Hemodynamic Patterns of Blunt and Penetrating Injuries

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BACKGROUND: The aims of this prospective observational study were to describe early hemodynamic patterns of blunt and penetrating truncal injury and to evaluate outcomes prediction using noninvasive hemodynamic monitoring with a mathematical model tested against actual in-hospital outcomes. The hypothesis was that traumatic shock is a circulatory disorder that can be monitored by noninvasive hemodynamic parameters that reflect cardiac, pulmonary, and tissue perfusion functions.

STUDY DESIGN: The cardiac index (CI), heart rate (HR), mean arterial pressure (MAP), pulse oximetry (SapO₂), transcutaneous oxygen tension indexed to FiO₂ (PtcO₂/FiO₂), and carbon dioxide (PtcCO₂) tensions were monitored beginning shortly after emergency department admission in 657 emergency patients with severe blunt and penetrating chest, abdominal, and extremity trauma. Of these, 113 patients had associated head injury, and these patients were analyzed separately. A search and display mathematical model, with a decision support program, was based on continuous online, real-time, noninvasive hemodynamic monitoring.

RESULTS: There were similar patterns in the blunt and penetrating injuries; the cardiac index, mean arterial pressure, pulse oximetry, transcutaneous oxygen tension indexed to FiO₂, and survival probability values of the survivors were significantly higher (p < 0.01) than the corresponding values of those who died, although heart rate and carbon dioxide tension were higher in the nonsurvivors during the first 24 hours after their emergency department admission. These patterns occurred more rapidly in patients with penetrating injuries. After initial resuscitation in the emergency department, results were correlated with actual outcomes at hospital discharge and found to be 88% correct.

CONCLUSIONS: Early noninvasive hemodynamic monitoring with a computerized information system provided a feasible pattern recognition program for outcomes prediction and therapeutic decision support. (J Am Coll Surg 2006;203:899–907. © 2006 by the American College of Surgeons)

Acute life-threatening circulatory disorders have the distinct advantage that their sequential circulatory patterns can be visually displayed by continuous, online, real-time, noninvasive hemodynamic monitoring systems. Invasive pulmonary artery thermodilution (Swan-Ganz) catheters provide the maximum circulatory data but require intensive care unit conditions. Because of this requirement, goal-directed therapy may be delayed until the patient has been transferred to an ICU. But noninvasive hemodynamic monitoring, which can be used anywhere in the hospital, may identify the early circulatory deficiencies that subsequently lead to shock, organ failure, and death. In addition, the survival probability (SP) may be predicted by an information system that analyzes early clinical-hemodynamic patterns in patients with the same diagnostic criteria and covariants. Illness severity may be quantitatively evaluated by the SP, the most severely ill patients having the lowest SP values and the least seriously ill patients having the highest values.

When invasive monitoring is started late in the course of illness or after onset of an organ failure, there is no outcomes improvement with goal-directed therapy and pulmonary artery catheters. By contrast, early nonin-
Noninvasive monitoring identifies hemodynamic deficits as soon as the patient enters the emergency department (ED). Time is an important factor in the resuscitation and expeditious management of acute emergency patients because delays in correcting circulatory deficiencies of shock and trauma lead to organ failures and death. Similar to early diagnosis and therapy for cancer, early diagnosis and therapy for severe circulatory problems may be more cost-effective than therapy delayed until late stages, when the diagnosis is more certain. The hemodynamic patterns of severe head injury patients (Glasgow Coma Score ≤ 8) and patients with brain death must be analyzed separately because they have high cardiac index (CI) with increased tissue oxygenation, attributed to the loss of central vasoconstrictive mechanisms with unopposed peripheral vasodilatation.

Recently, Bayard and colleagues and Shoemaker and associates developed a mathematical model that used a large database of noninvasively monitored hemodynamic variables to predict outcomes for acute emergency patients. This article analyzes the survival probability of patients with severe injuries, separate from those with severe head injury and brain death.

**METHODS**

**Clinical series**

We studied 657 consecutive noninvasively monitored blunt and penetrating trauma patients. Of these, 221 patients had chest injuries, 266 had abdominal trauma, 175 had both chest and abdominal trauma, 57 had extremity injuries, and 113 had head injuries associated with their truncal trauma. Patients with severe head injury and those with brain death were evaluated separately because they had different hemodynamic patterns. In addition, hemodynamic patterns of this series were calculated with and without associated head injury.

There were 570 survivors and 87 nonsurvivors; hospital mortality was 13.2%. Noninvasive monitoring was begun in the ED shortly after admission and the patients were followed to the radiology suite, operating room (OR), and ICU at frequent periods until stable or unresponsive to therapy. The pulmonary artery was catherized when indicated after the patient arrived in the OR or ICU. The time of monitoring, time of operation, time of ICU admission, and discharge or death were recorded relative to time elapsed after ED admission. We also included in the database age; gender; presence of sepsis; Glasgow Coma Score (GCS); Injury Severity Score (ISS); primary bodily injuries; covariates such as sepsis, diabetes, and earlier cardiac problems; hemodynamic patterns by invasive and noninvasive methods; complications; organ failures; hospital days; ICU days; and hospital outcomes. Table 1 lists the salient clinical features. The Institutional Review Board approved the protocol.

**Noninvasive hemodynamic monitoring**

We evaluated the patterns of hemodynamic values after observing and recording the continuous displays of noninvasively monitored cardiac, respiratory, and tissue perfusion functions. Data were downloaded every 30 seconds, averaged over 5-minute intervals, and entered into the database. When consistent hemodynamic patterns were demonstrated, they were averaged over various periods for presentation.

**Cardiac output, cardiac index (CI)**

A thoracic bioelectric impedance device (Noninvasive Medical Technology) or PhysioFlow (VasoCOMand Manatec Biomedical) was applied shortly after arrival in the ED. The noninvasive, disposable, prewired hydrogel electrodes were positioned on the skin, and electrocardiogram leads were placed on the precordium and shoulders. A 100-kHz, 4-mA alternating current was passed through the patient’s thorax by the outer pairs of electrodes, and the voltage was sensed by the inner pairs of electrodes, which captured the baseline impedance (Zo), and the first derivative of the impedance waveform (dZ/ dt). Previous studies have documented satisfactory correlations between thermodilution and bioimpedance cardiac output values for trauma patients in the ED, OR, and ICU. Correlations between the noninvasive bioimpedance and thermodilution estimates of cardiac output were $r = 0.85$; $r^2 = 0.73$; precision and bias measurements, $-0.124 ± 0.75$ L/min/m$^2$.1,2

**Abbreviations and Acronyms**

- CI = cardiac index
- ED = emergency department
- HR = heart rate
- MAP = mean arterial pressure
- OR = operating room
- PrCO$_2$/FiO$_2$ = transcutaneous oxygen tension indexed to the fractional inspired oxygen concentration
- SapO$_2$ = pulse oximetry
- SP = survival probability

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Figure 1. Hemodynamic patterns and the effects of resuscitation therapy on hemodynamic patterns and survival probability of a 24-year-old man who sustained gunshot wounds to the abdomen and left thigh, with lacerations of the right iliac artery and vein, colon, small bowel, stomach, and spleen and an estimated blood loss of 20,000 mL. During a 5.5-hour operation, this was replaced with rapid transfusions of 46 U of packed red cells, 16 U of fresh frozen plasma (FFP), 4,000 mL starch, and 16,000 mL Ringer’s lactate solution. Upper row: cardiac index (CI); second row: mean arterial pressure (MAP); third row: pulse oximetry (SapO2); fourth row: transcutaneous oxygen tension indexed to the fractional inspired oxygen concentration (PtcO2/FiO2); bottom row: survival probability. Time, in hours from emergency department admission, is noted below the bottom horizontal line. The patient developed ARDS, cardiac failure, and renal failure the next day, and subsequently died.
Pulse oximetry
Routine pulse oximetry (Nellcor) was used to continuously assess arterial hemoglobin oxygen saturation (Sao2). Values were recorded at the time of the cardiac index measurements. Sudden changes in these values were confirmed by in vitro arterial oxygen saturation obtained by conventional blood gas analysis.1,2

Transcutaneous oxygen tension
Conventional transcutaneous oxygen tension measurements were indexed to the fractional inspired oxygen concentration (PaO2/FiO2) continuously monitored throughout the observation period. This technology uses the Clark polarographic oxygen electrode routinely used in standard blood gas measurements.26,31 Oxygen tensions were measured in a representative area of the skin surface heated to 44°C to increase diffusion of oxygen across the stratum corneum and to avoid vasoconstriction in the local area of the skin being measured.28 Transcutaneous oxygen tension was indexed to the FiO2 to give a PtcO2/FiO2 ratio, because changes of the inspired oxygen produce marked PtcO2 changes.29 The electrode must be changed to a nearby site and be recalibrated at 4-hour intervals to avoid skin erythema.

Patient database
A database for acute abdominal and chest trauma emergencies was developed to organize demographic, clinical, and hemodynamic data, including age; gender; specific diagnostic categories for the primary injury or illness; covariates such as sepsis, diabetes, and earlier cardiac problems; hemodynamic patterns by invasive and noninvasive methods; outcomes, including survival or death; complications; organ failures; hospital days; and ICU days. The database also included the presence of sepsis, APACHE II scores, Glasgow Coma Scores, and Injury Severity Scores.3,20

### Table 1. Clinical Features of the Penetrating and Blunt Trauma Series

<table>
<thead>
<tr>
<th></th>
<th>Penetrating trauma</th>
<th>Blunt trauma</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Survivors (n = 280)</td>
<td>Nonsurvivors (n = 35)</td>
</tr>
<tr>
<td>Age, y</td>
<td>28 ± 10</td>
<td>33 ± 7*</td>
</tr>
<tr>
<td>Gender, n</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>259</td>
<td>33</td>
</tr>
<tr>
<td>Female</td>
<td>21</td>
<td>2</td>
</tr>
<tr>
<td>Bodily injury, n</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chest</td>
<td>187</td>
<td>21</td>
</tr>
<tr>
<td>Abdomen</td>
<td>190</td>
<td>21</td>
</tr>
<tr>
<td>Spinal cord injury</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>Fractures</td>
<td>17</td>
<td>1</td>
</tr>
<tr>
<td>Extremity</td>
<td>53</td>
<td>8</td>
</tr>
<tr>
<td>Estimated blood loss, mL</td>
<td>2,904 ± 2,784</td>
<td>6,750 ± 6,678*</td>
</tr>
<tr>
<td>Injury Severity Score</td>
<td>20 ± 12</td>
<td>31 ± 15*</td>
</tr>
<tr>
<td>Glasgow Coma Score</td>
<td>14 ± 2</td>
<td>10 ± 5*</td>
</tr>
<tr>
<td>APACHE II</td>
<td>21 ± 8</td>
<td>28 ± 14*</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± SD.
*p < 0.01 comparing survivors’ values with their corresponding nonsurvivors’ values.

Mathematical search and display program
Bayard and colleagues4,32 and Shoemaker and associates20,21 developed a mathematical (probability) analysis and control program to determine individual patient’s survival probability (SP), from a database of 1,150 patients with similar clinical-hemodynamic “states,” which are defined in terms of the primary diagnosis, covariates, and hemodynamic variables. “Similar” means a group of patients, referred to as “nearest neighbors,” with the same diagnosis, who share the same or similar covariates and have very close hemodynamic patterns to those of the newly admitted study patient. They are used as surrogates or substitutes in place of the newly admitted study patient.20,21

Both clinical covariates and process noise help to explain the variability of patient responses seen in the database. The covariates help to distinguish gross differences in responses of patients with major differences in their disorders. Process noise may explain small differences between patients with the same covariates but dif-
different responses to the same therapy. It is a measure of unmodeled dynamics, or intraindividual variability, because of other sources of variability in the system.4,32

**Survival probability**

A patient’s SP for a given state was calculated by first extracting from the database 40 or more nearest neighbor states of patients having diagnosis, covariates, and hemodynamic values that are closest to the study patient’s values. Each time a set of hemodynamic measurements was made, the SP was computed as the percentage of these nearest neighbors that survived. Figure 1 represents the data of an illustrative patient who sustained a lethal gunshot to the abdomen. Survival probabilities were continuously calculated and displayed throughout the first 48 hours after ED admission, including the initial resuscitation. The SP serves as an outcomes predictor and a measure of severity of illness that can be used to track changes in the patient’s overall status throughout the hospital course.

**Statistical analyses**

The mean ± SD or SEM of the survivors’ and nonsurvivors’ MAP, cardiac output or index, SapO2, and transcutaneous O2 were calculated for the periods of monitoring. We used the unpaired, two-tailed Student’s t-test to evaluate differences between the survivors’ and nonsurvivors’ values at comparable time periods. For categorical variables, differences in proportions between survivors and nonsurvivors were tested using the chi-square test. GraphPad Prism statistical software was used for computations.

**RESULTS**

Continuous noninvasive hemodynamic patterns from the time of ED admission

Table 2 summarizes the mean hemodynamic values ± SD for all trauma survivors compared with all nonsurvivors. Hemodynamic values included CI, HR, MAP, SapO2, PtcO2/FiO2, and the calculated SP. The CI, MAP, SapO2, PtcO2/FiO2, and SP values of the survivors were significantly higher than the corresponding values of those who died, although the HR and PtcCO2 were higher in the nonsurvivors during the first 24 hours after ED admission. Figure 2 illustrates the time course of survivors’ and nonsurvivors’ hemodynamic patterns of the trauma series averaged over intervals during the first 48-hour period.

**Table 2. Hemodynamic and Survival Probability Values in First 24 Hours (n = 657)**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Optimal value</th>
<th>Survivors (n = 570)</th>
<th>Nonsurvivors (n = 87)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>CI, L/min/m²</td>
<td>4.0</td>
<td>3.96 ± 0.96</td>
<td>3.60 ± 0.96</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>MAP, mmHg</td>
<td>85</td>
<td>89 ± 13</td>
<td>76 ± 19</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>HR, beat/min</td>
<td>&lt; 100</td>
<td>105 ± 19</td>
<td>114 ± 18</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>SapO2, %</td>
<td>&gt; 98</td>
<td>99 ± 2</td>
<td>95 ± 7</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>PtcCO2, torr</td>
<td>&lt; 50</td>
<td>47 ± 15</td>
<td>61 ± 55</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>PtcO2/FiO2</td>
<td>&gt; 200</td>
<td>223 ± 109</td>
<td>100 ± 70</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>SP, %</td>
<td>&gt; 80</td>
<td>86 ± 13</td>
<td>69 ± 15</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Hematocrit, %</td>
<td>&gt; 33</td>
<td>34 ± 5</td>
<td>30 ± 7</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>DO2, mL/min/m²</td>
<td>&gt; 600</td>
<td>610 ± 172</td>
<td>513 ± 171</td>
<td>&lt; 0.01</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± SD.

CI, cardiac index; DO2, oxygen delivery; Het, hematocrit; HR, heart rate; MAP, mean arterial pressure; PtcCO2, transcutaneous CO2 tension; PtcO2/FiO2, transcutaneous O2 tension indexed to FiO2; SapO2, arterial hemoglobin saturation by pulse oximetry; SP, survival probability.

**Hemodynamic patterns in blunt and penetrating injuries**

Table 3 lists the mean hemodynamic values ± SEM for surviving and nonsurviving patients with blunt and with penetrating trauma. There were similar patterns in these blunt and penetrating injuries; the CI, MAP, SapO2, PtcO2/FiO2, and SP values of the survivors were significantly higher than the corresponding values of those who died, although HR was higher in the nonsurvivors during the first 24 hours after their ED admission. These patterns occurred more rapidly in penetrating injuries than in blunt trauma.

**Survival probability calculation compared with actual hospital outcomes**

SP values after the initial resuscitation in the prospectively monitored trauma patients were compared with the actual hospital outcomes at the time of their discharge from the hospital a week or more later (Tables 2 and 3). For comparison of the initial SP values with their final hospital outcomes, we used half the difference between survivors and nonsurvivors as the SP cut-point. The mean SP ± SD of survivors during the first 24 hours was 86 ± 13%, and for nonsurvivors, it was 69 ± 15%. There were 12.1% misclassifications for penetrating and blunt trauma emergencies (Table 4).

**Receiver operating characteristic curves**

Figure 3 shows receiver operating characteristic curves for data collected during the first 4-hour period after ED admission. The areas under the receiver operating characteristic curves were: SP, 0.87; PtcO2/FiO2, 0.81;
Figure 2. Survivors’ (n = 571, solid line) and nonsurvivors’ (n = 86, dashed line) temporal patterns are shown for the first 48 hours of the entire series beginning with their emergency department admission. Mean values ± SEM are shown for cardiac index (Cl), heart rate (HR), mean arterial pressure (MAP), pulse oximetry (SapO2), transcutaneous carbon dioxide tension (PtcCO2), transcutaneous oxygen tension indexed to the fractional inspired oxygen concentration (PtcO2/FiO2), and survival probability (SP). All values are keyed to the time of admission to the emergency department. Note the survivors’ cardiac index, MAP, SapO2, PtcO2/FiO2, and SP values were generally higher than those of the nonsurvivors. The mean survivors’ SP values were significantly higher than the mean nonsurvivors’ SP values.
SapO₂, 0.64; MAP, 0.63; and CI, 0.62. But the receiver operating characteristic curves may vary with different times after admission, with different etiologic events and, possibly, with differing degrees of injury.

Comparison of various predictors by misclassification rates

Misclassifications were 12.1% overall. Table 5 shows misclassification rates of single initial or lowest values of MAP, CI, APACHE II score, and the present SP analysis. APACHE II values were calculated on a daily basis; discriminant analysis produced one value that covered the entire observation period. The SP was calculated prospectively online in real time with each set of observed hemodynamic values throughout the observation period. Single values of commonly used shock-related parameters were less related to outcomes compared with SP, a computerized composite of multiple variables.

DISCUSSION

The proposed mathematical representation of circulatory status defines the patient’s clinical-circulatory state by specific diagnostic categories; clinical covariates; and the patterns of hemodynamic variables, their first and second derivatives, and their integrals. Simply stated, the program picks out the patterns of patients in the database who have the closest clinical and hemodynamic patterns to the newly admitted study patient. These are statistically referred to as “nearest neighbors” and are used as surrogates for the study patient. The accuracy and reliability of this approach depends on the size and comparability of the database needed to provide an adequate number of nearest neighbors. This database contains more than 1,100 high-risk patients with more than 40,000 time lines, each of which represents a patient’s clinical-hemodynamic state that potentially may be used as a nearest neighbor. The average difference between patients’ variables and their nearest neighbors’ was less than 0.3 standard deviation for the database variables. Preliminary evaluation suggests the need for a database of at least 200 patients with about 10,000 time lines for each etiologic category to be evaluated.

These methods were tested in a public hospital ED, where the survival probability was found to track clinical changes throughout the initial observation period. During and just after the initial resuscitation, the program correctly predicted subsequent hospital outcomes in 88% of the series. The program was developed to aid less
experienced clinicians who may not have been widely exposed to trauma patients.

Early diagnosis, prognosis, and assessment of physiologic alterations are essential because this allows therapy to be initiated sooner in the hope that earlier therapy may improve outcomes in emergency conditions when time is crucial.6-12 The probability analysis and decision support program are based on noninvasive hemodynamic monitoring, which is safe, inexpensive, available throughout the hospital and prehospital areas, and is comparable in accuracy to invasive monitoring.1-4 They provide independent mathematical tools to evaluate therapeutic responses objectively. If confirmed by further studies, these information systems may provide objective methods to manage critically ill emergency patients.

**Author Contributions**

Study conception and design: Lu, Shoemaker, Demetriades
Acquisition of data: Lu, Chien
Analysis and interpretation of data: Lu, Wo
Drafting of manuscript: Shoemaker, Wo

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**Table 5. Misclassifications in Outcomes Prediction by Various Analyses**

<table>
<thead>
<tr>
<th>Method</th>
<th>Criteria</th>
<th>Misclassification rate</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>n</td>
</tr>
<tr>
<td>Initial heart rate</td>
<td>S &lt; 95, NS &gt; 96 beat/min</td>
<td>70</td>
</tr>
<tr>
<td>Initial MAP</td>
<td>S &gt; 85, NS &lt; 70 mmHg</td>
<td>76</td>
</tr>
<tr>
<td>Lowest MAP</td>
<td>S &gt; 50, NS &lt; 50 mmHg</td>
<td>83</td>
</tr>
<tr>
<td>Initial cardiac index</td>
<td>S &gt; 3.8, NS &lt; 3.8 L/min/m²</td>
<td>72</td>
</tr>
<tr>
<td>APACHE II</td>
<td>S &lt; 27, NS &gt; 27</td>
<td>30</td>
</tr>
<tr>
<td>Discriminate analysis</td>
<td>Multiple</td>
<td>23</td>
</tr>
<tr>
<td>Survival probability, this study</td>
<td>S &gt; 75%, NS &lt; 75%</td>
<td>73</td>
</tr>
</tbody>
</table>

MAP: mean arterial pressure; NS, nonsurvivors; S, survivors.
REFERENCES


