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

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**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.

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## 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].

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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

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