A Multidisciplinary Community Hospital Program for Early and Rapid Resuscitation of Shock in Nontrauma Patients*

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**Objective:** To determine the effect of a community hospital-wide program enabling nurses and prehospital personnel to mobilize institutional resources for the treatment of patients with nontraumatic shock.

**Design:** Historically controlled single-center study.

**Setting:** A 180-bed community hospital.

**Patients:** Patients in shock who were candidates for aggressive therapy.

**Interventions:** From January 1998 to May 31, 2000, patients in shock received standard therapy (control group). During the month of June 2000, intensive education of all health-care providers (ie, prehospital personnel, nurses, and physicians) took place. From July 1, 2000, through June 30, 2001, patients in shock (protocol group) were managed with a hospital-wide shock program. The program included early recognition of shock and the initiation of therapy by nonphysicians. Frontline personnel mobilized a shock team, which used goal-directed resuscitation protocols, early intensivist involvement, and rapid transfer to the ICU where protocols specific to shock etiology were implemented.

**Measurements and main results:** Eighty-six and 103 patients, respectively, were enrolled in the control and protocol groups. Baseline characteristics were similar. The protocol group had significant reductions in the median times to interventions, as follows: intensivist arrival, 2:00 h to 50 min (p < 0.002); ICU/operating room admission, 2 h 47 min to 1 h 30 min (p < 0.002); 2 L fluid infused, 3 h 52 min to 1 h 45 min (p < 0.0001); and pulmonary artery catheter placement, 3 h 50 min to 2 h 10 min (p 0.02). Good outcomes (ie, discharged to home or to a rehabilitation center) were more likely in the protocol group than in the control group (p = 0.02). The hospital mortality rate was 40.7% in the control group and 28.2% in the protocol group (p = 0.035).

**Conclusion:** Similar to current practice in patients who have experienced trauma or cardiac arrest, the empowerment of nonphysician providers to mobilize hospital resources for the care of patients with shock is effective. A community hospital program incorporating the education of providers, the activation of a coordinated team response, and early goal-directed therapy expedited appropriate treatment and was temporally associated with improved outcomes. Randomized multicenter trials are needed to further assess the impact of the shock program on outcomes.

**Key words:** critical illness; intensive care; medical emergency team; resuscitation; sepsis; shock

**Abbreviations:** APACHE = acute physiology and chronic health evaluation; APS = acute physiology score; CI = cardiac index; CVP = central venous pressure; ED = emergency department; FIO₂ = fraction of inspired oxygen; HR = heart rate; IV = IV push; IVPB = IV piggy back; LR = lactated Ringer solution; MAP = mean arterial pressure; NS = normal saline solution; PA = pulmonary artery; PCWP = pulmonary capillary wedge pressure; PRBC = packed RBC; SBP = systolic BP; SVO₂ = mixed venous saturation; SVT = supraventricular tachycardia; UO = urine output; VT = tidal volume

In the community hospital setting, 24-h in-house physician coverage is generally available only through the emergency department (ED). Significant alterations in vital signs or neurologic status in the field or hospital necessitate a call from the nurse to the ED and a response from the ED, primary physician, or consultant, delaying further assessment and treatment. A community-based program that educates all health-care providers to rapidly identify and initiate treatment in life-threatening conditions,

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coupled with treatment protocols based on best-practice guidelines, may improve outcomes. This approach has been exemplified by cardiac arrest and trauma teams in which nonphysician personnel mobilize institutional resources and initiate frontline therapy. A significant group of at-risk patients who may benefit from a similar approach are patients in shock.

Shock is a syndrome of inadequate tissue perfusion. If it is not recognized and treated during a narrow window of opportunity, critical tissue hypoxia develops, and initiates a cascade of events leading to multiorgan failure and death.1,2 The estimated mortality rate in patients with cardiogenic shock with acute myocardial infarction ranges from 50 to 80%.3 In patients with septic shock, the mortality rate varies from 39 to 60% and has not significantly improved in the past few decades.4 Even with major advances in the therapeutic armamentarium, septic shock alone has been estimated to claim at least 90,000 lives per year in the United States.5,6 Despite the high incidence and mortality rate of shock, a comprehensive systems-based approach to rapidly identify and treat shock has been slow to evolve.

A team approach to the resuscitation of patients with shock was first described in 1967.7 This concept reemerged as the medical emergency team, a group of physicians and nurses that can be activated by frontline nonphysician providers to immediately evaluate and treat patients with significant alterations in vital signs or neurologic deterioration.8,9 This approach led to decreases in the incidence of in-hospital cardiac arrest, bed occupancy of cardiac arrest survivors, and the overall in-hospital mortality rate.10,11 It is possible that a similar approach may prove especially beneficial to the subset of patients with shock whether in the hospital or in the field. In addition, early goal-directed hemodynamic therapy has been shown to reduce mortality in patients undergoing high-risk surgery, and those who have experienced trauma, severe sepsis, and septic shock.1,12–15 However, studies of goal-directed therapy16,17 that were initiated later in the course of critical illness have yielded disappointing results. These observations suggest that the resuscitation of shock patients is more likely to improve survival if it is instituted early in the disease process. However, the appropriate resuscitation of shock patients is often hampered by a lack of recognition of early shock, and inadequate knowledge, experience, and skills of health-care providers, which results in avoidable delays in appropriate treatment and patient transfer to the ICU.1,18–22 The above factors could potentially be remedied with a systems-based team approach incorporating staff education to enhance early recognition, empowerment of nonphysicians to mobilize hospital resources, rapid protocol-directed therapy, early intensivist involvement, and a dedicated shock bed to allow prompt transfer to the ICU.1,23–33 Our hypothesis was that this multidisciplinary, point-of-care-driven approach to the treatment of shock would reduce the time to treatment and mortality in our hospital. We developed such a program and evaluated its effect.

**Materials and Methods**

**Study Design and Approval**

We conducted a single-center pilot study in a 180-bed community hospital with 44 critical care beds (open ICU) using a prospective two-group comparison of all patients who were identified as being in shock (prehospital, ED, or inpatient) before and after implementation of a comprehensive program of early recognition and rapid treatment in a nonteaching hospital. After-hours in-house physician coverage was provided solely by the ED. The institutional review board for human research approved the study. Informed consent was waived.

**Development and Education**

In 1997, the screening criteria for the early recognition of shock and for inclusion in the study were developed. From 1997 to June 2000, a design team, including intensivists, emergency physicians, nurses and other health-care providers created the Shock Program. This included the development of an educational program, procedures for the activation of shock alerts, and initial resuscitation protocols. Frontline personnel were to mobilize the shock team, laboratory panel, equipment cart, ICU shock bed, and protocols based on type of shock. Goal-directed treatment protocols were formulated based on the “VIPPS” approach to shock, including early support of ventilation and oxygenation, rapid infusion of volume, addressing pump and pressor requirements, appropriate pharmacologic therapy, and disease-specific interventions (eg, surgery).34,35 Early respiratory support, initial aggressive fluid resuscitation, and goal-directed hemodynamic management were emphasized.1,36–38 Treatment algorithms were devised for shock resuscitation inprehospital, ED, general ward, and ICU settings. Additional protocols were developed for septic,
hypovolemic, cardiogenic, and anaphylactic shock. In June 2000, > 500 health-care providers (prehospital personnel, nurses, and physicians) received intensive education using a standardized teaching package, which included a 1-h slide presentation and introduction to the shock manual. Subsequent interactive classes covered the early recognition, pathophysiology, and management of shock, and the appropriate application of screening criteria and treatment algorithms. The critical importance of the early mobilization of the shock team without unnecessary delay was emphasized to frontline providers, and educational posters were placed in all units. Mock shock alerts were held to practice the shock protocols before the implementation of the program. Ongoing education was provided throughout the protocol phase based on a need assessment after a review of each shock alert.

**Patient Selection**

Screening criteria designed to have high sensitivity for the identification of shock were used to find patients and were posted in all critical care units at the beginning of the control period (Table 1). Confirmation criteria assured that the controlled patients had a significant degree of shock. Exclusion criteria eliminated patients who were not candidates for aggressive therapy, patients without survivable conditions based on illness prior to the onset of shock, or who should have been treated by preexisting systems (eg, trauma and acute myocardial infarction). Patients were enrolled in the control group from January 1998 through May 2000, and in the protocol group from July 2000 until June 2001. The majority of patients who were enrolled in both groups were identified prospectively. Patients missed by prospective identification were subsequently enrolled retrospectively. In June 2000, patients were not enrolled while education was undertaken before implementation of the Shock Program. Critical care nurses, intensivists, and case managers identified patients prospectively using screening criteria during the entire study period. During the protocol period, hospital floor nurses, ED personnel, and emergency medical system personnel also identified patients who were in shock prospectively. To avoid missing patients, relevant diagnosis-related groups (Table 1) and all hospital deaths in both intervals were reviewed retrospectively to identify patients in shock who had not been found by prospective screening. The critical care research coordinator then reviewed all screened patients for inclusion in the study (Fig 1). Patients were classified as having different types of shock on review by the research coordinator and a critical care physician. In the protocol phase, all identified shock patients were included if they satisfied screening, confirmatory, and exclusion criteria, irrespective of whether they had been treated as a result of a shock alert or had received the full protocol treatment (Table 1).

<table>
<thead>
<tr>
<th>Variables</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Screening criteria</td>
<td>Hypotension SBP &lt; 90 bpm, MAP &lt; 60 mm Hg with one or more of the following or normotension with three or more of the following</td>
</tr>
<tr>
<td></td>
<td>Temperature &lt; 36 °C</td>
</tr>
<tr>
<td></td>
<td>Respirations ≥ 20 bpm</td>
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<tr>
<td></td>
<td>Altered mental status (anxiety, apathy, agitation, lethargy, stupor, or coma)</td>
</tr>
<tr>
<td></td>
<td>Cool extremities or skin mottling</td>
</tr>
<tr>
<td></td>
<td>Oliguria &lt; 30 mL/h</td>
</tr>
<tr>
<td></td>
<td>Lactic acid &gt; 2.0 mmol/L or base excess &lt; −5 mmol/L</td>
</tr>
<tr>
<td>Patients had a shock alert called if after a fluid bolus of 250 mL for hospitalized patients or 1,000 mL for field or ED patients, the screening criteria persisted.</td>
<td></td>
</tr>
<tr>
<td>Confirmation criteria (to assure presence of significant shock)</td>
<td>Required one of the following:</td>
</tr>
<tr>
<td></td>
<td>Administration of ≥ 4 L fluid in first 24 h</td>
</tr>
<tr>
<td></td>
<td>Use of vasoconstrictors</td>
</tr>
<tr>
<td></td>
<td>Lactic acid &gt; 2 mmol/L</td>
</tr>
<tr>
<td></td>
<td>Death as a result of hemodynamic instability</td>
</tr>
<tr>
<td>Exclusion criteria</td>
<td>Trauma as cause of shock (call trauma alert)</td>
</tr>
<tr>
<td></td>
<td>Acute myocardial infarction as cause of shock (call cardiologist)</td>
</tr>
<tr>
<td></td>
<td>Patients already receiving mechanical ventilation or pressors in a critical care area</td>
</tr>
<tr>
<td></td>
<td>Patients not candidates for aggressive treatment by advanced directive, or presenting with a nontreatable condition that preceded the shock state (ie, brain death, nontreatable metastatic carcinoma, or ruptured thoracic aneurysm)</td>
</tr>
<tr>
<td>DRG and ICD-9 codes for retrospective review</td>
<td>Septicemia NOS</td>
</tr>
<tr>
<td>038.9</td>
<td>Shock NOS</td>
</tr>
<tr>
<td>785.50</td>
<td>Shock w/o trauma</td>
</tr>
<tr>
<td>785.59</td>
<td>Hypovolemic shock</td>
</tr>
<tr>
<td>995.0</td>
<td>Anaphylactic shock</td>
</tr>
<tr>
<td>458.9</td>
<td>Hypotension NOS</td>
</tr>
<tr>
<td>785.51</td>
<td>Cardiogenic shock</td>
</tr>
<tr>
<td>785.52</td>
<td>Septic shock</td>
</tr>
<tr>
<td>998.0</td>
<td>Postoperative shock</td>
</tr>
<tr>
<td>958.4</td>
<td>Traumatic shock</td>
</tr>
</tbody>
</table>

*Sbpm = breaths/minute; NOS = nitric oxide synthase; ICD-9 = International Classification of Diseases with revision; DRG = diagnosis-related group.*
Interventions

During the protocol phase, when nurses or other health-care providers identified a patient who met the screening criteria for shock that persisted despite a fluid bolus, the shock team was activated by calling a shock alert (Table 1). Prehospital personnel initiated the field protocol and notified the ED of a shock alert. Within the hospital, any health-care provider who identified a patient with shock activated the shock team by radio and overhead page, and the primary care physician was notified simultaneously. There were 10 board-certified intensivists who had received Shock Program education, rotating on call for shock at all times. To expedite treatment in the most appropriate setting, an ICU bed was kept available at all times for shock patients. Shock carts containing equipment and fluids were placed in the ED and the ICU, and were transported to the shock patient’s bedside for use by the shock team. This team, which involved an intensivist, an emergency physician, an ICU nurse, and a respiratory therapist, and electrocardiography, clinical laboratory, radiology, pastoral care, and social services (for family support), responded to the patient in the hospital or the ED. On arrival, primary care was transferred to the intensivist. A general resuscitation protocol (Fig 2) was initially followed, and a specific treatment protocol was applied once the type of shock was identified as hypovolemic, septic, cardiogenic, or anaphylactic (see the “Appendix”). Specific treatment goals included decreased work of breathing, arterial oxygen saturation of >92%, mean arterial pressure ≥ 70 mm Hg, urine output (UO) of ≥ 30 mL/h, mixed venous oxygen saturation (SvO₂) of ≥ 60%, and cardiac index (CI) of ≥ 2.5 L/min/m² for cardiogenic shock, ≥ 2.7 L/min/m² for hypovolemic shock, and ≥ 3.0 L/min/m² for septic or anaphylactic shock (Fig 2). During the course of the study, the therapies that were available for the treatment of shock did not change.

Measurements

BP, heart rate (HR), temperature, and respiratory rate on presentation were collected in both groups. In the control group, the available laboratory data obtained by the attending physician were collected. The shock panel obtained in the protocol group included baseline lactic acid level, arterial blood gas levels, CBC count, electrolyte levels, hepatic and renal function test results, urinalysis findings, coagulation screen and blood culture results.

The acute physiology and chronic health evaluation (APACHE) III data on day 1 of the patient in the ICU were extracted for both groups (Table 2). For both groups, time zero was designated as the earliest point in the illness when screening criteria were met on retrospective review. Intervals from time zero to shock alert activation, intensivist arrival, ICU admission, administration of 2 L fluid, vasopressor therapy, antibiotic administration, central venous catheterization, tracheal intubation, and pulmonary artery (PA) catheterization were recorded. Data on mortality, hospital and ICU lengths of stay, and discharge location were collected.

Statistical Analysis

Hospital mortality was the primary end point. Secondary end points were the identification of shock patients, times to interventions, length of stay, and discharge location. Based on a control group mortality rate of 40%, we performed power calculations that determined that 100 patients should be acquired in each group to demonstrate a relative mortality reduction of 30%, using an α of 0.05 with a power of 0.8. Our hypothesis was that earlier recognition of shock and a systems-based approach to the treatment of shock patients incorporating best practice at our institution would reduce mortality. We emphasize that we were interested in a clinically significant mortality reduction that would change the practice. Increased mortality would not modify the practice at our institution; because of this, we used a one-sided z-test. Baseline characteristics and other nonmortality comparisons were performed as two-sided tests, as there is no reason to believe that one group would have better, rather than different, outcomes for these variables.

We were unable to control the baseline characteristics of our subjects; therefore, we used these confounding variables in a logistic regression model for mortality to ensure that the difference between the groups’ mortality rates was not due to some other underlying difference in the groups. Of particular concern to us was the "severity of illness," as measured by the acute physiology score (APS) and APACHE III score. In addition to the logistic regression model, the two-population z-test and t test, the Satterthwaite t test, and the Fisher exact test were used to determine statistically significant relationships. In order to use parametric statistical methods, transformations to the data were
Figure 2. Interventions. SBP = systolic BP; bpm = breaths per minute; MI = myocardial infarction; SaO₂ = arterial oxygen saturation; ER = ED; OR = operating room; ACLS = advanced cardiac life support; BE = base excess.
### Table 2—Baseline Patient Characteristics

<table>
<thead>
<tr>
<th>Variables</th>
<th>Control Group (n = 86)</th>
<th>Protocol Group (n = 103)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shock patients/ICU admissions</td>
<td>86/20,976</td>
<td>103/9,120</td>
<td>&lt;0.01†</td>
</tr>
<tr>
<td>Female patients</td>
<td>48.8%</td>
<td>35.9%</td>
<td>0.08†</td>
</tr>
<tr>
<td>Male patients</td>
<td>51.2%</td>
<td>64.1%</td>
<td>0.08†</td>
</tr>
<tr>
<td>Age, yr</td>
<td>64.3 ± 17.7</td>
<td>64.6 ± 15.5</td>
<td>0.89†</td>
</tr>
<tr>
<td><strong>Baseline physiologic values</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>115 ± 22.6</td>
<td>115 ± 29.0</td>
<td>0.96††</td>
</tr>
<tr>
<td>Systolic BP, mm Hg</td>
<td>72.7 ± 17.2</td>
<td>75.3 ± 21.3</td>
<td>0.43‡‡</td>
</tr>
<tr>
<td>Mean arterial pressure, mm Hg</td>
<td>52.2 ± 10.8</td>
<td>52.6 ± 17.2</td>
<td>0.71‡‡</td>
</tr>
<tr>
<td>Respiratory rate, bpm</td>
<td>20.5 ± 11.0</td>
<td>20.2 ± 9.3</td>
<td>0.90‡‡</td>
</tr>
<tr>
<td>Temperature, °C</td>
<td>36.2 ± 1.4</td>
<td>36.4 ± 1.8</td>
<td>0.33</td>
</tr>
<tr>
<td>Serum lactate, mmol/L</td>
<td>5.9 ± 5.1**</td>
<td>4.2 ± 4.1††</td>
<td>0.05‡‡</td>
</tr>
<tr>
<td>Base excess, mmol/L</td>
<td>- 6.9</td>
<td>- 6.5</td>
<td>0.80</td>
</tr>
<tr>
<td>Oliguric (&lt; 500 mL first 24 h in ICU)</td>
<td>16.3%</td>
<td>13.6%</td>
<td>0.55†</td>
</tr>
<tr>
<td>Hematocrit</td>
<td>28.7 ± 7.6</td>
<td>29.2 ± 7.4</td>
<td>0.69†</td>
</tr>
<tr>
<td>Platelet count, × 10^3/mL</td>
<td>236 ± 151</td>
<td>223 ± 156</td>
<td>0.27‡‡</td>
</tr>
<tr>
<td>International normalized ratio</td>
<td>1.7 ± 1.1</td>
<td>1.6 ± 0.8</td>
<td>0.81‡‡</td>
</tr>
<tr>
<td>Creatinine, mg/dL</td>
<td>2.1 ± 2.4</td>
<td>1.9 ± 1.8</td>
<td>0.94‡‡</td>
</tr>
<tr>
<td>Initial pO2/FiO2 ratio</td>
<td>229 ± 138</td>
<td>227 ± 139</td>
<td>0.94‡‡</td>
</tr>
<tr>
<td>Total bilirubin, mg/dL</td>
<td>2.1 ± 3.4</td>
<td>1.7 ± 1.1</td>
<td>0.71‡‡</td>
</tr>
<tr>
<td>Glasgow coma score</td>
<td>Median 12.5 ± 3.5</td>
<td>12.1 ± 4.0</td>
<td>0.53 ± 1.0</td>
</tr>
<tr>
<td>Mean 14</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Preexisting conditions</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>COPD</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severe</td>
<td>4.7 (4)</td>
<td>5.8 (6)</td>
<td>0.76††</td>
</tr>
<tr>
<td>Moderate</td>
<td>11.6 (10)</td>
<td>8.7 (9)</td>
<td>0.51††</td>
</tr>
<tr>
<td>AIDS</td>
<td>0.0 (0)</td>
<td>0.0 (0)</td>
<td></td>
</tr>
<tr>
<td>Leukemia/melanoma</td>
<td>0.0 (0)</td>
<td>1.0 (1)</td>
<td>1.01†</td>
</tr>
<tr>
<td>Tumor/mets/lymphoma</td>
<td>3.5 (3)</td>
<td>4.9 (5)</td>
<td>1.00†</td>
</tr>
<tr>
<td>Immunosuppressed</td>
<td>0.0 (0)</td>
<td>8.7 (9)</td>
<td>&lt;0.01††</td>
</tr>
<tr>
<td>Cirrhosis</td>
<td>8.1 (7)</td>
<td>2.9 (3)</td>
<td>0.19†</td>
</tr>
<tr>
<td>Dialysis</td>
<td>2.3 (2)</td>
<td>3.9 (4)</td>
<td>0.69†</td>
</tr>
<tr>
<td>Diabetes</td>
<td>19.8 (17)</td>
<td>19.4 (20)</td>
<td>0.95†</td>
</tr>
<tr>
<td>No medical history</td>
<td>47.7 (41)</td>
<td>52.4 (54)</td>
<td>0.52†</td>
</tr>
<tr>
<td><strong>ICU admission source</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Direct admit (Physician Office)</td>
<td>2.3 (2)</td>
<td>2.9 (3)</td>
<td>1.00†</td>
</tr>
<tr>
<td>ED</td>
<td>69.8 (60)</td>
<td>62.1 (64)</td>
<td>0.27†</td>
</tr>
<tr>
<td>Floor</td>
<td>17.4 (15)</td>
<td>20.4 (21)</td>
<td>0.61†</td>
</tr>
<tr>
<td>Stepdown unit</td>
<td>4.7 (4)</td>
<td>5.8 (6)</td>
<td>0.76†</td>
</tr>
<tr>
<td>Operating room/recovery</td>
<td>3.5 (3)</td>
<td>6.8 (7)</td>
<td>0.35†</td>
</tr>
<tr>
<td>ICU</td>
<td>1.5 (1)</td>
<td>0.0 (0)</td>
<td>0.46†</td>
</tr>
<tr>
<td>Other hospital</td>
<td>1.5 (1)</td>
<td>1.9 (2)</td>
<td>1.00†</td>
</tr>
<tr>
<td><strong>Shock type</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Septic</td>
<td>41.9 (36)</td>
<td>47.5 (49)</td>
<td>0.43†</td>
</tr>
<tr>
<td>Hypovolemic</td>
<td>38.4 (33)</td>
<td>41.7 (43)</td>
<td>0.64†</td>
</tr>
<tr>
<td>Cardiogenic/obstructive</td>
<td>12.8 (11)</td>
<td>9.7 (10)</td>
<td>0.50†</td>
</tr>
<tr>
<td>Anaphylactic</td>
<td>4.7 (4)</td>
<td>0.0 (0)</td>
<td>0.04†</td>
</tr>
<tr>
<td>Spinal</td>
<td>0.0 (0)</td>
<td>0.0 (0)</td>
<td></td>
</tr>
<tr>
<td>Unclassified</td>
<td>2.3 (2)</td>
<td>1.0 (1)</td>
<td>0.59†</td>
</tr>
<tr>
<td><strong>APS</strong></td>
<td>60.5 ± 26.1</td>
<td>61.8 ± 32.2</td>
<td>0.77††</td>
</tr>
<tr>
<td>APACHE III score</td>
<td>74.0 ± 28.1</td>
<td>75.7 ± 33.0</td>
<td>0.71††</td>
</tr>
</tbody>
</table>

*Values given as mean ± SD or No. (%), unless otherwise indicated.
†Fisher exact test.
‡Χ² test.
§Square of age transformation.
¶Satterthwaite t test.
**Two-sided two-population t test.
††Logarithm transformation.
**A total of 50% of patients in shock had lactic acid levels measured.
††A total of 95% of patients in shock had lactic acid levels measured.
‖Square root transformation.
‖‖International normalized ratio squared transformation.
||Median two-sample test.
sometimes necessary. In those circumstances, the form of the transformation is given, along with the results (Table 3 and Appendix).

**RESULTS**

**Implementation**

After 2.5 years of planning and 1 month of intensive education, we launched the Shock Program (described in the “Materials and Methods” section) and continued the education of providers in the early recognition and treatment of shock. Prehospital personnel, nurses, and physicians were empowered to mobilize a Shock Team and cart, initiate rapid goal-directed therapy, and the rapid transfer of the patient to a shock bed in the ICU, which was available at all times. This program has been adopted by the medical staff and administration as the standard of practice at our institution and has been implemented at another community hospital.42 We recognized multiple barriers to implementing this program, including acceptance by the medical staff, competing critical care groups, multiple departments, (eg, the ED and ICU), the nursing staff, and ancillary services. Nursing required a substantial change in culture to initiate frontline therapy before there was 100% support by the medical staff. In addition, fortitude was required from the administration, given the conflicting reactions from different parts of the medical staff. We were able to demonstrate that it was possible to overcome these barriers in a relatively short period with limited resources.

**Sensitivity and Specificity of Criteria**

Screening criteria by definition were 100% sensitive to identifying patients who were in shock; the criteria had a specificity of 83%. Seventeen percent of patients for whom shock alerts were called had screening criteria reversed quickly with prompt medical interventions such as respiratory treatment, antiarrhythmic drug therapy, or fluid challenge, and they were not screened further (n = 18). Nine of these patients had acute respiratory insufficiency, and the remainder had dysrhythmia or mild hypovolemia.

We enrolled 86 patients in the control group and 103 patients in the protocol group (Fig 1). There were no significant differences in baseline characteristics, source of referral, and types of shock across treatment groups except that there were more immunosuppressed patients in the protocol group (Table 2). After the protocol interval began, the control group was reanalyzed, which identified patients who should not have been enrolled; as a result, our control group was reduced to 86 patients.

**Admission Source**

Approximately 66% of our shock patients were admitted to the ICU from the ED, 24% were admitted from general wards, and the remaining 10% of patients were admitted from diverse locations (Table 2). There was no difference in the ICU admission source between the control and protocol groups. In the protocol group, emergency medical technicians in the field identified shock, called shock alerts, and initiated therapy independently. In the ED, nurses frequently identified patients who were in shock and generally activated shock alerts in consultation with the ED physician. Most of the in-hospital shock alerts outside of the ED were activated by nonphysicians.

**Table 3—Results**

<table>
<thead>
<tr>
<th></th>
<th>Control Group</th>
<th>Protocol Group</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Survivors</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>APS</td>
<td>53.4 ± 24.1</td>
<td>50.9 ± 25.6</td>
<td>0.58†</td>
</tr>
<tr>
<td>APACHE III score</td>
<td>64.7 ± 26.0</td>
<td>63.9 ± 25.5</td>
<td>0.87†</td>
</tr>
<tr>
<td>ICU length of stay, d</td>
<td>8.9</td>
<td>6.8</td>
<td>0.18‡</td>
</tr>
<tr>
<td>Hospital length of stay, d</td>
<td>14</td>
<td>15.6</td>
<td>0.45‡</td>
</tr>
<tr>
<td><strong>Nonsurvivors</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>APS</td>
<td>71.2</td>
<td>90.6</td>
<td>0.008†</td>
</tr>
<tr>
<td>APACHE III score</td>
<td>88</td>
<td>106.9</td>
<td>0.01†</td>
</tr>
<tr>
<td><strong>Discharge location of survivors</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Home</td>
<td>68.6% (35)</td>
<td>73% (54)</td>
<td>0.60†</td>
</tr>
<tr>
<td>Rehabilitation</td>
<td>9.8% (5)</td>
<td>14.9% (11)</td>
<td>0.40†</td>
</tr>
<tr>
<td>Skilled nursing facility</td>
<td>17.6% (9)</td>
<td>6.8% (5)</td>
<td>0.06†</td>
</tr>
<tr>
<td>Other</td>
<td>3.9% (2)</td>
<td>5.4% (4)</td>
<td>1.00†</td>
</tr>
<tr>
<td>Combined home/rehabilitation</td>
<td>78.4% (40)</td>
<td>87.8% (65)</td>
<td>0.16†</td>
</tr>
</tbody>
</table>

*Values given as mean ± SD or % (No.), unless otherwise indicated.†Two-sided, two-population t test.‡Logarithm transformation.$χ^2$ test.¶Fisher exact test.
**Time to Treatment**

Time-to-treatment intervals, referenced from time zero (ie, the earliest point at which the screening criteria were met) are shown in Figure 3. In the protocol group, the mean time interval to the activation of a shock alert was 49 min, and the median time interval was 15 min. This interval included time to obtain IV access, administer fluid, and decide that the criteria had been met to activate a shock alert. Compared to the control group, there was a significant reduction in the mean times to the infusion of 2 L fluid (p < 0.0001), intensivist arrival (p < 0.001), ICU admission (p < 0.01), and PA catheterization (p < 0.03) in the protocol group. Intensivist involvement was more common in the protocol group (99% vs 87%, respectively; p < 0.002). Five control patients (5.8%) were not attended by an intensivist prior to their death vs one patient (1.0%) in the protocol group (p = 0.16). In addition, three control patients (3.5%) died before ICU arrival compared to one (1.0%) in the protocol group (p = 0.52). There were trends toward earlier antibiotic therapy (p = 0.16). There were no significant differences in the time to central line placement (p = 0.4), tracheal intubation (p = 0.94), and pressor therapy (p = 0.73) [Fig 3].

**Outcome**

The overall mortality rate in the protocol group (28.2%) was significantly lower than that in the control group (40.7%), with an absolute mortality rate reduction of 12.5% and a relative mortality rate reduction of 31% (p = 0.035) [Fig 4]. A logistic regression model, with mortality as the outcome, and including the confounders APACHE III score and APS as explanatory variables, showed the odds ratio of mortality was 2.4 (95% confidence interval, 1.2 to 5.1) for the control group compared with the protocol group. Nine patients need to be treated to save one additional life. The shock etiology and ICU admission sources were not significant confounders in this model. In patients who died, the APACHE III score (< 0.01) and APS score (p = 0.008) were significantly lower in the control group (Table 3). Therefore, patients who died in the control interval were on the average less ill at the time of presentation to the ICU than those who died in the protocol interval.

In a post hoc analysis, we compared “good outcomes” (defined as patients who had been discharged to home or discharged to rehabilitation) to “poor outcomes” (defined as death, patients discharged to a skilled nursing facility, or other). This comparison showed that the protocol group (65 of

![Figure 3](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/22025/ on 06/27/2017)
103 patients; 63.1%) had a significantly higher likelihood of a good outcome than the control group (40 of 86 patients; 46.5%; p = 0.02). ICU and hospital length of stay were not significantly different across treatment groups (Table 3).

**Discussion**

Shock continues to exact a high mortality despite advances in intensive care. Delay in treatment beyond the critical "golden hour" leads to multiorgan dysfunction, and increased morbidity and mortality. Health-care providers who are first on the scene, whether they are prehospital personnel, nurses, or physicians, vary greatly in their knowledge and skills, and often underestimate the severity of illness until it is too late. Outside the critical care setting, the initial resuscitation may be further impaired by the delayed initiation of therapy by nonphysician personnel, inadequate venous access, inadequate fluid resuscitation, delayed intensivist involvement, and difficulty in obtaining an ICU bed. We created a comprehensive program that addressed these problems by the intensive education of providers to improve the early identification of shock, the mobilization of hospital resources by nonphysician personnel, a rapid multidisciplinary team response, early intensivist involvement, protocol-directed treatment, and expedited patient transfer. Our study showed that this multifaceted systems-based approach to shock management was feasible in the setting of a community hospital. The Shock Program empowered nonphysician providers, identified more patients in shock, reduced the time to treatment, and was temporally associated with improved survival.

Implementation of the Shock Program was followed by an absolute mortality rate reduction of 12.5% and a relative mortality rate reduction of 31% (p = 0.035). As the treatment groups were similar and the spectrum of therapies available for the treatment of shock did not change during the study, it is possible that the reductions in mortality and times to interventions were the result of the Shock Program. A possible mechanism for the reduction in mortality may be the earlier restoration of organ perfusion before the initiation of the inflammatory cascade and coagulation disturbances that lead to multiorgan failure. Although our study was not designed to assess the relationship of the contributions of individual components of the program to outcomes, important interventions that probably contributed include the education and empowerment of nonphysician providers, the prompt transfer of patients to the ICU, and early goal-directed hemodynamic therapy. The education of nonphysician providers has been shown to reduce the duration of receiving mechanical ventilation in patients with acute respiratory failure, and the door-to-treatment time in patients with acute coronary syndromes. After the education of all health-care providers at our hospital, the median interval from time zero to shock alert activation was only 15 min (Fig 3), and the time to the administration of 2 L fluid was reduced by > 50% (p < 0.0001). The critical importance of early fluid resuscitation is supported by studies in children with burns and septic shock, which showed that earlier fluid administration led to reduced organ failure and mortality. The medical emergency team approach has shown that empowering nonphysicians to mobilize hospital resources to evaluate patients with clinical deterioration improves outcomes in teaching hospitals. Our study suggests that the application of this approach to critically ill patients is feasible, even in community hospitals without 24-h in-house intensivists or residents.

Intensivist involvement was more common and intensivist arrival took place earlier in the protocol group. Intensivist supervision has been associated with improved survival in septic shock patients, although there are surprisingly few studies of the impact of intensivists on the outcome of patients who are in shock. The availability of a dedicated ICU bed at all times required a sustained effort to triage patients. The reduced time to ICU admission in the protocol group may have expedited the placement of invasive lines, as shown by the trend toward early central venous catheterization and reduced time to PA catheterization. Even though the utility of the PA catheter has been debated, the earlier use of invasive hemodynamic monitoring in the protocol group may have initiated goal-directed therapy earlier. Prior studies have shown that early hemodynamic optimization using central venous or PA catheters...
improve survival in patients with trauma, those who have undergone high-risk surgery, and those with severe sepsis. Aggressive therapy late in the course of a critical illness has not been shown to improve outcomes and may indeed increase mortality.\textsuperscript{16,17} Unfortunately, the latter scenario often occurs in community or academic hospitals and happened much more often in our hospital before the implementation of the Shock Program.

In the protocol group, there were trends toward lower mortality in the subgroups of patients with cardiogenic, hypovolemic, and septic shock, which did not reach statistical significance (Fig 5). The control group septic shock mortality rate was 50.0\%, which is comparable to current studies.\textsuperscript{14,48} In the protocol septic shock subgroup, the mortality rate was reduced to 32.6\% (p = 0.053). This 17.4\% absolute reduction is similar to the findings of a study\textsuperscript{3} of patients with severe sepsis and septic shock who had been treated with early goal-directed therapy at the ED of an urban academic institution, which decreased the mortality rate by 16\% (from 46.5 to 30.5\%). Since the subgroup with septic shock had the greatest mortality, we would expect these patients to benefit the most from an effective treatment. A similar outcome was observed in a trial\textsuperscript{49} of activated protein C for the treatment of severe sepsis. In our study, the septic subgroup appeared to benefit the most from the Shock Program. Since more protocol group patients were sent home or to rehabilitation, it seems likely that their functional status was the same or better than the control group.

Although the Shock Program shortened most times to treatment, some patients still had delayed identification of shock. The median time-to-treatment intervals were lower than the mean intervals, suggesting that outliers prolonged the mean values. The time interval to antibiotic administration is partially delayed because it was measured from time zero, which was generally earlier than the time at which infection was recognized. There was a trend toward higher lactic acid concentration in the control group. However, lactic acid levels were obtained for only half of the control patients and were usually measured later in the course of illness when they were specifically ordered by the physician. In comparison, 95\% of patients in the protocol group had lactic acid levels measured usually minutes after the shock alert was called. The base excess measured, which was obtained in all patients, was equivalent between the two groups.

Feedback regarding the Shock Program from non-physician providers and the hospital administration has been universally positive. Physician feedback has been generally positive, although a minority of phy-

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure5.png}
\caption{Mortality by shock type. $\beta$ = one-sided $z$ test; $\psi$ = Fisher exact test.}
\end{figure}
Physicians expressed concern over the loss of patient control, this concern has diminished with time. Intensive education and the ability to activate Shock Alerts have empowered nurses and emergency medical personnel to initiate intervention without unnecessary delay. We thought that this change in the prevailing culture at our hospital was a positive outcome in itself.

Since our program is protocol-driven, it can be replicated at institutions without intensivists by utilizing existing personnel (eg, emergency physicians, hospitalists, residents, or physician extenders with training in the fundamentals of critical care support) to form the shock team. This study adds to a growing body of literature advancing the concept of critical care without walls (ie, the extension of critical care expertise beyond the ICU to the ED, general hospital wards, and in the field). Currently, some academic medical centers are studying the use of Condition-C teams in which house officers can rapidly mobilize hospital resources for critically ill patients in general hospital wards. Our program differs from this approach in that it was conducted in a community hospital setting and, more importantly, the shock team was activated prior to physician consultation for patients in shock, many of whom would not be recognized as being in a prearrest state until too late.

It is conceivable that quality of care improved during the study period independent of the influence of the Shock Program, although this is unlikely during this short time span without a substantial change in available treatment options. The study was completed before convincing data regarding the use of hydrocortisone in pressor-dependent sepsis and insulin infusion for tight glucose control in critically ill patients, or the availability of recombinant activated protein C was made available. In the protocol group, 103 patients were enrolled in the study over > 1 year, while the control group included 86 patients who had been enrolled over 2.5 years. The difference was likely due in part to the increased capacity of the hospital to accept shock patients. Just before the implementation of the Shock Program, the hospital expanded from 130 to 180 beds and from 22 to 45 ICU beds. This reduced the number of patients who were diverted to other hospitals. It is possible that a sicker cohort of patients may have presented during either period, and that the improved identification of shock by a larger number of trained personnel with heightened awareness of and ability to apply the screening criteria increased enrollment during the protocol period. These biases could have potentially selected patients who were either less or more ill in the protocol group. The protocol group contained all of the immunosuppressed patients and none of the anaphylactic patients. However, none of the anaphylactic shock patients died, and, since immunosuppressed patients tend to be sicker (three of nine patients died), it is likely to have contributed to the slightly higher severity of illness in the protocol group. In addition, the baseline characteristics at the time of enrollment, including vital signs, renal function, UO in the first 24 h, \( \text{PO}_2/\text{fraction of inspired oxygen (FiO}_2 \) ratio, total bilirubin level, base excess, APACHE III scores, and preexisting illness were similar between the two groups. Furthermore, all patients had to meet confirmatory criteria, which selected the sickest patients in both groups, in order to be included in the study. Therefore, it appears that our methodology did not select for less ill shock patients. A retrospective review of relevant diagnosis-related groups and deaths was performed during both periods to identify patients who had been missed by prospective screening (Table 1).

Our study is limited by the use of an historical control group in that confounding factors unrelated to the Shock Program could have resulted in improved outcomes. However, a randomized, controlled trial comparing the Shock Program with standard care at a single institution is not possible, since the program implementation requires a substantial change in knowledge base, attitudes, and skill set for most health-care providers. In addition, randomizing patients within a single institution to standard-of-care therapies vs therapy per primary physician discretion may be unethical. Ultimately, the best evaluation would be a multicenter trial including hospitals with and without a shock program; however, this single-center pilot study serves as proof of the concept. Seventy-three percent of protocol patients had shock alerts called. The remainder had most of the shock team’s resources present, and protocol treatment was being applied as soon as shock was recognized. Finally, we did not include serial measurements of resuscitation end points. However, a prior study has already demonstrated that early aggressive therapy results in the more rapid resolution of lactic acidosis and organ dysfunction and the earlier achievement of resuscitation goals.

**Conclusion**

To our knowledge, this is the first time that a program incorporating the education of health-care providers in the earlier recognition and treatment of shock, a systems-based team approach, and the early goal-directed therapy of patients with nontraumatic shock has been shown to be possible and beneficial...
in a community hospital setting. This program empowered nurses and prehospital personnel to rapidly mobilize hospital resources, expedited appropriate therapy, and was temporally associated with improved outcomes. Research funding is urgently needed to conduct multicenter trials evaluating the impact of shock programs on resource utilization, survival, and long-term outcome, including subsequent quality of life and cost-utility compared with other health-care interventions.

ACKNOWLEDGMENT: The authors wish to give special thanks to Wayne Park Wagers, MD, for his inspiration and Harry Daniel, MD, Derek C. Angus, MD, Mark Astiz, MD, and Joseph Carello, MD for their review of the manuscript. We also wish to thank Debbie Shoffner and Linda Sebat for their support and assistance throughout the study and in the preparation of the manuscript.

APPENDIX: SHOCK PROTOCOLS

Hypovolemic Shock ED Protocol (June 2000)

The goals of the protocol are as follows: MAP, ≥ 70 mm Hg; UO, > 30 mL/h (excluding patients in oliguric renal failure); warm extremities; capillary refill, ≤ 3 s; central venous pressure (CVP), > 12 mm Hg; CI, ≥ 2.7, or SvO₂, ≥ 60; and avoid significant worsening of oxygenation with volume therapy (ie, a significant decrease in PaO₂/FIO₂ ratio).

The protocol is as follows:

- Systolic BP (SBP) < 90 mm Hg: Administer crystalloid (normal saline solution [NS]/lactated Ringer solution [LR]) or packed RBCs (PRBCs) by rapid infusion for hemoglobin level of < 9 g/dl
- SBP < 90 mm Hg: Continue treatment with NS/LR colloid or PRBCs for hemoglobin level of < 9, as ordered by physician
- SBP < 90 mm Hg: add dopamine to treatment
- Address acute bleeding if indicated by the following: GI consult; surgical consult; rapid transfer to operating room if indicated; and correct coagulopathy per protocol
- SBP < 90 mm Hg: after treatment with 4 L fluid, increase dopamine infusion

Hypovolemic Shock ICU Protocol

The protocol is as follows:

- Intubate if shock continues.
- Consider placement of central line, and placement of PA line if there is a history of heart failure, age ≥ 70 years, respiratory failure, or renal failure.
- Check SvO₂.
- Use as index of resuscitation the following: BP; UO; peripheral perfusion; CVP; and, if a PA line is placed, CI and pulmonary capillary wedge pressure (PCWP).
- Continue aggressive volume therapy, and therapy with crystalloid, colloid, and RBCs, as ordered by physician.
- Transfuse PRBCs to target hemoglobin level, as ordered by physician.
- Continue volume therapy and assess effect on goals; continue volume therapy if goals have been affected in a positive way, without worsening oxygenation (ie, significant decrease in PaO₂/FIO₂ ratio).
- If shock continues after euvolemia has been established, then continue per appropriate protocol (ie, decreased systemic vascular resistance/septic protocol or decreased CI/cardiogenic shock protocol).
- Use appropriate tidal volume (VT) and rate to prevent air-trapping and hypotension on assist control mode (ie, 6 to 10 mL/kg) to keep static inspiratory pressure at ≤ 30 cm H₂O.
- If lungs are deteriorating, or > 50% FIO₂, or PaO₂/FIO₂ ratio of < 200, use appropriate VT to keep static inspiratory pressure at ≤ 30.
- Dopamine to keep MAP at > 70 mm Hg (if euvolemic).
- Dobutamine to keep CI at > 3.0 (if euvolemic).
- For GI prophylaxis, administer famotidine drip, 40 mg per 24 h.
- Sequential compression device.
- Deep vein thrombosis prophylaxis, if no contraindication, with low-molecular-weight heparin; check with physician for dose.
- Placement of duodenal feeding tube per protocol in first 24 h.
- Consider enteral feeding after 24 h of hospital admission.
- If PaO₂/FIO₂ ratio is < 200, place patient on 62° continuous lateral rotational bed.

Septic Shock ED Protocol (June 2000)

The goals of the protocol are as follows: MAP, ≥ 70 mm Hg; UO, ≥ 30 mL/h (excluding patients in oliguric renal failure); warm extremities; capillary refill, ≤ 3 s; CVP, > 12 mm Hg; CI, ≥ 3.0; or SvO₂, ≥ 60%; avoid significant worsening of oxygenation with fluid therapy (ie, significant decrease in PaO₂/FIO₂ ratio).

The protocol is as follows:

- SBP < 90 mm Hg: administer crystalloid by rapid infusion
- SBP < 80 mm Hg: administer norepinephrine
- Continue rapid infusion of NS/LR, colloid, or PRBCs for hemoglobin level of < 9, as ordered by physician
- SBP < 90 mm Hg: increase norepinephrine treatment
- Administer antibiotic per algorithm

ICU Septic/Spinal/Low Systemic Vascular Resistance Shock Protocol

The protocol is as follows:

- Intubate if shock continues.
- Consider placement of PA catheter.
- Arterial line.
- Norepinephrine therapy to keep MAP at ≥ 70 mm Hg.
- When MAP is ≥ 70 mm Hg, add dobutamine, 2 μg/kg/min, to norepinephrine regimen.
- As ordered by physician, use volume therapy with NS/LR, colloid, or PRBCs until PCWP is at > 14 mm Hg or goals are met.
- Wean patient from norepinephrine for MAP of ≥ 80 mm Hg.
- Continue volume infusion to assist with norepinephrine weaning if PCWP is at ≥ 18 mm Hg and lungs are not deteriorating, and/or FIO₂ is ≤ 50%.
- Use appropriate VT and rate to prevent air-trapping on assist control mode (ie, 6 to 10 mL/kg) to keep static inspiratory pressure at ≤ 30.
• If lungs are deteriorating, or \(F_{\text{I}}O_2 > 50\%\), or \(P_{\text{aO}_2}/F_{\text{I}}O_2\) ratio is < 200 (ie, patient has ARDS), use further volume therapy judiciously.
• Dobutamine therapy to keep CI at > 3.0.
• For GI prophylaxis, administer famotidine drip, 40 mg per 24 h.
• Sequential compression device.
• Deep vein thrombosis prophylaxis, if no contraindication, with low-molecular-weight heparin; check with physician for dose.
• Placement of duodenal feeding tube per protocol in first 24 h.
• Consider enteral feeding after 24 h of hospital admission.
• If there is a \(P_{\text{aO}_2}/F_{\text{I}}O_2\) ratio of < 200, place patient on 62° continuous lateral rotational bed.

Antibiotic Guidelines for Septic Shock Based on Likely Source of Infection (June 2000)

Obtain blood and other appropriate cultures prior to antibiotic administration.
1. Community-acquired pneumonia: gatifloxacin, 400 mg IV piggyback (IVPB), plus ceftaxone, 2 g IVPB.
2. Hospital-acquired pneumonia: imipenem/cilastatin, 500 mg IVPB, plus tobramycin, 5 mg/kg IVPB.
3. Abdominal infection, peritonitis, or perirenal abscess: imipenem/cilastatin, 500 mg IVPB, plus gentamicin, 5 mg/kg IVPB; consider liposomal amphotericin, 3 mg/kg infused over 2 h.
4. Urinary tract infection: piperacillin, 2 g IVPB, plus gentamicin, 2 mg/kg IVPB.
5. Vascular: native valve approximately congruent with vancomycin, 15 mg/kg IVPB; prosthetic valve or vascular device approximately congruent with vancomycin, 15 mg/kg IVPB, plus gentamicin, 1 mg/kg IVPB, plus rifampin, 300 mg po; consider liposomal amphotericin therapy, 3 mg/kg infused over 2 h.
6. Meningitis: vancomycin, 15 mg/kg IVPB, plus ceftaxone, 2 g IVPB.
7. Skin cellulitis or abscess: oxacillin, 2 g IVPB, plus clindamycin, 600 mg IVPB; start continuous infusion of oxacillin, 12 g per 24 h.
8. Unknown: vancomycin, 15 mg/kg IVPB, plus imipenem/cilastatin, 500 mg IVPB, plus gentamicin, 5 mg/kg IVPB; consider liposomal amphotericin therapy, 3 mg/kg infused over 2 h.
9. For suspected toxic shock syndrome: add clindamycin, 600 mg IVPB every 6 h, to one of the above regimens.

Anaphylactic Shock ED Protocol (June 2000)

The goals of the protocol are as follows: MAP, ≥ 70 mm Hg; UO, ≥ 30 mL/h (excluding patients in oliguric renal failure); warm extremities; capillary refill, ≤ 3 s; CVP, > 12 mm Hg; CI, ≥ 3.0 or \(S_{\text{vO}_2} ≥ 60\%\); and avoid significant worsening of oxygenation with fluid therapy (ie, a significant decrease in \(P_{\text{aO}_2}/F_{\text{I}}O_2\) ratio).

The protocol is as follows:
• Epinephrine 1:10,000, 1 mL IV push (IVP) every 5 to 10 min prn, until SBP is at > 90 mm Hg ↓
• Rapidly infused crystalloid ↓
• Inhaled albuterol, 0.25 mg/kg in 3 mL/NS ↓
• 100 mg solumedrol ↓
• Diphenhydramine, 1 mg/kg IVP and up to 50 mg, and cimetidine, 300 mg IVPB ↓
• SBP < 90 mm Hg: continue therapy with crystalloid, colloid, or PRBCs, as indicated by physician ↓
• SBP < 90 mm Hg: repeat epinephrine therapy ↓
• SBP < 90 mm Hg: norepinephrine therapy

Anaphylactic Shock ICU Protocol

The protocol is as follows:
• Intubate if shock continues.
• Consider placement of PA catheter.
• Arterial line.
• Norepinephrine to support mean BP of ≥ 70 mm Hg.
• When MAP is ≥ 70 mm Hg, add dobutamine, 2 µg/kg/min, to norepinephrine therapy.
• As ordered by physician, use volume therapy with NS/LR, colloid, or PRBCs until PCWP is at > 14 mm Hg or until goals are met.
• Wean patient from norepinephrine for MAP of ≥ 80 mm Hg.
• Continue volume therapy to assist with norepinephrine weaning if PCWP is ≥ 18 mm Hg, lungs are not deteriorating, and/or \(F_{\text{I}}O_2\) is ≤ 50%.
• Use appropriate VP and rate to prevent air-trapping on assist control mode (ie, 6 to 10 mL/kg) to keep static inspiratory pressure at ≤ 30 mm Hg.
• If lungs are deteriorating, or \(F_{\text{I}}O_2\) is at < 50%, or \(P_{\text{aO}_2}/F_{\text{I}}O_2\) ratio is < 200 (ie, patient has ARDS), use further volume therapy judiciously.
• Dobutamine therapy to keep CI at > 3.0.
• For GI prophylaxis, administer famotidine drip, 40 mg per 24 h.
• Use sequential compression device.
• Deep vein thrombosis prophylaxis, if no contraindication, with low-molecular-weight heparin; check with physician for dose.
• Placement of duodenal feeding tube per protocol in first 24 h.
• Consider enteral feeding within first 24 h of hospital admission.
• If \(P_{\text{aO}_2}/F_{\text{I}}O_2\) ratio is < 200, place patient on continuous lateral 62°E rotational bed.

Cardiogenic Shock ER Protocol (June 2000)

The goals of the protocol are as follows: MAP, ≥ 60 mm Hg; UO, ≥ 30 mL/h (excluding patients in oliguric renal failure); warm extremities; capillary refill, ≤ 3 s; CI ≥ 2.5 or \(S_{\text{vO}_2} ≥ 60\%\); optimize oxygenation without excessive intrathoracic pressure.

The protocol is as follows:
• Treat HR of < 60 beats/min or supraventricular tachycardia (SVT) of > 120 beats/min, per protocol ↓
• SBP < 90 mm Hg: dopamine therapy ↓
• SBP > 90 mm Hg: dobutamine therapy ↓
• SBP > 110 mm Hg: therapy with nitroglycerine and/or sodium nitroprusside and dobutamine ↓
• Without pulmonary edema ↓
• SBP < 90 mm Hg: fluid challenge protocol ↓
• SBP < 90 mm Hg: dopamine therapy ↓
• Immediate echocardiogram, treat underlying cause: relative hypovolemia; right ventricular or left ventricular ischemia; pulmonary embolism; or air-trapping.
The protocol is as follows:

- Intubate and check immediate echocardiogram if not already done, and check pulsus paradoxus with Doppler ultrasound probe.
- Consider cardiology consultation.
- Treat atrial dysrhythmias.
- Consider placement of \( \text{SvO}_2 \) PA catheter; if HR is < 70 beats/min, place pacing PA catheter.
- If goals are unmet and PCWP is \( \leq 20 \) mm Hg, then give 500-mL fluid bolus and recheck CI and PCWP.
- If not volume responsive and MAP is \( \leq 60 \) mm Hg, add norepinephrine to therapy.
- Add dobutamine, 2 \( \mu \)g/kg/min, when MAP is > 60 mm Hg.
- Repeat fluid challenge if CI increases with fluids and PCWP is \( \leq 22 \) mm Hg without significant worsening of oxygenation (ie, a significant decrease in the \( \text{PaO}_2/\text{FiO}_2 \) ratio).
- If right atrium pressure is greater than PCWP, consider right ventricular dysfunction due to coronary artery disease, pulmonary embolism, COPD, or pulmonary vascular disease. If right atrial pressure is approximately equal to PCWP, consider pericardial tamponade.
- Use volume therapy and dobutamine to wean patient use of vasoconstrictors to lowest dose to keep \( \text{MAP} \) at > 60 mm Hg, CI at \( \geq 2.5 \), \( \text{SvO}_2 \) at \( \geq 60 \%), and UO at \( \geq 30 \) mL/kg.
- Use appropriate \( \text{Vt} \) and rate to prevent air-trapping on assist control mode (ie, 6 to 10 mL/kg) to keep static inspiratory pressure at \( \leq 30 \) mm Hg.
- Treat atrial dysrhythmias:
  1. For SVT, may need therapy with adenosine, 6 to 12 mg IVP, to distinguish flutter and atrial tachycardia from sinus tachycardia.
  2. If in atrial fibrillation/flutter or atrial tachycardia rate of > 120 beats/min and \( \text{MAP} \) is < 60 mm Hg, use electrical cardioversion; if this is unsuccessful, then use rapid infusion of digoxin, 0.5 mg followed by 0.25 mg every 4 h up to a maximum dose of 1.5 mg.
  3. Repeat cardioversion for treatment of atrial tachycardia, flutter, and fibrillation.
  4. If atrial tachycardia continues at \( \geq 120 \) beats/min, consider administering procainamide or amiodarone.
  5. Is atrial dysrhythmia occurs with an HR of > 120 beats/min, \( \text{MAP} \) at > 70 mm Hg, and CI at greater than the target value, consider therapy with a diltiazem drip.
  6. If HR is < 70 beats/min and the patient remains in shock, administer atropine, 1 mg; if unsuccessful, then temporarily use pacing.

- For all cardiogenic shock:
  1. If lungs are deteriorating, or \( \text{FiO}_2 \) is > 50%, or \( \text{PaO}_2/\text{FiO}_2 \) ratio is < 200 (ie, the patient has ARDS), use further volume therapy judiciously.
  2. Administer dobutamine to keep CI at \( \geq 3.0 \), if patient is euvolemic.
  3. For CI prophylaxis, administer fentanyl drip, 40 mg per 24 h.
  4. Use sequential compression device.
  5. For deep vein thrombosis prophylaxis, if there is no contraindication, administer low-molecular-weight heparin; check with physician for dose.
  6. Placement of duodenal feeding tube per protocol in first 24 h.
  7. Consider enteral feeding after 24 h of hospital admission.
  8. If \( \text{PaO}_2/\text{FiO}_2 \) ratio is < 200, place patient on continuous lateral 60°E rotational bed.


29 Bland JM, Altman DG. One and two sided tests of significance. BMJ 1994; 309:248


37 Scolanboring S. On alert. Adv Nurses 2004; 16–18, 28

38 Scolanhoring S. On alert. Adv Nurses 2004; 16–18, 28