Evidence-Based Practice Habits: Transforming Research Into Bedside Practice

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Actions speak louder than words. If that statement is true clinically, it could be said that nursing practice is more connected to tradition than it is evidence based. Many common practices in critical care nursing continue today despite clear and reliable research that contradicts them. The barriers to research implementation that were identified 3 decades ago—lack of time, insufficient administrative support, and limited access to information—are still daunting clinicians today. The importance of basing practice on research is well understood. The barrier is the actual transformation of research findings into practice. The accreditation bodies starting to mandate and evaluate evidence-based practices may help move the implementation of research forward. We must create a culture of inquiry in which nurses are not only aware of the current evidence but also are applying it to practice and asking more questions about traditions that should be supported or refuted with research. Such a culture of inquiry will help to improve patient care and clinical outcomes.

This article is a published report of a 2008 session at the National Teaching Institute and is the second report from that annual session on evidence-based practice. We focus on 4 areas common to everyday critical care practice. Elizabeth Bridges addresses positioning of patients for monitoring hemodynamic parameters. Mary Beth Flynn Makic discusses 2 topics: (1) whether low-dose dopamine prevents or can be used to prevent or treat renal dysfunction and (2) prevention of deep vein thrombosis. Carol A. Rauen describes the facts and physiology of fluid replacement.

PRIME POINTS

- Positioning of patients for monitoring hemodynamic parameters.
- Can low-dose dopamine prevent or be used to prevent or treat renal dysfunction?
- How to prevent deep vein thrombosis.
- Facts and physiology of fluid replacement.

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CE Continuing Education

This article has been designated for CE credit. A closed-book, multiple-choice examination follows this article, which tests your knowledge of the following objectives:

1. Identify the reference lines for the phlebostatic axis
2. Describe the best procedures for prevention of venous thromboembolism
3. Discuss the fluid replacement guidelines established by the Surviving Sepsis Campaign

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current body of evidence that can assist clinicians in moving research to bedside practice are reviewed and recommendations are outlined.

Positioning Patients for Hemodynamic Monitoring

One challenge critical care nurses face is how to answer the question, does my patient need to lie flat for hemodynamic monitoring? In order to address this challenge, a series of questions must be answered: (1) What is the correct reference level for a given position? (2) Are studies in a given population of patients (eg, patients with heart failure, acute respiratory distress syndrome [ARDS], sepsis, cardiac surgery) available that describe the differences in hemodynamic parameters in the supine vs backrest elevated position or supine vs lateral or prone position? (3) Are the observed differences in pulmonary artery pressure (PAP) and central venous pressure (CVP) with the patient in the flat and supine position compared with an alternative position greater than the spontaneous variability in pressure? An exciting aspect of the evidence to answer these questions is that most research on positioning of patients for monitoring hemodynamic parameters has been conducted by nurse researchers.

Position-Specific Reference Level

Regardless of a patient’s body position, the key to accurate measurements of hemodynamic parameters is the use of a position-specific reference level to correct for hydrostatic pressure (Table 1, Figure 1). By convention, the phlebostatic axis is the reference point for the right and left atria.4,5,7,10 The phlebostatic level is a horizontal line through the phlebostatic axis. The air-fluid interface of the stopcock of the transducer must be level with this axis for accurate measurements. In patients with a normal chest wall configuration, the midaxillary line is a valid reference level for the right and left atria; however, use of the midaxillary line in patients with a different chest configuration may result in a pressure difference of up to 6 mm Hg.12 An alternative reference point is 5 cm below the angle of the sternum. This reference point reflects the middle of the right atrium and remains the same up to 60º backrest elevation.13 Use of this alternative reference point, which is also recommended for evaluation of jugular venous distention, results in a CVP measurement that is 3 mm Hg lower than a CVP measured from a system referenced to the phlebostatic axis.13,14 In the lateral position, reference points have been validated for the 30º and 90º lateral positions with a 0º backrest elevation5-7,15 (Table 1). In studies6,16-22 done to evaluate the effects of a prone position on hemodynamic parameters, the midaxillary line or the midanteroposterior

Table 1 Reference level appropriate for various positions of patients

<table>
<thead>
<tr>
<th>Position</th>
<th>Reference level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Supine</td>
<td>Half of the anteroposterior diameter of the chest at the fourth intercostal space (not midaxillary)</td>
</tr>
<tr>
<td>Supine with head of bed elevated</td>
<td>Half of the anteroposterior diameter of the chest at the fourth intercostal space (not midaxillary)</td>
</tr>
<tr>
<td>30º lateral</td>
<td>Half of the vertical distance from the left sternal border to the surface of the bed</td>
</tr>
<tr>
<td>90º lateral</td>
<td>Left lateral decubitus: fourth intercostal space/left parasternal border</td>
</tr>
<tr>
<td></td>
<td>Right lateral decubitus: fourth intercostal space/midsternum</td>
</tr>
</tbody>
</table>

Authors

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Mary Beth Flynn Makic is a researcher nurse scientist for critical care and an assistant professor at the University of Colorado, Denver.

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The diameter of the chest has been used as the reference point, although the accuracy of this reference has not been validated. The reference point should be marked on the patient's chest, and the air-fluid interface of the system should be leveled by using a laser or carpenter's level and not the "eyeball" method.23

Effect of Position on Hemodynamic Parameters

**Supine, Head of Bed Elevated.** Studies in a variety of patients in medical-surgical and cardiac intensive care units (ICUs) indicate that in general PAP and CVP can be obtained reliably with a patient supine with the head of the bed elevated from 0° to 60° if the patient’s legs are parallel to the floor (ie, the patient is not sitting up with the legs in a dependent position).8,24-31 Thermodilution cardiac output can be reliably measured with the head of the bed elevated up to 20°.32,33 In one study,34 continuous cardiac output was measured with the head of the bed elevated up to 45°. A limitation of this research is that it has involved primarily patients whose hemodynamic condition was stable; thus, each patient’s response to a given body position should be evaluated.

**Supine, Trendelenburg/Reverse Trendelenburg.** Hemodynamic measurements should not be obtained with patients in the Trendelenburg position. Although PAP and CVP increase when patients are in the Trendelenburg position, neither intrathoracic blood volume (preload) nor cardiac function increases.35 No research has been done on the effect of the common practice of elevating the head of the bed and then placing the entire bed in the Trendelenburg position to prevent the patient from sliding down in the bed. Also, no research has been done to directly evaluate the effect of use of the reverse Trendelenburg position on PAP and CVP. However, compared with the supine position, a 15° passive tilt decreases cardiac output by 10%, and a 45° tilt decreases cardiac output by approximately 20%.36 This

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**Figure 1** Reference levels for various body positions. A. Supine/prone. The reference point is the phlebostatic axis, which is the intersection of 2 reference lines: first, an imaginary line from the fourth intercostal space at the point where the space joins the sternum, drawn out to the side of the body; second, a line drawn midway between the anterior and posterior surfaces of the chest. B. Supine with the head of the bed elevated. The phlebostatic level is a horizontal line through the phlebostatic axis. Measurements of pulmonary artery pressure and central venous pressure can be obtained at backrest elevations of up to 60°. C, 30° lateral position. The reference point is one-half the distance from the left sternal border to the surface of the bed. (Based on data from VanEtta et al.6) D, 90° lateral position. In the 90° right lateral position, the reference point is the intersection of the fourth intercostal space at the midsternum. In the 90° left lateral position, the reference point is the intersection of the fourth intercostal space at the left parasternal border.

Abbreviations: ICS, intercostal space; L, left; LA, left atrium; R, right.
C and D, Reprinted from Bridges and Woods,9 with permission.
research suggests that patients’ legs should be parallel to the ground while hemodynamic measurements are being obtained.

Lateral Position. Results of early studies showed significant differences in PAP and CVP when measured with the patient in the lateral position (20°-90°) rather than flat and supine. However, in these studies, the phlebostatic axis or the mid-sternum was generally used as the reference point. These reference points, which are not accurate during lateral rotation, introduced measurement error into the results. For example, in the 30° lateral position, use of the mid-sternum rather than the validated angle-specific reference would introduce an error of approximately 7 mm Hg. In contrast, in 2 studies, in which the validated reference point was used, investigators found no clinically significant changes in CVP and PAP in most trauma patients and patients who had undergone cardiac surgery. In the cardiac surgery patients, the supine and lateral measurements of pulmonary artery occlusion pressure differed by less than 2 mm Hg, a finding that most likely reflects the 80- to 460-mL position-induced increase in cardiac output. If the effects of the incorrect reference point were corrected, these original studies would on average have results similar to the results of studies that used the angle-specific reference. In addition, in cardiac and medical-surgical ICU patients, PAP and CVP measured in patients in the 90° position were similar to measurements obtained with the patients supine, as long as the correct angle-specific reference was used. No studies in which the correct angle-specific reference was used have been completed in patients with severe lung disease or in a combined lateral position with the head of the bed elevated.

Prone Position. Patients may be placed prone as a part of therapy for ARDS or during surgical procedures. In patients with acute lung injury or ARDS, if adequate time (30-60 minutes) is allowed for stabilization after repositioning, no clinically significant differences in PAP, CVP, or cardiac output are apparent (Table 2). However, in patients with normal pulmonary function, such as those undergoing spinal surgery, cardiac index may be slightly lower when the patient is prone. Questions to ask include whether abdominal compression in the prone position increases intra-abdominal pressure, and if so, does the increased intra-abdominal pressure affect the accuracy of the PAP and CVP measurements. As demonstrated in Table 3, in patients with normal intra-abdominal pressure, prone positioning does not significantly increase intra-abdominal pressure or intrathoracic blood volume and does not falsely increase CVP. However, the effect of the prone position on hemodynamic parameters in patients with intra-abdominal hypertension (intra-abdominal pressure >12 mm Hg) is not known, an important situation because intra-abdominal hypertension occurs in up to 50% of ICU patients. Additionally, no studies have been done in patients in automated proning beds (eg, Rotoprone), and studies are needed to describe the effects that combined prone positioning with lateral rotation with the bed flat and the prone/reverse Trendelenburg position have on hemodynamic parameters. Normal Variability in Hemodynamic Measurements

The final step is to determine if the observed change in pressure between having a patient supine and having the patient in an alternative position is within the normal variability of the measurements. The following changes are clinically significant (ie, do not reflect normal spontaneous variability):

• Change in pulmonary artery systolic pressure greater than 4 to 7 mm Hg
• Change in pulmonary artery end-diastolic pressure greater than 4 to 7 mm Hg
• Change in pulmonary artery occlusion pressure greater than 4 mm Hg
• Change in cardiac output greater than 10%

Finally, evidence on the effect of position on hemodynamic parameters must be interpreted cautiously. Although on average, hemodynamic parameters do not differ significantly with patients in the various positions, individual patients may respond to a given position in different ways. Thus, it is imperative to systematically assess each patient’s hemodynamic response in a given position before assuming that the measurement will not differ from measurements obtained with the patient supine and flat (Figure 2). The evidence-based recommendations related to monitoring hemodynamic parameters for various body positions are summarized in Table 4.

Renal Dose Dopamine: Does It Exist or Not?

Use of low-dose dopamine, or renal dose dopamine, has become a
widely accepted clinical practice for preventing or treating renal dysfunction. Does this agent truly protect the kidneys from acute dysfunction? The evidence does not support the use of low-dose dopamine to prevent or treat renal dysfunction. In fact, multiple studies have shown no evidence that dopamine prevents renal dysfunction or provides renal protection, and the agent may even be harmful for patients.

Dopamine is a drug with diverse effects at multiple receptor sites in the body; this endogenous catecholamine regulates cardiac, vascular, and endocrine function. Dopamine is a complex agent; the

### Table 2  Measurements of hemodynamic parameters: prone vs supine

<table>
<thead>
<tr>
<th>Type of patients</th>
<th>Reference level</th>
<th>Parameters</th>
<th>Supine</th>
<th>Prone</th>
<th>Stabilization, min</th>
</tr>
</thead>
<tbody>
<tr>
<td>ARDS (n = 15)¹⁵</td>
<td>Mid-AP</td>
<td>MAP, mm Hg</td>
<td>84 (19)</td>
<td>84 (16)</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td></td>
<td>PAM, mm Hg</td>
<td>31 (8)</td>
<td>29 (8)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>PAOP, mm Hg</td>
<td>14 (5)</td>
<td>13 (5)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>CI⁵</td>
<td>5.1 (1.9)</td>
<td>4.9 (1.6)</td>
<td></td>
</tr>
<tr>
<td>ALI (n = 16)¹⁶</td>
<td>MAL</td>
<td>MAP, mm Hg</td>
<td>77 (10)</td>
<td>82 (11)</td>
<td>60</td>
</tr>
<tr>
<td></td>
<td></td>
<td>PAM, mm Hg</td>
<td>16 (5)</td>
<td>16 (6)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>PAOP, mm Hg</td>
<td>987 (191)</td>
<td>1033 (189)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>ITBVIF⁶</td>
<td>4.1 (1.1)</td>
<td>4.4 (0.7)</td>
<td></td>
</tr>
<tr>
<td>ALI (n = 12)¹⁷</td>
<td>MAL</td>
<td>HR, beats per min</td>
<td>78 (16)</td>
<td>82 (16)</td>
<td>60</td>
</tr>
<tr>
<td></td>
<td></td>
<td>MAP, mm Hg</td>
<td>75 (10)</td>
<td>81 (11)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>PAM, mm Hg</td>
<td>16 (5)</td>
<td>15 (5)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>PAOP, mm Hg</td>
<td>3.8 (0.9)</td>
<td>4.2 (0.6)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>ITBVIF⁶</td>
<td>1008 (187)</td>
<td>1036 (180)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>DO₂id</td>
<td>558 (122)</td>
<td>620 (74)</td>
<td></td>
</tr>
<tr>
<td>ARDS (n = 23)¹⁹</td>
<td>Mid-AP</td>
<td>CO, L/min</td>
<td>15</td>
<td>14</td>
<td>60-90 (20 minutes after stabilization of oxygen saturation obtained via pulse oximetry)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>PAOP, mm Hg</td>
<td>15</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>CVP, mm Hg</td>
<td>3.5</td>
<td>3.4</td>
<td></td>
</tr>
<tr>
<td>ARDS (n = 11)²¹</td>
<td>No data</td>
<td>CVP, mm Hg</td>
<td>10 (2)</td>
<td>11 (3)</td>
<td>30</td>
</tr>
<tr>
<td></td>
<td></td>
<td>CVP, mm Hg</td>
<td>4.9 (1.1)</td>
<td>4.8 (1.3)</td>
<td></td>
</tr>
<tr>
<td>ARDS (n = 19)²³</td>
<td>No data</td>
<td>MAP, mm Hg</td>
<td>87 (9)</td>
<td>89 (14)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>PAM, mm Hg</td>
<td>12 (4)</td>
<td>13 (5)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>PAOP, mm Hg</td>
<td>34 (8)</td>
<td>37 (8)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>CI⁵</td>
<td>15 (5)</td>
<td>17 (6)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>ITBVIF⁶</td>
<td>5.5 (1.5)</td>
<td>5.7 (1.7)</td>
<td></td>
</tr>
<tr>
<td>Routine cardiac imaging (n = 48)²⁶</td>
<td>No data</td>
<td>EDV, mL</td>
<td>117 (36)</td>
<td>111 (39)</td>
<td>60-90 (20 minutes after stabilization of oxygen saturation obtained via pulse oximetry)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SV, mL</td>
<td>61 (15)</td>
<td>56 (13)</td>
<td></td>
</tr>
<tr>
<td>Lumbar spine surgery (n = 15)²⁴</td>
<td>No data</td>
<td>CVP, mm Hg</td>
<td>9 (2)</td>
<td>11 (2)</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SVI⁸</td>
<td>34 (9)</td>
<td>32 (11)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>CI⁵</td>
<td>2.2 (0.5)</td>
<td>2.0 (0.6)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>EDV, mL</td>
<td>128 (46)</td>
<td>100 (48)</td>
<td></td>
</tr>
<tr>
<td>Lumbar spine surgery (n = 40)²⁴</td>
<td>No data</td>
<td>MAP, mm Hg</td>
<td>92 (17)</td>
<td>87 (16)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>CVP, mm Hg</td>
<td>3.1 (0.7)</td>
<td>2.7 (0.6)</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: ALI, acute lung injury; ARDS, acute respiratory distress syndrome; CI, cardiac index; CO, cardiac output; CVP, central venous pressure; DO₂, oxygen delivery; DO₂id, oxygen delivery index; EDV, end-diastolic volume; HR, heart rate; ITBVIF, intrathoracic blood volume index; MAL, midaxillary line; MAP, mean arterial pressure; Mid-AP, midanteroposterior chest diameter; PAM, mean pulmonary artery pressure; PAOP, pulmonary artery occlusion pressure; SV, stroke volume; SVI, stroke volume index.

²⁵ P < .05 (prone vs supine measurement).
²⁶ Cardiac index, calculated as cardiac output in liters per minute divided by body surface area in square meters.
²⁷ Intrathoracic blood volume index, calculated as intrathoracic blood volume in milliliters divided by body surface area in square meters.
²⁸ Oxygen delivery index, calculated as oxygen delivery in milliliters per minute divided by body surface area in square meters.
²⁹ Stroke volume index, calculated as stroke volume in milliliters divided by body surface area in square meters.
response to it depends on which receptors in the body are stimulated (Table 5). Conventional dosing of dopamine suggests that low dosages (0.5-3.0 μg/kg per minute) stimulate dopaminergic receptors and result in coronary and renal vasodilatation, natriuresis, and diuresis. Midrange dosing (3-8 μg/kg per minute) activates β-adrenergic receptors, increasing cardiac inotropy and chronotropy. Dosages greater than 8 μg/kg per minute predominantly stimulate α-adrenergic receptors, resulting in splanchnic and peripheral vasoconstriction.64-67 This conventional dosing is inaccurate. Research suggests that dopamine infusions at similar infusion rates produce different responses from patient to patient.66 One explanation of the variation in responses is that the activation of the receptor sites depends more on the patient than on the dose; thus the concept of dose range affecting specific receptors is not universal for all patients.66,67 As a result, traditional dopamine dosing should not be used as a standard regimen. The desired effects of dopamine infusion depend on the specific patient’s response to the agent.56,64,66,67

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Position, mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abdominal pressure, mm Hg</td>
<td>10 (3) 13 (4)</td>
</tr>
<tr>
<td>Mean arterial pressure, mm Hg</td>
<td>75 (10) 81 (11)</td>
</tr>
<tr>
<td>Cardiac indexb</td>
<td>3.8 (0.9) 4.2 (0.6)</td>
</tr>
<tr>
<td>Heart rate, beats per min</td>
<td>78 (16) 82 (16)</td>
</tr>
<tr>
<td>Right atrial pressure, mm Hg</td>
<td>16 (5) 15 (5)</td>
</tr>
<tr>
<td>Intrathoracic blood volume, mL/m²</td>
<td>1008 (187) 1036 (180)</td>
</tr>
</tbody>
</table>

a Based on data from Hering et al.17,18
b Cardiac index, calculated as cardiac output in liters per minute divided by body surface area in square meters.

What is the evidence for the effectiveness of renal dose dopamine in preventing acute renal failure? Concern about the use of renal dose dopamine to prevent and treat renal dysfunction began to appear in the literature in the early 1990s.66 Denton et al.66 wrote a classic article discussing the science related to renal dose dopamine. They reviewed the literature and discussed the findings from several research studies in which low-dose dopamine augments renal blood flow, glomerular filtration rate, and urine output in healthy humans. Denton et al., however, did not find similar outcomes when critically ill patients were studied. They reported that most studies in humans on the effects of renal dose dopamine and critical illness did not indicate an improvement in renal function and prevention of acute renal failure. Denton et al. discouraged the use of renal dose dopamine in critically ill patients to prevent or treat renal dysfunction.

In a report published in 1999, Marik and Iglesias65 concluded that giving low-dose dopamine to patients with septic shock and oliguria did not lead to any significant differences in the incidence of acute renal failure, need for dialysis, or 28-day survival. Then Kellum and Decker69 did a meta-analysis of the use of dopamine in acute renal failure. They concluded that the use of low-dose dopamine to treat or prevent acute renal failure cannot be justified on the basis of available evidence and should be eliminated from critical care protocols.50

Despite this evidence, the ongoing clinical use of low-dose dopamine continued. Friedrich et al.62 published a meta-analysis and evidence-based review on low-dose dopamine, concluding that after 15 years of research on the effectiveness of renal dose dopamine, the evidence indicates that low-dose dopamine temporarily improves renal output but does not prevent renal dysfunction or death. Thus, the evidence is conclusive: use of low-dose dopamine does not prevent or improve renal dysfunction long-term in critically ill patients.53,60,62,64,66 These findings should not be confused with results of studies that examined the effectiveness of higher doses of dopamine in critically ill patients with heart failure and septic shock. In such patients, dopamine is beneficial for its inotropic and vasoactive properties.46,65

So why does urine output increase when a dopamine infusion is started? Dopamine has both natriuretic and diuretic properties that stimulate urine output, and the response appears to be more pronounced at lower dosages.54,66,67 This response, however, is often temporary, and urine output tapers off within the first 24 hours.45 Dopamine at doses as low as 2 μg/kg per minute improves cardiac output and mean arterial pressure, enhancing renal perfusion and urine output.66-68

Concerns about the use of low-dose dopamine extend beyond the evidence that the drug is not effective in preventing renal dysfunction. [28]
Current evidence suggests low-dose dopamine may cause harm by worsening splanchnic oxygen consumption, impairing gastric motility, inducing tachyarrhythmias (especially in elderly patients), and blunting ventilatory response to hypercarbia. Administration of dopamine should be continually evaluated to match the dose to the desired outcome without causing adverse consequences for the patient.

Does renal dose dopamine exist? No, it does not. Dopamine does not protect the kidneys from renal dysfunction.


Venous thromboembolism is the combined term that describes both...
deep vein thrombosis (DVT) and pulmonary embolism. Recent estimates suggest that venous thromboembolism is diagnosed in more than 900,000 patients in the United States annually, with approximately 400,000 cases manifested as DVT and 500,000 cases as pulmonary embolism. In 60% of the patients with pulmonary embolism, the embolism is fatal.71,72 Patients in whom venous thromboembolism develops are also at risk for post-thrombotic syndrome, in which tissue injury follows DVT and lasts indefinitely, causing damage of venous valves, pain, paresthesia, hyperpigmentation, pruritus, venous dilatation, edema, and ulceration.73,74 Research findings71,75 indicate that clinical interventions, including mechanical and pharmacological therapies, are effective in preventing venous thromboembolism; however, only approximately one-third of all patients at risk for venous thromboembolism receive prophylactic therapy. The

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**Table 4** Summary of recommendations for hemodynamic pressure measurements with patients in different body positions

<table>
<thead>
<tr>
<th>Position-specific reference levels</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Supine</td>
<td>Phlebostatic axis (half of the anteroposterior diameter of the chest at the fourth intercostal space)</td>
</tr>
<tr>
<td>Supine with head of bed elevated</td>
<td>Phlebostatic axis/level (half of the anteroposterior diameter of the chest at the fourth intercostal space)</td>
</tr>
<tr>
<td>30° lateral</td>
<td>Half the vertical distance from the left sternal border to the surface of the bed</td>
</tr>
</tbody>
</table>
| 90° lateral                       | Left lateral decubitus: fourth intercostal space/left parasternal border  
                                          Right lateral decubitus: fourth intercostal space/midsternum |
| Prone/flat                        | Phlebostatic axis or midaxillary line (limited research to validate this reference level) |

**Effect of patient’s position on hemodynamic parameters (compared with supine/flat position)**

| Supine/head of bed elevated       | Pulmonary artery pressure and central venous pressure: head of bed elevated up to 60°; patient’s legs must be parallel to the floor424–27  
                                          Thermodilution cardiac output: head of bed elevated up to 20°23,28  
                                          Continuous cardiac output: head of bed elevated up to 45°34 |
| Supine with Trendelenburg; reverse Trendelenburg | Not recommended: patient’s legs must be parallel to floor |
| 30° lateral                       | Pulmonary artery pressure and central venous pressure: pressures can be measured reliably as long as the correct angle-specific reference point/level is used  
                                          No studies have validated this position with the head of the bed elevated |
| 90° lateral                       | Pulmonary artery pressure and central venous pressure: can be measured reliably as long as the correct angle-specific reference point/level is used |
| Prone (flat)                      | Pulmonary artery pressure, central venous pressure, and cardiac output: can be measured reliably; allow 20-60 minutes for patient to stabilize after position change |

**Normal variability in hemodynamic parameters (use this information to evaluate if change in value is greater than expected variability for that parameter)**

| Change in pulmonary artery systolic pressure | >4–7 mm Hg (increased variability with decreased ejection fraction) |
| Change in pulmonary artery end-diastolic pressure | >4–7 mm Hg (increased variability with decreased ejection fraction) |
| Change in pulmonary artery occlusion pressure | >4 mm Hg |
| Change in cardiac output | >10% |

---

**Table 5** Characteristics of dopamine

<table>
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<tr>
<th>Receptor sites stimulated</th>
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</table>
| Dopaminergic              | β-Adrenergic  
                                          α-Adrenergic |
| Physiological actions     | Improves cardiac output and mean arterial pressure  
                                          Stimulates natriuresis and diuresis (limited response)  
                                          Has no effect on renal function/glomerular function |
| Adverse actions           | Worsens splanchnic oxygen consumption  
                                          Impairs gastric motility  
                                          Induces tachyarrhythmias, especially in elderly patients  
                                          Blunts ventilatory response to hypercarbia |

---
most common reasons cited for lack of proper prophylaxis of venous thromboembolism include lack of knowledge among providers, underestimation of patients’ risk for venous thromboembolism, and overestimation of the potential risk of bleeding associated with prophylaxis.74-77

Prevention of venous thromboembolism is considered a clear opportunity for improving safe care of patients.77 Several national organizations and accrediting bodies list the prevention of venous thromboembolism as a patient safety indicator and measure of quality of care or “never events.”71,78 In 2003, The American Public Health Association published guidelines to advance public awareness of DVT.76 Geerts et al75 published evidence-based guidelines for the prevention of venous thromboembolism, and that seminal article was followed in 2008 with practice guidelines for antithrombotic therapy for venous thromboembolism.79 Evidence is available to guide practice for providing interventions to prevent venous thromboembolism. The challenge is to use this evidence in the daily practice of critical care nursing.

Prevention of venous thromboembolism begins with assessment of a patient’s risk factors. A venous thromboembolism is an intravascular fibrin clot that usually forms in regions of slow or disturbed blood flow. Typically the clot forms in a large vein in the lower extremities, but it may form in any large vein and poses a great risk when it occludes a pulmonary vessel, potentially resulting in a fatal pulmonary embolism. Classic risk factors for ICU patients are well known by nurses. The variables are referred to as the Virchow triad: venous stasis or obstruction, blood vessel injury, and increased coagulability. Frequent procedures disrupt a patient’s vessels, and the fluid shifts, immobility, and coagulation disorders associated with critical illness place ICU patients at high risk for venous thromboembolism. DVT develops in up to 30% of ICU patients within the first week of admission, a characteristic that further emphasizes the importance of early interventions.80 To decrease the prevalence of venous thromboembolism, critical care nurses must evaluate each patient’s risk and implement preventative interventions when the patient is admitted to the unit.

Additional risk factors beyond venous stasis, immobility, and vascular injury should be included in the assessment of each patient’s risk for venous thromboembolism. Risk factors can be grouped in many ways to include patient-specific variables, type of procedure a patient is undergoing (eg, orthopedic surgery), and reason for admission (eg, traumatic event; Table 6). Top risk factors include prolonged immobility, including use of neuromuscular blockade and/or heavy sedation; an indwelling central venous catheter; major surgery; cancer; active infection; pregnancy; hormone therapy; obesity; respiratory failure; heart failure; cerebral vascular accident; trauma (especially fractures of the pelvis, hip, or leg); history of previous venous thromboembolism; and older age (ie, risk increases in patients 40 years or older).75,81 The more risk factors a patient has, the more aggressive the interventions should be.

Preventive actions are categorized as mechanical or pharmacological interventions and are implemented on the basis of the assessment of

<table>
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<th>Table 6 Risk assessment and interventions for venous thromboembolism</th>
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<td><strong>General risk factors for all patients</strong></td>
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<td>History of venous thromboembolism</td>
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<td>Age &gt; 40 years</td>
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**Interventions**
- Unfractionated or low-molecular-weight heparin
- Mechanical devices
- Fondaparinux, or warfarin
- Mechanical devices

**Risk assessment**
- Low risk
- Age < 40 years
- Minor surgery
- Moderate risk
- Age > 40 years
- Minor surgery with additional risk factor
- Age 40-60 years with no additional risk factor
- High risk
- Surgery in patients > 60 years old
- Highest risk
- Age > 40 years with multiple risk factors in the first week of admission
- Hip or knee surgery or interventions
- Major trauma, spinal cord injury
Mechanical prevention encompasses early ambulation, graduated compression stockings (also known as elastic compression stockings), and intermittent pneumatic compression devices. However, compliance of patients and health care practitioners limits the effectiveness of mechanical prevention. For mechanical prevention to be effective, the device(s) must be appropriately fitted to each patient and consistently applied to the lower extremities. Often, patients are not appropriately measured for correct fit of a mechanical device. In addition, ongoing assessment of the fit of the device as body fluids shift may not be addressed, and the device is not consistently placed on the patient’s extremities, limiting the effectiveness of mechanical therapy. 

Pharmacological prevention consists of unfractionated heparin, low-molecular-weight heparin, fondaparinux, and vitamin K antagonists (eg, warfarin). Many variables must be assessed before pharmacological interventions are started, including the risk for venous thromboembolism, presence of bleeding, and desired duration of therapy. As a patient’s risk increases, more aggressive pharmacological therapy combined with mechanical interventions is required. Low-molecular-weight heparin is often prescribed because the evidence suggests that this agent is as effective as unfractionated heparin in preventing DVT, has fewer adverse effects (eg, bleeding, heparin-induced thrombocytopenia) and better bioavailability, and is safe in the outpatient setting, allowing for long-term therapy as needed. High-risk patients may require more aggressive treatment with low-molecular-weight heparin, fondaparinux, or warfarin. The evidence on aspirin is well established: aspirin alone is not effective in preventing DVT. The 2008 guidelines of the American College of Chest Physicians provide a full review of the evidence supporting antithrombotic therapy.

What are the best procedures for preventing DVT? First and foremost, preventive interventions must be implemented consistently for all critically ill patients to effectively decrease the incidence of venous thromboembolism. Second, patients should be assessed for severity of risk on the basis of age, medical and surgical history, and projected course of the critical illness. Third, each patient’s plan of care should be reviewed and pharmacological therapy that may help reduce the risk for venous thromboembolism should be discussed. Fourth, mechanical devices must be correctly fitted and consistently applied for effective preventative therapy. Finally, when possible, patients should ambulate (Table 7). Although all venous thromboembolism may not be preventable in critically ill patients, the evidence indicates that its occurrence can be reduced, and nurses owe it to patients to base practice on the best evidence to minimize the risk for venous thromboembolism.

Fluid Replacement: Facts and Physiology

Fluid replacement has long been a cornerstone of critical care practice. Historically, the question was not whether a patient needed fluids but what type of fluid would be best. Now the physiological value of administering fluids is being questioned. The goal in fluid replacement is clear: maintain adequate intravascular volume to ensure cellular oxygen delivery and cardiac output. The means to achieve that goal, however, is much more complex.

Both crystalloids and colloids have significant advantages and disadvantages. Crystalloids, which include normal saline and lactated Ringer solutions, are isotonic, inexpensive, readily available, good volume expanders that are easy to store and administer. These fluids do not transmit diseases or cause allergic reactions, and both can replace some
electrolytes. However, normal saline and lactated Ringer solutions do not have oxygen-carrying capacity, and approximately 75% of the fluid administered leaks into the interstitial space within hours of administration. Large volumes of these fluids can lead to pulmonary edema and, because the blood components are diluted, can actually lead to more bleeding. The natural and synthetic diluted, can actually lead to more bleeding. The increase in blood pressure in the pulmonary capillary pressures because of the high hydrostatic pressure and left ventricular failure. Tachycardia is often used as an indicator of anemia or dehydration, but Tachycardia is a warning sign for abnormal heart rhythm, which is not a particularly sensitive measure.

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In a study of early goal-directed therapy, Rivers et al attempted to answer the question of replacement end points. Emergency department patients in septic shock had better outcomes when they had early goal-directed replacement with the following end points:

\[
\begin{align*}
\text{CVP: } & 8 \text{ to } 12 \text{ cm H}_2\text{O} \\
\text{Mean arterial pressure: } & \text{greater than } 65 \text{ mm Hg}
\end{align*}
\]

Dubois et al studied a mixed population of medical-surgical ICU patients and found that albumin might even have an advantage over normal saline: Crystalloids dilute clotting factors and platelet volumes. The current recommendation for suspected arterial bleeding is to delay fluid replacement until surgery to control the bleeding is under way. It is widely believed that warm fluid is better than cold. Being cold lowers the core temperature and makes coagulopathies worse. The question remains: what should the end-point parameters be for fluid replacement?

The issue of how much fluid to administer or when to stop is a difficult one. Vincent and Weil question the traditional clinical parameters that have been used for decades. Historically, fluids were cut back when CVP increased. If the goal is intravascular fluid replacement, it must be remembered that CVP reflects only the pressure in the central veins. It does not represent total vascular volume. The same could be said for pulmonary edema. Fluid could be leaking into the lung interstitial spaces because of the high hydrostatic pressure in the pulmonary capillary bed or because of the low oncotic pressure and left ventricular failure. Tachycardia is often used as an indicator of anemia or dehydration, but Tachycardia is a warning sign for many problems in critical care.

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Urine output: greater than 0.5 mL/kg per hour

Central venous oxygen saturation: 70%

These parameters were part of the Surviving Sepsis Campaign guidelines in 2004 and were recommended a second time in the 2008 guidelines. The other recommendations in the 2008 guidelines include fluid replacement with 300 to 500 mL of colloid or 1 L of crystalloids in 30 minutes and reducing the fluid challenge rate if filling pressures in 30 minutes and reducing the fluid replacement with 300 to 500 mL of colloid or 1 L of crystalloids in 30 minutes and reducing the fluid challenge rate if filling pressures increase without improvement in hemodynamic status.

How much blood is the right amount of blood has also been researched. The first hemoglobin trigger that was recommended dates back to Adam and Lundy in 1942. The “10/30” rule was used in clinical practice for more than 4 decades. If a patient’s hemoglobin level decreased to less than 10 g/dL or the hematocrit decreased to less than 0.30, the patient was given blood. The discovery that human immunodeficiency virus was transmitted via blood and the expanding knowledge of the risks of blood administration have led clinicians to reevaluate the risks and benefits of administering blood and to search for a better trigger threshold. The report of the current landmark study, published in 1999, included recommendations that hemoglobin level be maintained between 7 and 9 g/dL. The 2004 and 2008 Surviving Sepsis Campaign guidelines included this recommendation. The standard exceptions to the trigger value of 7 to 9 g/dL are patients with ischemic heart disease and patients who have had an acute myocardial infarction. Corwin et al reported that, despite the commonly known risks of blood administration and the new recommendations, clinical practice in the United States did not change much between 1999 and 2003. In a 2008 analysis of data on blood transfusions included in the Sepsis Occurrence in Acutely Ill Patients database, Vincent et al found that administration of blood was associated with improved mortality. More randomized controlled studies are needed in critically ill patients to answer these age-old questions of which solution, how much, and when best to deliver fluids to critically ill patients.

In the absence of specific evidence-based practice guidelines for fluid replacement in critically ill patients, “For the present, the choice is best made contingent on the underlying disease, the type of fluid that has been lost, the severity of circulatory failure, the serum albumin concentration of the patient and the risk of bleeding.”

The short answer to the simple question of fluid replacement is that no perfect solution exists. Patients should be given what they lost or what they need that will cause the least harm. Nurses must continue to conduct research to find a better answer.

Summary

This article is the second article published in Critical Care Nurse that outlines evidence-based practices that should be applied at the bedside. The challenge before us is threefold: we must continue to ask the hard clinical questions, conduct the research to answer these questions, and implement the discoveries that are made. This final challenge is probably the most difficult. We fear that in today’s environment of cost cutting and staffing shortages, uncritical adherence to tradition will become the norm again. We must create a culture of inquiry and practice changes based on research and implement new standards that are based on the latest available evidence. CCI

Acknowledgments

The authors served as an expert panel on evidence-based practice at the 2008 National Teaching Institute in Chicago, Illinois. We thank the American Association of Critical-Care Nurses, Linda Bell, Nancy Munro, and the entire Advance Practice Work Group for their insight in pulling the panel together to present this information at the 2008 National Teaching Institute.

Financial Disclosures

None reported.

References


88. American College of Surgeons’ Committee on Trauma. Advanced Trauma Life Support for Doctors. 8th ed. Chicago, IL: American College of Surgeons; 2008.


Renal Dose Dopamine: Does It Exist or Not?

- Low-dose dopamine does not prevent or improve renal dysfunction long term in critically ill patients.
- Low-dose dopamine may worsen splanchnic oxygen consumption, impair gastric motility, induce tachyarrhythmias (especially in elderly patients), and blunt the ventilatory response to hypercarbia.


- Patients should be assessed for risk of venous thromboembolism on the basis of age, medical and surgical history, and projected course of the critical illness (see Table).
- Both mechanical and pharmacological interventions to prevent venous thromboembolism must be implemented consistently.

Fluid Replacement: Facts and Physiology

- More research is needed to determine which solution, how much, and when best to deliver fluids to critically ill patients.

### Table: Risk assessment and interventions for venous thromboembolism

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</tr>
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<td>Age &lt; 40 years</td>
<td>Early ambulation</td>
</tr>
<tr>
<td>Cancer</td>
<td>Minor surgery</td>
<td>Elastic stockings</td>
</tr>
<tr>
<td>Pregnancy</td>
<td>Moderate risk</td>
<td>Moderate to high risk</td>
</tr>
<tr>
<td>Hormone therapy</td>
<td>Age &gt; 40 years</td>
<td>Unfractionated or low-molecular-weight heparin</td>
</tr>
<tr>
<td>Obesity</td>
<td>Minor surgery with additional risk factor</td>
<td>Mechanical devices</td>
</tr>
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<td>Age 40-60 years with no additional risk factor</td>
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<tr>
<td>Acute infection</td>
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This article and an online version of the CE test may be found online at www.ccnonline.org
1. Which of the following reference levels is used for measuring hemodynamic parameters in supine patients with the head of the bed (HOB) elevated?  
   a. Half of the anterior-posterior diameter of the chest at the second intercostal space  
   b. Half of the anterior-posterior diameter of the chest at the third intercostal space  
   c. Half of the anterior-posterior diameter of the chest at the fourth intercostal space  
   d. Half of the anterior-posterior diameter of the chest at the fifth intercostal space

2. Which of the following HOB elevations provides reliable measurements of pulmonary artery pressure and central venous pressure in supine patients?  
   a. 0° to 30°  
   b. 0° to 45°  
   c. 0° to 60°  
   d. 0° to 90°

3. Which of the following HOB elevations provides reliable measurements of thermodilution cardiac output?  
   a. 20°  
   b. 30°  
   c. 45°  
   d. 60°

4. The reverse Trendelenburg position has which of the following effects on hemodynamic parameters?  
   a. Decreased pulmonary artery pressure  
   b. Decreased mean arterial pressure  
   c. Decreased central venous pressure  
   d. Decreased cardiac output

5. Which of the following dosages of dopamine activates beta-adrenergic receptors, increasing cardiac inotropy and chronotropy?  
   a. 1 to 3 μg/kg per minute  
   b. 3 to 8 μg/kg per minute  
   c. 8 to 12 μg/kg per minute  
   d. 12 to 16 μg/kg per minute

6. Current evidence suggests that low-dose dopamine can be harmful due to which of the following?  
   a. Worsening splanchic oxygen consumption  
   b. Excessive natriuresis and diuresis  
   c. Increased gastric emptying  
   d. Blunting ventilatory response to hypocarbia

7. Which of the following percentage of patients at risk for venous thromboembolism receives prophylactic therapy?  
   a. 25%  
   b. 33%  
   c. 50%  
   d. 66%

8. Which of the following patients are at the highest risk for venous thromboembolism?  
   a. Patients younger than 40 years old who have had minor surgery  
   b. Patients older than 40 years old who have with minor surgery and an additional risk factor  
   c. Patients older than 60 years old with surgery  
   d. Age greater than 40 years with multiple risk factors

9. Evidence suggests that the most effective method for providing mechanical prevention of venous thromboembolism is which of the following?  
   a. Thigh-high compression stockings  
   b. Knee-high compression stockings  
   c. Intermittent pneumatic compression devices  
   d. Compression stockings and compression devices

10. Which of the following endpoints is used for fluid replacement in the Surviving Sepsis Campaign guidelines?  
    a. Central venous pressure of 12 to 15 cm H2O  
    b. Urine output greater than 1 mL/kg per hour  
    c. Central venous oxygen saturation of 60%  
    d. Mean arterial pressure greater than 65 mm Hg

11. Which of the following is recommended as fluid replacement in the Surviving Sepsis Campaign guidelines?  
    a. 250 mL of colloids over 30 minutes  
    b. 500 mL of colloids over 60 minutes  
    c. 1000 mL of crystalloids over 30 minutes  
    d. 2000 mL of crystalloids over 60 minutes

12. Which of the following hemoglobin levels is recommended as a trigger value for blood replacement in the Surviving Sepsis Campaign guidelines?  
    a. 6 to 7 g/dL  
    b. 7 to 9 g/dL  
    c. 9 to 10 g/dL  
    d. 10 to 12 g/dL

Test answers: Mark only one box for your answer to each question. You may photocopy this form.