanisms of origin as determined by the system analysis are listed in Table 2. The etiology of the neurogenic shock was found by system analysis to be due to a decrease in PVR in 3 of the cases (33%; 95% confidence interval [CI], 12%-65%), a loss of vascular capacitance in 2 cases (22%; 95% CI, 6%-55%), and a mixed combination of peripheral resistance and capacitance responsible in 3 of the cases (33%; 95% CI, 12%-65%). In only 1 subject with neurogenic hypotension was the etiology thought to be purely cardiac in origin (11%; 95% CI, 3%-48%). The markers of sympathetic outflow (PEP and LVET) were found to have no correlation to any of the elements in the patients’ hemodynamic profiles.

4. Discussion

Neurogenic shock is typically considered a clinical diagnosis of exclusion in patients with spinal cord injury [3]. A diagnosis of this condition depends on the observation of hypotension and possibly bradycardia without any other determined etiology [1,2]. Although neurogenic shock is commonly described as a distributive type of shock, secondary to a loss of systemic vascular resistance, the precise circulatory elements responsible for the hypotension are often uncertain and undetermined [3]. In this study, a unique analytic methodology using a system-based approach was used to characterize the hemodynamic profiles in a cohort of patients with acute neurogenic shock due to trauma [18]. From this analysis, we found a variety of circulatory components that interface with neurogenic control mechanisms that were thought to be involved in the development of the observed hypotension [19,20]. This finding suggests that clinical neurogenic shock is really a spectrum of hemodynamic states all of which result in hypotension but might require decidedly different management strategies [4-7,21]. Management protocols that include the collection of more extensive hemodynamic data have been shown to improve outcomes for this group of patients. Therapies that target the specific circulatory mechanism implicated in the causation of the shock state should be thoughtfully selected as opposed to a generalized approach to management.

There are a number of limitations to this study that should be noted. Although the combination of hypotension and bradycardia (systolic pressure <100 mm Hg and heart rate <80 beats per minute) is thought to be classic for neurogenic shock, the incidence of this finding is really very uncommon [1]. Our analysis determined that only 1 of the 9 patients had a combination of both vascular and cardiac involvement. It is interesting to note that the analysis resulted in a suggested heart rate range of less than 60 beats per minute for a neurogenic shock of cardiac origin. This is much less than the less than 80 beats per minute that is commonly used clinically in the diagnosis of this condition. Despite this discrepancy, 22% of the patients in our small sample had cardiac involvement, which is very similar to that noted in other studies [1,2,4]. However, the historic choice of cutoff of 80 beats per minute is arbitrary and based upon an

<table>
<thead>
<tr>
<th>Table 1</th>
<th>The general ranges of some specific variables that typified the categories of circulatory origin as suggested from the overall systems analyses of the hemodynamic profiles of patient with neurogenic shock</th>
</tr>
</thead>
<tbody>
<tr>
<td>General ranges of hemodynamic variables typifying the origin of acute neurogenic shock</td>
<td></td>
</tr>
<tr>
<td>Peripheral origin</td>
<td>SVRI &lt; 2000 dyne-sec/cm²/m²</td>
</tr>
<tr>
<td>Capacitance origin</td>
<td>CI &lt; 2.8 lpm/m² and HR &gt; 60 beats per min</td>
</tr>
<tr>
<td>Cardiac origin</td>
<td>CI &lt; 2.8 lpm/m² and HR &lt; 60 beats per min</td>
</tr>
<tr>
<td>Mixed origin</td>
<td>Combination of above</td>
</tr>
<tr>
<td>HR indicates heart rate.</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Hemodynamic profiles of patients with acute neurogenic shock</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.</td>
<td>Sex</td>
</tr>
<tr>
<td>1</td>
<td>m</td>
</tr>
<tr>
<td>2</td>
<td>m</td>
</tr>
<tr>
<td>3</td>
<td>m</td>
</tr>
<tr>
<td>4</td>
<td>m</td>
</tr>
<tr>
<td>5</td>
<td>f</td>
</tr>
<tr>
<td>6</td>
<td>m</td>
</tr>
<tr>
<td>7</td>
<td>m</td>
</tr>
<tr>
<td>8</td>
<td>m</td>
</tr>
<tr>
<td>9</td>
<td>m</td>
</tr>
<tr>
<td>AVG</td>
<td></td>
</tr>
</tbody>
</table>

m indicates male; f, female; MVC, motor vehicle collision; MAP, mean arterial pressure in millimeters of mercury; SBP, systolic blood pressure in millimeters of mercury; DBP, diastolic blood pressure in millimeters of mercury; HR, heart rate is in beats per minute; CI, cardiac index is in liters per minute per body surface area in square meter; SVRI, systemic vascular resistance index dyne-seconds per square centimeter per body surface area in square meter; LVET and PEP in seconds.
expected response to the hypotension. Unfortunately, heart rate is known to correlate poorly with hypotension and is frequently not a factor especially if the neurologic reflex arc is disrupted [22]. It also is important to remember that a loss of cardiac sympathetic input results in an unopposed vagal tone. Therefore, it might be expected that a true neurogenic shock with a component of cardiac origin would have a heart rate in keeping with current common definitions of bradycardia (<60 beats per minute) [3].

It is also important that this study contains only a small number of patients and a larger study might discern a significantly different spectrum. The collection of detailed hemodynamic information (including cardiac output) in the early emergency stages of this disease process and before aggressive therapies are initiated that obscure the true circulatory state is very difficult logistically. A large scale study would require a multicenter effort over an extended period to capture significant numbers of patients in these early stages. Such a study might also clarify what levels of spinal cord injury are commonly associated with derangements of specific elements of the circulation. There is no apparent correlation between the spinal level involved and the apparent effected circulatory mechanisms that could be derived from our small study. It is possible that the dynamic processes accompanying this type of traumatic injury may preclude such an association. It is known that acute spinal injury can result in paradoxical neurogenic reflexes and unpredictable sympathetic discharges [23–25]. The intent of the current study was to simply demonstrate that the general phenotypical state of clinical neurogenic shock actually can be the result of a diverse variety of hemodynamic conditions arising from distinctly different physiologic mechanisms. An understanding of the spectrum nature of this disease process might assist clinicians in more informed management decisions.

Lastly, we should note that some the measurement tools used in the collection of the hemodynamic variables also have some inherent limitations that could impact the analysis. Arterial pressures measured by blood pressure cuffs are often sporadic and unreliable when compared with direct measurements using arterial catheters. In addition, impedance cardiography is a well-accepted and practical technique for determining cardiac output, but it is not a “gold standard” and can be subject to significant measurement error [9]. The advantage of using both of these noninvasive techniques in this study is that they could be readily implemented in the very early stages of the clinical shock before significant resuscitative measures were initiated.

5. Conclusions

Through the process of diagnosis, patients are sorted into disease categories that define etiology of their condition. Clinicians can then make management decisions based upon knowledge of the pathophysiology of the type of disease process and the mechanisms expected to result in the observed abnormalities. Neurogenic shock has traditionally been considered as a particular pathophysiology resulting from a singular causation. The current study suggests that neurogenic shock really represents a potential spectrum of hemodynamic derangements that can occur after a spinal cord injury. Although all the patients in this study presented with hypotension, the circulatory elements involved in the etiology of the hemodynamic condition were quite varied.

References


