Seven Evidence-Based Practice Habits: Putting Some Sacred Cows Out to Pasture
Carol A. Rauen, Marianne Chulay, Elizabeth Bridges, Kathleen M. Vollman and Richard Arbour

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Evidence-Based Practice

Seven Evidence-Based Practice Habits: Putting Some Sacred Cows Out to Pasture

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Nursing has deeply rooted traditions. As far back as Florence Nightingale in the 19th century, nurses prided themselves on patient advocacy, infection control (before germ theory), and physical care of the entire body, not limiting the focus to management of disease or signs and symptoms.1 These early roots established the philosophy of nursing. Nurses labeled what they do as caring or the art of nursing.

Critical care nurses find themselves in a unique situation. We have our feet deeply rooted in the art of nursing. Yet our hands and minds reach for the scientific basis that our highly technical, physiological, and pharmacological specialty requires. To base our practice on science, we must use research to answer questions, establish protocols, and promote critical thinking and decision making at the bedside. Doing so requires us to be willing and able to change practices, regardless of the tradition and commonly held beliefs, if validated, reliable, and useful evidence leads to such change. Nurses are at the forefront of evidence-based approaches.2

The Institute of Medicine defines evidence-based practice (EBP) as “the integration of best research, clinical expertise, and patient values in making decisions about the care of individualized patients.”3 Research findings are a collection of facts. They become evidence when the findings are relevant and useful in particular clinical circumstances.4 Using research to guide clinical decision making is a shift in culture from basing decisions on opinion, past experiences, and precedents to basing decisions on science, research, and evidence.5 The Agency for Healthcare Research and Quality published Making Health Care Safer: A Critical Analysis of Patient Safety Practices.6 This document outlines 79 evidence-based practices and targets related to patient safety. The 11 recommendations with the strongest research support have a direct connection to critical care practice (Table 1).

PRIME POINTS

• About 30% to 40% of patients do not receive care consistent with current scientific evidence.

• Are we doing what is best for our patients with the current evidence available to us?

• Do not instill normal saline (physiological salt solution) before endotracheal suctioning.

• Use chest radiography to confirm correct placement of nasogastric tubes.

• Attention to correct placement of electrocardiography leads is imperative.
In this article, we cover 7 evidence-based practice (EBP) recommendations that clinicians should consider implementing into their practice. Much of this research is not new and has met with resistance at the bedside despite clear evidence that it represents best practice. We also address the traditional approach and offer recommendations for implementing the changes. Marianne Chulay addresses instillation of normal saline (physiological salt solution) with endotracheal suctioning and verification of nasogastric tube placement. Elizabeth Bridges reviews the current evidence and recommendations for accurate measurement of blood pressure and selection of electrocardiography leads. Kathleen Vollman delineates the research and recommendations for patients’ positioning and mobility. Richard Arbour discusses use of the Glasgow Coma Scale in neurological assessment and management of intracranial hypertension.

**Instillation of Normal Saline Before Endotracheal Suctioning: Helpful or Harmful?**

Most hospital policies and procedures for management of artificial airways include instilling 5 to 10 mL of normal saline before endotracheal suctioning is done. This nursing and respiratory therapy routine was advocated as a way to improve oxygenation and removal of secretions by thinning thick secretions and stimulating coughing to assist with mobilization of secretions. Although instillation of normal saline is a long-practiced suctioning intervention, no research has ever documented the benefit of this practice, and some researchers have found the practice potentially harmful.

**Effect on Oxygenation**

In most experimental studies on the effect of instillation of normal saline before endotracheal suctioning, oxygen saturation or PaO₂ was evaluated as the primary end point; in only a single study was mixed venous oxygenation evaluated. In these studies, oxygen saturation was significantly lower with instillation of saline than with no instillation of saline, or the results of the 2 methods (saline vs no saline) did not differ. In no studies to date did instillation of normal saline before suctioning improve oxygenation.

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**Table 1** Practices related to patients’ safety strongly supported by evidence

<table>
<thead>
<tr>
<th>Practice</th>
</tr>
</thead>
<tbody>
<tr>
<td>Appropriate use of prophylaxis to prevent venous thromboembolism in patients at risk</td>
</tr>
<tr>
<td>Use of perioperative β-blockers in appropriate patients to prevent perioperative morbidity and mortality</td>
</tr>
<tr>
<td>Use of maximum sterile barriers while placing central intravenous catheters to prevent infections</td>
</tr>
<tr>
<td>Appropriate use of antibiotic prophylaxis in surgical patients to prevent postoperative infections</td>
</tr>
<tr>
<td>Asking that patients recall and restate what they have been told during the informed consent process</td>
</tr>
<tr>
<td>Continuous aspiration of subglottic secretions to prevent ventilator-associated pneumonia</td>
</tr>
<tr>
<td>Use of pressure-relieving bedding materials to prevent pressure ulcers</td>
</tr>
<tr>
<td>Use of real-time ultrasound guidance during insertion of central catheters to prevent complications</td>
</tr>
<tr>
<td>Patient self-management of warfarin to achieve appropriate outpatient anticoagulation and prevent complications</td>
</tr>
<tr>
<td>Appropriate provision of nutrition, with a particular emphasis on early enteral nutrition in critically ill and surgical patients</td>
</tr>
<tr>
<td>Use of antibiotic-impregnated central venous catheters to prevent catheter-related infections</td>
</tr>
</tbody>
</table>

*Based on information from Shojania et al.*

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the results. In the one, small study\textsuperscript{16} (N = 12) in which the weight of the saline was taken into account, serious flaws in the study design (lack of randomization of the interventions) make the results invalid.

Although an alleged benefit of instillation of saline is improvement in removal of secretions, to date no adequately reported scientific studies support that contention. This lack of research is no doubt partly due to the methodological issues associated with the measurement of secretion volumes in clinical studies, meriting further research to determine the best way to quantify removal of pulmonary secretions.\textsuperscript{17}

\textbf{Effect on Thinning Secretions}

Although clinicians often believe that instillation of normal saline “thins” thick pulmonary secretions, no research has ever shown that this belief is correct. In fact, experts in airway humidification long ago pointed out the fallacy of this notion, because small-particle humidification, not administration of a fluid bolus, is required to achieve any semblance of incorporation of fluid into thick secretions.\textsuperscript{18}\textsuperscript{[p504]} And even small-particle humidification falls short of actually “thinning” secretions noticeably. Experts\textsuperscript{19,20} recommend systemic hydration to decrease the viscosity of pulmonary secretions, because thick secretions reflect dehydration of mucous glands. The topical application of a 5- or 10-mL bolus of normal saline to thick mucus will not lead to incorporation of the saline into the mucus.\textsuperscript{21}

For clinicians who believe that normal saline thins secretions, try the following experiment to see for yourself what impact administration of a bolus of normal saline has on thick secretions.\textsuperscript{22} The next time you use suctioning, use a mucus trap to collect some of the thick secretions. Then, insert 5 to 10 mL of normal saline into the trap and observe how the saline remains separate from the mucus, even after vigorous shaking. Let the mixture sit a while to validate that even with exposure over time, the mucus and fluid remain separate from each other. If normal saline cannot thin thick secretions in a mucus trap with really vigorous shaking, it certainly cannot do it in a patient’s lungs.
Risks of Bacterial Contamination

In 2 studies,\textsuperscript{23,24} researchers reported that instillation of normal saline may place the patient at risk for hospital-acquired pneumonia. Rutula et al\textsuperscript{23} found that the rims of the individual-dose vials of normal saline were often contaminated with bacterial organisms just before insertion of the fluid into the endotracheal tube. On the basis of the type of bacterial organisms found on the rim, they hypothesized that the contamination of the vial had occurred when clinicians had “popped” the top off the vial with a thumb. Although the researchers\textsuperscript{23} did not evaluate infection of patients, introduction of bacterial organisms because of contamination during administration of the fluid is certainly theoretically possible.

In a laboratory study\textsuperscript{24} of endotracheal tubes that had recently been removed from patients in the intensive care unit (ICU), the amount of bacteria evacuated from the end of endotracheal tubes was 5 times greater when a bolus of normal saline was administered through the endotracheal tube before the suction catheter was introduced than when a suction catheter alone was passed through the endotracheal tube. The investigators\textsuperscript{24} hypothesized that a similar high load of bacterial contamination of the pulmonary system might occur when normal saline is instilled into the endotracheal tube during suctioning. The instillation of normal saline may act as a vehicle to “wash” the bacteria that normally cling to the inner aspects of the artificial airway into the lung, potentially leading to infection. Hagler and Traver\textsuperscript{24} did not evaluate clinical infection; however, they pointed out that instillation of saline before endotracheal suctioning may have some unintended outcomes.

Although the normal saline that is instilled should be sterile and without preservatives, isolated cases of outbreaks of bacterial pneumonia due to vials of normal saline contaminated during the manufacturing process have been reported.\textsuperscript{25,26}

Surveys of Nursing Practice

In several reports\textsuperscript{27-29} since 1996, researchers have described how often nurses and respiratory therapists instill normal saline before endotracheal suctioning. In most of the studies,\textsuperscript{27,29} 25% to 33% of nurses routinely or frequently instilled normal saline before suctioning. Twice as many respiratory therapists as nurses instilled normal saline.\textsuperscript{27,29} In a 1996 survey,\textsuperscript{28} pediatric critical care nurses almost universally instilled normal saline before doing suctioning. Most of the hospitals surveyed indicated that instillation of normal saline before endotracheal suctioning was included in the hospital’s policy/procedure for suctioning.\textsuperscript{7}

EBP Recommendations

The incidence of inadvertent placement of gastric or postpyloric tubes into the lungs, instead of the gastrointestinal system, with blind insertion at the bedside is not clearly known. Most of the information about inadvertent placement has come from case reports.\textsuperscript{35,36}

According to 2 research studies done to determine the sensitivity and specificity of capnography for detecting inadvertent pulmonary placement of gastric and postpyloric tubes, the incidence of pulmonary placement was 11% (11 of 100 attempts) when verified by chest radiography\textsuperscript{37} and 20% (4 of 20 attempts) when verified by carbon dioxide waveforms.\textsuperscript{38} Even if the actual clinical incidence is lower than observed in these limited studies, the complications associated with a feeding tube placed in the lung can be lethal; thus, a 100% effective method for verifying proper location of such tubes is needed.

Methods of Detecting Inadvertent Pulmonary Placement

A variety of methods have been advocated to detect when a gastric or postpyloric tube has been introduced into the pulmonary system:
Auscultation during air insufflation through the tube, pH testing of aspirated fluid, visual inspection of aspirated fluid, detection of carbon dioxide in the tube, and radiographic tube verification.  

Auscultation During Air Insufflation Through the Tube. Auscultation over the gastric abdominal area during rapid insufflation of air into the distal end of a gastrointestinal tube is commonly performed after a tube is inserted. Research on air insufflation has never documented that this technique is accurate for identifying inadvertent intubation of the lungs. Numerous case reports of documented inadvertent pulmonary intubation despite auscultation over the gastric area of air during insufflation, though, have been published.  

In the early 1990s, researchers found that air insufflation with auscultation over the gastric area could not be used to predict the inadvertent placement of a gastric tube into the lungs.  

Because of the proximity of the lungs and stomach, it is not surprising that the sounds created by air insufflation through the tube could easily be transmitted to adjacent areas, causing clinicians to err in determining proper tube placement.  

Testing the pH of Aspirated Fluid. Another technique that has been advocated over the years to identify inadvertent pulmonary intubation with gastrointestinal tubes is measuring the pH of fluids aspirated immediately after tube placement. If the fluid is alkaline, the gastric contents may be alkaline or the tube may be in the lung. Because of the lack of specificity of the pH technique and the numerous situations and conditions that lead to alkaline gastric contents, experts no longer advocate the use of pH testing to verify tube location.  

Visual Inspection of Aspirated Fluid. Visual inspection of the color of fluid aspirated from the tube has been advocated as a method to differentiate gastric fluid (green, dark yellow) from pulmonary fluid (white, light yellow). In the only study in which visual inspection of fluid was evaluated as a way of determining gastric or pulmonary location of the tube, visual inspection was a poor predictor of tube location. Similar to gastric pH, the colors of gastric and pulmonary secretions are altered by a variety of conditions, making development of a standard difficult.  

Presence of Carbon Dioxide in the Tube. Most recently, in several small studies, investigators evaluated the use of devices to measure the presence of carbon dioxide in the tube as a way to determine if the lungs have been inadvertently entered. Because carbon dioxide is present only in exhaled pulmonary gases and not in the gastric contents, this technique may be helpful in differentiating between the 2 locations. In studies in which end-tidal carbon dioxide monitors or disposable, color-indicator carbon dioxide devices were connected to the gastrointestinal tube during insertion, detection of carbon dioxide with the devices allowed successful detection of gastrointestinal tubes that had been placed in the lungs. In all but a single study, no instances of false identification of pulmonary placement were noted. The results of these studies show promise for finding a bedside technique that allows accurately detection of inappropriate pulmonary intubation. Because of the consequences of missing an incorrect placement of a gastric tube, additional studies are need to validate carbon dioxide detection techniques in larger and more diverse populations of patients and in a variety of clinical situations.  

Of particular interest is the ability of multiple caregivers to correctly interpret the color indications displayed by the disposable carbon dioxide device and to determine if fluid obstruction in the gastrointestinal tube and/or contamination

Table 2 Correct size of blood pressure cuff according to circumferences of patient’s arm

<table>
<thead>
<tr>
<th>Circumference, cm</th>
<th>Cuff size, cm</th>
</tr>
</thead>
<tbody>
<tr>
<td>22-26</td>
<td>Small adult, 12 x 22</td>
</tr>
<tr>
<td>27-34</td>
<td>Adult, 16 x 30</td>
</tr>
<tr>
<td>35-44</td>
<td>Large adult, 16 x 36</td>
</tr>
<tr>
<td>45-52</td>
<td>Adult–thigh, 16 x 42</td>
</tr>
</tbody>
</table>

a Based on data from Pickering et al.  
b Arm circumference is measured midway between acromion and olecranon.
of the carbon dioxide indicator affects the accuracy of the device.

**EBP Recommendations**

At this time, national guidelines and expert opinion indicate that the best method for confirming the location of blindly inserted gastrointestinal tubes is chest radiography. The radiopaque marker on each tube makes radiographic detection of inadvertent pulmonary placement clear, because the tube marker is easily seen by a radiologist in the right or left main bronchus, structures easily discerned on a chest radiograph.

Use of radiography to validate placement of small-bore gastrointestinal tubes is a clinically common policy in many facilities because inadvertent pulmonary intubations are thought to be more common with this type of tube. However, in a study by Burns et al., the incidence of pulmonary intubations did not differ between large- and small-bore gastric tubes. At this time, national guidelines recommend that proper placement of gastric tubes should be confirmed by radiographic means.

**Accurate Measurements of Blood Pressure**

In addition to the national guidelines for blood pressure measurement, a growing body of evidence supports specific procedural techniques that will improve the accuracy and reliability of noninvasive and invasive measurement of arterial blood pressure.

**How Do You Pick the Correct Cuff Size?**

The American Heart Association recommendations for correct sizes of blood pressure cuffs are summarized in Table 2. Selection of the appropriate cuff size is important because a cuff that is too small yields an overestimation of blood pressure and a cuff that is too large yields an underestimation of blood pressure.

**Does Arm Position Make a Difference for Noninvasive Measurement of Blood Pressure?**

As with intra-arterial blood pressure monitoring, the appropriate reference level for noninvasive measurement of blood pressure is the heart (Figure 1). Blood pressure will be overestimated if the arm is positioned above the heart and underestimated if the arm is positioned below the heart. Correct positioning of the arm is particularly important if the patient is sitting up or standing. If the arm is parallel to the patient or supported on the armrest, the systolic and diastolic blood pressures may be 10 mm Hg higher than if the arm is supported horizontally at heart level (level of the midsternum) and in patients with hypertension, the difference in arm position can cause an overestimation of systolic blood pressure 20 mm Hg. With the patient supine, the arm should be supported at the level of the phlebostatic axis (one-half the distance from the sternum to the back) rather than placed on the bed, a situation that causes an overestimation of systolic and diastolic blood pressures of 3 to 5 mm Hg. If a patient is in a lateral recumbent position, the noninvasive measurements of blood pressure taken from the “up arm” may be 13 to 17 mm Hg lower than measurements in supine patients, and blood pressure measurements from the “down arm” are either inconsistent or similar to measurements obtained with the patient supine.

**How Should Blood Pressure Be Measured in Obese Patients?**

Obesity alone does not affect the accuracy of blood pressure measurements. Blood pressure measured in the forearm can be
used if a correct cuff cannot be found; however, blood pressure in the forearm may be higher than blood pressure in the upper arm. For example, in a study of patients who were morbidly obese, only 19% had systolic and 28% had diastolic blood pressure measurements in the forearm within 10 mm Hg of the measurements in the upper arm.

The challenge with measuring blood pressure in patients who are morbidly obese is finding an appropriately sized cuff, although new cuffs are being developed that have long length but normal width. For every 5-cm increase in arm circumference, use of a standard cuff leads to an overestimation of systolic blood pressure by 3 to 5 mm Hg and diastolic blood pressure by 1 to 3 mm Hg compared with an appropriately sized large cuff. To size the cuff correctly, measure the circumference of the patient’s arm midway between the elbow and the wrist. Cuff size should be similar to that specified in the guidelines for upper arm circumference (Table 2). The cuff should be centered between the elbow and wrist, and the arm should be supported at the level of the heart.

Can You Use an Automatic (Oscillometric) Cuff to Measure Blood Pressure in Patients With Atrial Fibrillation?

No evidence-based guidelines are available for noninvasive measurement of blood pressure in patients with arrhythmias. Current recommendations based on the American Heart Association consensus49 for auscultated blood pressure in patients with arrhythmias are (1) measure the blood pressure 3 times and use the mean value, and (2) in patients with severe bradycardia, slow deflation of the cuff (target in bradycardia, 2 to 3 mm Hg per pulse) to prevent underestimation of systolic blood pressure and overestimation of diastolic blood pressure. A potential limitation of the use of oscillometric measurement of blood pressure in patients with marked arrhythmias is that with this method the maximal oscillation (mean arterial pressure) is detected and the systolic and diastolic blood pressures are estimated. In patients with atrial fibrillation or frequent ectopy, the beat-to-beat variability of stroke volume and the height of the oscillation may preclude the accurate measurement of the mean arterial pressure and thus the systolic and diastolic blood pressures. Conversely, auscultated systolic blood pressure may be overestimated or underestimated on the basis of selection of the first Korotkoff sound. In a comparison of 3 sets of auscultated and oscillometric measurements of blood pressure in patients with rate-controlled atrial fibrillation, the mean (standard deviation) measurements of blood pressure for each method were as follows: systolic, manual: 126 (18) mm Hg, oscillometric: 131 (12) mm Hg; diastolic, manual: 72 (15) mm Hg, oscillometric: 73 (15) mm Hg. These findings suggest that the methods are interchangeable. Because the algorithms for different oscillometric blood pressure machines vary, the results of a single study cannot be generalized to other monitors. If a patient is using an oscillometric cuff at home, the results should be validated by using auscultation. The accuracy of oscillometric measurements of blood pressure in patients with unstable atrial fibrillation has not been evaluated.

Should We Compare the Arterial Blood Pressure With the Cuff Pressure to Ensure That the Arterial Pressure Is Accurate?

The practice of using oscillometric brachial pressure to determine if an arterial pressure monitoring system is accurate and to decide whether to monitor the arterial pressure or the cuff pressure is not evidence based. The following factors should be considered when evaluating this practice. First, the aortic, brachial, and radial measurements of blood pressure are not the same. As a blood pressure wave moves into the peripheral vasculature, it is modified with an increase in systolic blood pressure and a decrease in diastolic blood pressure, whereas the mean arterial pressure is relatively unchanged. Generally, more central (aortic, femoral, brachial) measurements of systolic blood pressure are lower than radial measurements of systolic blood pressure by 7 to 14 mm Hg and are similar to or higher than diastolic blood pressure by 1 to 9 mm Hg, whereas the mean arterial pressure is unchanged. Second, the differences in systolic blood pressure change with aging (radial approximately the same as aortic systolic blood pressure), vasoconstriction (radial < brachial and femoral), and vasodilatation (femoral approximately the same as radial; aortic < radial). In addition to evaluating an absolute pressure, monitoring for trends or changes in blood pressure...
are (1) an increased emphasis on avoiding formation of microbubbles, including completely filling the drip chamber and using minimal pressure during initial flushing of the catheter, and (2) the use of the rocket flush (Never perform when catheter is in patient) to remove any hidden microbubbles. The rocket flush should never be performed when the catheter is in place in a patient because of the risk of retrograde air embolization. When this protocol (minus the fast flush) was used, 59% of pressure systems with a blood reservoir had adequate dynamic response characteristics and 41% were underdamped. The addition of the fast flush markedly improved the systems (92% adequate/optimal and 8% underdamped).88 Validated, evidence-based algorithms are also available to optimize a system once it is in use in a patient.

Table 3 Evidence-based protocol for preparation of an invasive pressure catheter

| 1. Cleanse hands |
| 2. Gather supplies (intravenous fluid, pressure monitoring kit, 10-mL syringe, and pressure bag) |
| 3. Prime pressure monitoring system to remove all air |
| a. Remove pressure monitoring kit from package, open blood salvage reservoir, tighten connections, close roller clamp, turn stopcock OFF to patient (off toward distal end), and remove vented stopcock cap |
| b. Invert bag of intravenous fluid and, using sterile technique, insert spike into it |
| c. Leave the spiked bag upside down, open roller clamp, and simultaneously activate fast-flush device continuously while gently squeezing to apply pressure to bag of intravenous fluids to slowly clear air from bag and drip chamber; completely fill the drip chamber with intravenous fluid |
| d. Turn bag upright once fluid is advanced sufficiently past the drip chamber |
| e. Apply gentle pressure (50 mm Hg) to the bag (or hang the bag about 30 inches above distal end of tubing) and activate fast-flush device, advance fluid, priming the stopcock |
| f. Orient the blood reservoir so that air will be completely removed by the advancing fluid (tilt distal end upright at 45°), and continue flushing to prime the entire catheter |
| g. Close reservoir and flush catheter to move any residual air bubbles from the reservoir |
| h. Perform rocket flush (Never perform when catheter is in patient) |
| (1) Turn stopcock off to distal end of catheter (“off to patient”) |
| (2) Attach 10-mL syringe to stopcock near the transducer by using sterile technique and slowly withdraw intravenous fluid to fill syringe |
| (3) Turn stopcock off to transducer |
| (4) Flush line quickly with 10 mL of normal saline from syringe to remove any remaining air bubbles |
| (5) Turn stopcock off |
| (6) Inspect catheter, remove any remaining air by fast flushing and rocket flush as needed |
| (7) Remove syringe and cap stopcock with closed cap by using sterile technique |
| 5. Place bag of fluid into a pressure bag and inflate bag to 250-300 mm Hg and check for air in catheter |
| 6. Evaluate dynamic response characteristics; goal: adequate or optimal |

4 Based on data from Bridges et al.88

What Steps Will Improve the Dynamic Response Characteristics of the Invasive Arterial Pressure Monitoring System?

Arterial pressure monitoring systems, particularly those with blood reservoirs, tend to be underdamped, which may lead to an overestimation of systolic pressure and an underestimation of diastolic pressure.86,87 A validated evidence-based protocol for preparation for an invasive catheter is presented in Table 3.88 Two points to note in this protocol are (1) an increased emphasis on avoiding formation of microbubbles, including completely filling the drip chamber and using minimal pressure during initial flushing of the catheter, and (2) the use of the rocket flush (ie, vigorously flushing the system with 10 mL of flush solution through the proximal port to remove any hidden microbubbles). The rocket flush should never be performed when the catheter is in place in a patient because of the risk of retrograde air embolization. When this protocol (minus the fast flush) was used, 59% of pressure systems with a blood reservoir had adequate dynamic response characteristics and 41% were underdamped. The addition of the fast flush markedly improved the systems (92% adequate/optimal and 8% underdamped).88 Validated, evidence-based algorithms are also available to optimize a system once it is in use in a patient.

Selection of Electrocardiographic Leads

Electrocardiographic (ECG) monitoring is performed for 3 primary reasons: detection of arrhythmia and conduction disturbance, monitoring of the ST segment, and monitoring of the QT interval.

Telemetry

Are 3-Lead Systems Equivalent to 5-Lead Systems for Monitoring Wide-Complex Tachycardia? For a 3-lead system, a modified chest lead (MCL-1 or MCL-6) should be used instead of lead II for the differential diagnosis of wide-complex tachycardia.91,92 Use of an MCL requires the following modifications in lead placement: right arm electrode on left shoulder,
left arm electrode at V1 position, left leg electrode at V6 position. After repositioning the leads, for MCL-1, select lead I, and for MCL-6, select lead II. However, the 3-lead system is not as accurate as 5-lead system for the differential diagnosis of aberrancy vs ectopy. The V1 criteria for the differential diagnosis of wide-complex tachycardia cannot be applied to MCL-1. For example, in a study by Drew and Scheinman, the QRS morphology in MCL-1 differed from that in V1 in 40% of cases (supraventricular tachycardia with aberrancy vs ventricular tachycardia), and use of MCL-1 resulted in 20% misdiagnosis compared with use of V1.

Which Leads Are Most Sensitive and Specific for Differentiating Ventricular Ectopy From Aberrancy? Leads V1 and V6 provide the most diagnostic clues for the differentiation of wide-complex tachycardia (Figure 2). Monitoring a lead (III or II) that allows evaluation of the relationship between the P wave and the QRS complex may also aid in the differential diagnosis (Table 4). Despite the utility of V1 for monitoring arrhythmia, it is not sensitive for monitoring the ST segment or QT interval.

**ST-Segment Monitoring**

What Is the Benefit of Continuous ST-Segment Monitoring? T-wave inversion may be a sign of myocardial ischemia; however, this change can be caused by factors other than ischemia. ST-segment depression of 0.5 mm or ST-segment elevation of greater than 1 to 2 mm indicates epicardial injury (Figure 3). Patients who have undergone percutaneous coronary intervention (PCI) or who have acute coronary syndrome may have periods of intermittent recurrent ischemia, which are often silent (ie, ST-segment changes indicative of ischemia or injury without angina or angina equivalents). For example, in a study of 11 532 hours of ST-segment monitoring in 250 patients, 55 (22%) had transient myocardial ischemia and of these, 55, 41 (75%) had silent ischemia. Similarly, in another study of 18 394 hours of 12-lead ST-segment monitoring in patients after an acute myocardial infarction or PCI, 463 ischemic events were detected and of these, 80% were silent. Silent ischemic episodes might be missed if ECG monitoring is intermittent.

![Figure 2](image1)  
**Figure 2** ST-segment deviation is defined as greater than 1- to 2-mm change in the ST segment from the patient’s baseline measured 0.06 seconds (60 ms) after the J point.  

![Figure 3](image2)  
**Figure 3** Measurement of QT interval.

### Table 4 Which leads?

<table>
<thead>
<tr>
<th>Monitoring purpose</th>
<th>Lead recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arrhythmia detection</td>
<td>3-lead system: MCL-1 (select lead I on monitor)</td>
</tr>
<tr>
<td>ST-segment monitoring</td>
<td>Right coronary artery: III or aVF</td>
</tr>
<tr>
<td>QTc</td>
<td>Identify 12-lead with most well-defined T wave: V6, V1, II</td>
</tr>
<tr>
<td>Axis deviation</td>
<td>I, aVF (detection of new onset/progrieving bundle branch block)</td>
</tr>
<tr>
<td>Best lead combinations</td>
<td></td>
</tr>
<tr>
<td>1 lead</td>
<td>V1 or V6 (MCL-1 or MCL-6)</td>
</tr>
</tbody>
</table>
| 2 lead | Arrhythmia: V1 and III  
| | ST segment: V5 and III  
| | Arrhythmia + ST segment: V1 or V6 + aVF or III |

Information from Kern et al.  
Information from Drew et al.
Should You Select the Bedside Lead on the Basis of the Patient’s ST-Segment Elevation “Fingerprint” During PCI?
The ST-segment “fingerprint” is the lead with the maximal ST-segment deviation (Figure 2) during an acute myocardial infarction or a PCI.\(^\text{101}\) The usefulness of the ST-segment fingerprint varies depending on the intent of the monitoring. The fingerprint is useful in detecting acute reocclusion after a PCI.\(^\text{100,101}\) However, Drew et al\(^\text{100}\) found that when the fingerprint was used, 53% of recurrent ischemic events were not detected. Of note, leads V1 and II did not show ST-segment deviation in 42% of cases. Additionally, ischemia may be detected in multiple leads for a given patient.\(^\text{102}\) If continuous multilead ST-segment monitoring is not available, the ECG leads that are most sensitive and specific for detecting ischemia should be used\(^\text{103,109}\) (Table 4).

**QT-Interval Monitoring**

**How Should the QT Interval Be Measured?** Most of the recommendations for QT-interval monitoring are based on expert opinion.\(^\text{95,102}\) The QT interval, which represents the duration of electrical activation (depolarization) and recovery (repolarization), is measured from the start of the QRS complex to the point where the T wave returns to the TP baseline (Figure 3). One suggestion to aid in identifying this point is to draw a tangent along the steepest part of the downslope of the T wave; the end of the QT interval is where this line intersects the TP baseline. If a U wave is present, the QT interval is measured from the onset of the QRS complex to the lowest point between the T and the U wave (Figure 4); however, if the U wave is large and merges with the T wave, it should be included in the measurement.\(^\text{103}\) Lead II may provide the best separation between the T and the U waves. If a biphasic T wave is present, the point of the final return of the T wave to baseline should be used. No consensus has been reached on how to measure the QT interval during atrial fibrillation. One suggestion is to take the QTc from the shortest and longest R-R intervals and average the 2 values.\(^\text{104}\) The same lead should be used for serial measurements.

Because the QT interval is inversely related to heart rate, it must be corrected or normalized to a heart rate of 60/min. The most commonly used formula to normalize the QT interval (QTc) is the Bazett formula,\(^\text{106}\) which is the QT divided by the square root of the preceding R-R interval in seconds:

\[
\text{QTc} = \frac{\text{QT}}{\sqrt{\text{R-R}}} 
\]

Debate is increasing about the use of the Bazett formula, because it results in an underestimation of the QTc at low heart rates and overestimations of it at high heart rates.\(^\text{106,107}\)

A normal QTc is less than 0.46 seconds in women and less than 0.45 seconds in men. An abnormal QTc for women is greater than 0.48 seconds and for men is greater than 0.47 seconds. A QTc greater than 0.5 seconds is considered an increased risk for torsades de pointes, although torsades de pointes may also develop in patients with a QTc less than 0.5 seconds.\(^\text{103}\) There is no QTc below which a patient is considered free of risk for arrhythmias.\(^\text{104}\)

**Can Bedside Monitoring Replace the 12-Lead ECG for the Diagnosis of Prolonged QTc?** 12-Lead ECG is the standard for the diagnosis of prolonged QT interval, and it cannot be replaced by bedside monitoring. The QT interval should be measured manually from the same lead, and the corrected value should be averaged over 3 to 5 beats.\(^\text{103}\) The QTc should be measured before the start of proarrhythmic therapy, at the time of the anticipated peak plasma level of the drug, after a change in drug dosage, and every 8 to 12 hours or more often if the QTc is prolonged.\(^\text{98,108}\)

Bedside monitoring may be useful in detecting changes in the QTc and determining if an additional 12-lead ECG should be obtained. In a study\(^\text{109}\) in which QTc measurements from a 12-lead ECG were compared with those from a bedside monitor (leads I/II), with a cutoff of 0.46 seconds, the monitor QTc agreed with the 12-lead ECG in 72%. However, in 26%, the QTc from the bedside monitor was greater than 0.46 seconds, whereas the 12-lead QTc was within normal limits; and in 2%, the 12-lead QTc was longer than 0.46 seconds, whereas the bedside monitor was within normal limits (bedside QTc sensitivity 50%, specificity 92%). This high specificity and low sensitivity means that episodes of prolonged QTc will not generally be missed when a bedside ECG is used; however, prolonged QTc may be overdiagnosed. The diagnosis of prolonged QTc made on the basis of values on the bedside
monitor should be confirmed with a 12-lead ECG.

**What Leads Should Be Used for QT-Segment Monitoring?** A 12-lead ECG should be used to determine which lead to choose for bedside monitoring of the QT interval. The lead with the most well-defined T wave (usually lead II) may have the clearest signal, particularly if a biphasic T wave or a U wave is present. On a 12-lead ECG, the anteroseptal leads generally have the longest QT, and in the study by Sadanaga et al, the leads with the highest sensitivity for detecting QT prolongation were V3 (94%), V4 (81%), II (66%), and V2 (63%).

**Does Lead Placement Really Make a Difference?** Attention to correct lead placement is imperative (Table 5). The most commonly misplaced leads are V1, V2, and V6. The displacement of V1 (from the fourth to the third intercostal space) can cause false ST-segment changes and morphological QRS changes that may lead to a misdiagnosis of myocardial infarction, right or left bundle branch block, or left ventricular hypertrophy. The recent American Heart Association guidelines also recommend that V5 and V6 be positioned parallel to V4 rather than in the fifth intercostal space (Table 5).

**Patients’ Position and Mobility**

“Teach us to live that we may dread unnecessary time in bed. Get people up and we may save our patients from an early grave.” In a 1947 article published in the *British Medical Journal*, Dr R. A. J. Asher made that statement. However, recognizing the science of positioning as a part of treatment in caring for acute and critically ill patients has taken a long time. For many years, nurses have recognized that positioning prevents skin breakdown, mobilizes secretions, and provides comfort. They have not, however, identified the effects that different types of positioning strategies have on pulmonary gas exchange, outcomes of weaning from ventilatory support, and prevention of deconditioning in survivors of intensive care.

The importance of positioning as a priority of practice is challenged in an environment based on high technology. In a study of positioning of critically ill patients during an 8-hour period, only 2.7% of patients had a change in position every 2 hours, and 49.5% never moved during an 8-hour period. Immobility is a problem, and the solution rests in increasing awareness of the importance of positioning on short- and long-term outcomes for patients.

**Impact of Immobility**

Immobility is a major factor in the development of atelectasis, ventilator-associated pneumonia (VAP), and functional limitations that linger long after a patient is discharged from the ICU and hospital. Most critical care patients spend most of their time supine, and supine positioning is an independent risk factor for mortality in patients receiving mechanical ventilation. Krishnagopalan et al found that during an 8-hour time frame, less than 3% of critically ill patients were turned every 2 hours (the standard). Close to 50% of patients during that same period had no change in body position. What effect does the stationary supine position have on lung physiology? Anzueto et al examined the impact of turning every 2 hours on the lungs of healthy adult baboons receiving mechanical ventilation. By study conclusion at 11 days, pathological examination of the lungs of baboons turned every 2 hours showed areas of bronchopneumonia. 5 of the 7 animals had surrounding bronchopneumonia.

**Mobility Strategies**

Positioning therapies have been targeted to meet specific pulmonary abnormalities. Researchers have shown conclusively that if a patient experiences a consolidated type pneumonia in one lung, then positioning with the good lung down will result in better oxygenation. Despite mechanical restriction in the downward position, the healthy lung has an adequate number of

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**Table 5** Correct position for precordial leads

<table>
<thead>
<tr>
<th>Lead</th>
<th>Placement</th>
</tr>
</thead>
<tbody>
<tr>
<td>V1</td>
<td>Fourth intercostal space, right sternal border</td>
</tr>
<tr>
<td>V2</td>
<td>Fourth intercostal space, left sternal border</td>
</tr>
<tr>
<td>V3</td>
<td>Halfway between V2 and V4</td>
</tr>
<tr>
<td>V4</td>
<td>Fifth intercostal space, left midclavicular line</td>
</tr>
<tr>
<td>V5</td>
<td>Halfway between V4 and V6 horizontal with V4 (at the anterior axillary line)</td>
</tr>
<tr>
<td>V6</td>
<td>Horizontal to V4 at the midaxillary line</td>
</tr>
</tbody>
</table>

a Based on data from Kligfield et al.

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http://ccn.aacnjournals.org
functioning alveoli to match gravity-dependent perfusion and thus promote effective gas exchange. For patients with bilateral lung disease, the best position is selected on the basis of the severity of the patient’s lung disease and critical illness. For many patients, turning every 2 hours is not enough to preserve the oxygenerating ability of the lungs or to prevent pneumonia.\(^{128,129}\) When the risk for complications of immobility are high, the use of rotational therapy is often considered.

Kinetic therapy/table-based rotation and continuous lateral rotation therapy reduce the incidence of VAP and atelectasis.\(^{129-135}\) The results of studies\(^{134,135}\) on the contribution of rotational therapy to reducing duration of ventilatory support and length of stay in the ICU are conflicting. In most studies, patients were rotated more than 18 hours a day to achieve maximum benefit and the therapy was started as early as possible. Researchers have not yet determined whether the degree or the frequency of rotation is the crucial factor. Ahrens et al\(^{134}\) randomized 234 medical-surgical trauma patients to receive rotation therapy or standard care and measured the impact on VAP, lobar atelectasis, and length of stay. Rotational therapy resulted in a significant reduction in the occurrence of VAP and lobar atelectasis but had no effect on length of stay.\(^{134}\)

Four systematic reviews\(^{129,134-138}\) of the literature on rotational therapy have indicated similar results. In the most recent review,\(^{138}\) regardless of the rotational degree achieved, the proportion of patients with VAP was significantly lower for the rotation groups than for the control groups \((P<.001; \text{Figure 5})\).

For patients with severe acute respiratory distress syndrome (ARDS), rotational therapy may not be sufficient to prevent complications and improve gas exchange. One strategy examined as a mechanism for recruiting alveoli and improving gas exchange in patients with ARDS is use of the prone position. In recent systematic reviews\(^{143-145}\)

<table>
<thead>
<tr>
<th>Study or subcategory</th>
<th>Proportion of patients with pneumonia</th>
<th>Odds ratio (fixed)</th>
<th>Weight, %</th>
<th>Odds ratio (fixed)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rotation</td>
<td>Control</td>
<td>95% CI</td>
<td></td>
</tr>
<tr>
<td>Pneumonia and prophylaxis</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Demarest et al(^{130})</td>
<td>1/16</td>
<td>4/14</td>
<td></td>
<td>3.45</td>
</tr>
<tr>
<td>Fink et al(^{131})</td>
<td>7/51</td>
<td>19/48</td>
<td></td>
<td>14.55</td>
</tr>
<tr>
<td>Gentilello et al(^{132})</td>
<td>5/27</td>
<td>13/38</td>
<td></td>
<td>7.58</td>
</tr>
<tr>
<td>Kelley et al(^{133})</td>
<td>5/18</td>
<td>13/25</td>
<td></td>
<td>6.77</td>
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<tr>
<td>Kirschenbaum et al(^{134})</td>
<td>3/17</td>
<td>10/20</td>
<td></td>
<td>6.52</td>
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<tr>
<td>Summer et al(^{135})</td>
<td>4/41</td>
<td>7/42</td>
<td></td>
<td>5.38</td>
</tr>
<tr>
<td>Traver et al(^{136})</td>
<td>8/44</td>
<td>17/59</td>
<td></td>
<td>10.24</td>
</tr>
<tr>
<td>Whiteman et al(^{137})</td>
<td>10/33</td>
<td>14/36</td>
<td></td>
<td>8.04</td>
</tr>
<tr>
<td>deBoisblanc et al(^{138})</td>
<td>6/69</td>
<td>11/51</td>
<td></td>
<td>9.95</td>
</tr>
<tr>
<td>Subtotal (95% CI)</td>
<td>49/316</td>
<td>108/333</td>
<td></td>
<td>72.49</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Study or subcategory</th>
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<th>Odds ratio (fixed)</th>
<th>Weight, %</th>
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<tbody>
<tr>
<td></td>
<td>Rotation</td>
<td>Control</td>
<td>95% CI</td>
<td></td>
</tr>
<tr>
<td>Pneumonia treatment</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ahrens et al(^{134})</td>
<td>14/97</td>
<td>45/137</td>
<td></td>
<td>27.51</td>
</tr>
<tr>
<td>Subtotal (95% CI)</td>
<td>14/97</td>
<td>45/137</td>
<td></td>
<td>27.51</td>
</tr>
<tr>
<td>Total (95% CI)</td>
<td>63/413</td>
<td>153/470</td>
<td></td>
<td>100.00</td>
</tr>
</tbody>
</table>

**Figure 5** Meta-analysis of pneumonia (with subgroups of prophylaxis and treatment for respiratory dysfunction): rotation versus control.

Abbreviation: CI, confidence interval.

Reprinted from Goldhill et al,\(^{138}\) with permission.
of the literature on prone positioning, more than 70% of all ARDS patients studied responded to prone positioning with a 20% increase in $\text{Pao}_2$ or an increase greater than 20 in the ratio of $\text{Pao}_2$ to fraction of inspired oxygen within 2 hours of the turn. Gattinoni et al\textsuperscript{146} and Guerin et al\textsuperscript{147} published the anticipated definitive

**Figure 6** Suggested algorithm for positioning critically ill patients.

Reprinted from Ahrens et al,\textsuperscript{150} with permission from ADVANCING NURSING LLC.
outcomes research for the use of prone positioning in patients with ARDS/acute lung injury. The results were less than promising, indicating no improvement in mortality in a diverse critically ill population. These negative outcomes may have been influenced by a number of methodological challenges. In both studies, the average range of tidal volume was greater than 9 mL/kg in both groups, and as indicated in the landmark ARDS Network ventilator study, tidal volumes greater than 6 mL/kg may contribute to ventilator-induced lung injury. A phase 3 trial of prone positioning with patients receiving 6 mL/kg tidal volume and positioned prone more than 18 hours a day is under way.

**Progressive Mobility: Combating Deconditioning**

Once a patient’s hemodynamic status allows forms of mobilization, every attempt should be made to progressively mobilize the patient to dangle the legs, sit in a chair, bear weight, and walk to decrease the severe muscle wasting that occurs in critically ill patients. Hemodynamic instability is due to spending prolonged periods in a stationary position or the establishment of a “gravitational equilibrium.” The physical deconditioning and challenges with hemodynamic instability that occur with bed rest can be dealt with by using a stepwise mobility progression program (Figure 6). Once a patient’s cardiovascular system is stable when the head of bed is higher than 30°, a progressive mobility program can be started. The goal is to progress in a stepwise fashion by increasing the height of the head of the bed, followed by placing the legs in a dependent position. If this change is tolerated, dangling of the legs and then weight bearing should begin as soon as possible. The next step is supported ambulation. The mobility program can be performed safely while the patient is intubated and receiving mechanical ventilation. One group who used the bed-chair position 3 times a day for patients who met the criteria for mobilization reported a decrease in ICU length of stay and occurrence of VAP.

Methods and equipment that support stepwise mobility progression are essential to meet the physiological demands of the healing process. Having the methods and equipment will allow nurses to do what the evidence indicates is the right thing with minimal use of manpower. Mobility is a fundamental nursing activity that requires in-depth knowledge and skill for effective use in critically ill patients. As a core component of care, mobility helps in managing secretions, reducing feelings of powerlessness, preventing muscle wasting, improving gas exchange, and decreasing the incidence of atelectasis and VAP. Matching the right time of the illness to the right positioning strategy will help achieve good outcomes for patients. Nurses have a unique opportunity to affect patients outcomes noninvasively through the independent activity of mobility.

**EBP Recommendations**

EBP practice recommendations for positioning are as follows:

- Progressive mobilization to dangling legs, standing, and walking are safe for intubated patients.
- Patients breathe better and experience improved oxygenation with higher elevations of the head of the bed if their hemodynamic status is such that they can tolerate the elevation.
- For many critically ill patients, turning every 2 hours is not enough to preserve the oxygenating ability of the lungs or to prevent health care–acquired pneumonia.
- Kinetic and continuous lateral rotation therapy reduces the risk of VAP in patients receiving mechanical ventilation. Optimal benefit depends on early placement and more than 18 hours of rotation per day. Research has not yet determined whether the degree or the frequency of rotation is the crucial factor.
- Prone positioning improves oxygenation but has not yet been shown to affect mortality.
- Use of the prone position should be considered after conventional strategies for lung recruitment have been tried unsuccessfully.

**The Glasgow Coma Scale in Neurological Assessment**

For decades, the level of consciousness has been deservedly described as the most sensitive and the earliest indicator of progression in intracranial abnormalities such as intracranial hypertension. Since its introduction in 1974, the Glasgow Coma Scale (GCS) has been used in many clinical areas to assess and document consciousness and responsiveness. The GCS is used to assign a numerical value to a set of responses in 3 spheres: eye opening,
motor responses, and verbal responses (Table 6).

**Limitations of the GCS in Neurological Evaluation**

The GCS was initially intended to standardize patients’ assessment and improve communication about neurological status. The GCS is widely used but does have some important drawbacks and limitations (Table 7). Patients with specific clinical states such as locked-in syndrome, catatonia, and psychogenic coma may have a GCS score indicating depression of consciousness and responsiveness. These patients may actually have a higher degree of brain responsiveness than initially estimated solely on the basis of GCS assessment.

In locked-in syndrome, patients are awake and can follow commands such as select eye movements. Additional skeletal muscle movement is not possible because of paralysis below the third cranial nerve. In catatonic states, patients may appear unresponsive, but electroencephalographic evaluation indicates low-amplitude, high-frequency activity rather than slow-wave, high-amplitude activity associated with unresponsive states due to structural or metabolic brain injury. In psychogenic coma, electroencephalographic evaluation indicates an awake rhythm.

Even with these limitations, the GCS is used extensively, and the GCS score is incorporated into many critical care documentation records, trauma and emergency medicine documentation systems, and other clinical scoring systems. Additional limitations exist in each of the 3
appropriate to preserve the option of organ donation for family members.153 GCS scores determined in the prehospital setting and after arrival in the acute care setting correlate closely and are predictive of outcome as well as potential need for neurosurgical intervention.161 A GCS score of 3 in a patient with reactive pupils is predictive of potentially better outcomes and suggests that the patient might benefit from aggressive resuscitation.162

Research and Alternatives to the GCS
Because of the limitations of the GCS, research into alternative neurological assessment tools is ongoing. One tool, the Full Outline of UnResponsiveness (Table 9), has been studied in multiple clinical settings by members of several disciplines, including critical care/neuroscience nurses, neurology residents, and neurointensivists.163 The tool is easy to use and has good interrater

<table>
<thead>
<tr>
<th>Table 8 Clinical uses of the Glasgow Coma Scalea</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rapid assessment, documentation and communication of neurological status</td>
</tr>
<tr>
<td>Guidance for therapeutic decisions and timing of invasive procedures such as monitoring of intracranial pressure and ventricular drainage</td>
</tr>
<tr>
<td>Assessing progression of brain injury or neurological improvement over time</td>
</tr>
<tr>
<td>Determining baseline neurological status as a starting point for therapeutic interventions</td>
</tr>
<tr>
<td>Assessment of neurological status after ingestion of a toxin</td>
</tr>
<tr>
<td>Predictive value regarding clinical and neurological outcome after brain injury</td>
</tr>
</tbody>
</table>

a Based on data from Edwards,158 Heard and Bembart,159 Tien et al,160 and Davis et al.161

<table>
<thead>
<tr>
<th>Table 9 Criteria used to determine scores on the Full Outline of UnResponsivenessa</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eye response</td>
</tr>
<tr>
<td>4 = Eyelids open or opened, tracking, or blinking on command</td>
</tr>
<tr>
<td>3 = Eyelids open but not tracking</td>
</tr>
<tr>
<td>2 = Eyelids closed but open to loud voice</td>
</tr>
<tr>
<td>1 = Eyelids closed but open to pain</td>
</tr>
<tr>
<td>0 = Eyelids remain closed with pain</td>
</tr>
<tr>
<td>Motor response</td>
</tr>
<tr>
<td>4 = Thumbs-up, fist, or peace sign</td>
</tr>
<tr>
<td>3 = Localizing to pain</td>
</tr>
<tr>
<td>2 = Flexion response to pain</td>
</tr>
<tr>
<td>1 = Extension response to pain</td>
</tr>
<tr>
<td>0 = No response to pain or generalized myoclonus status</td>
</tr>
<tr>
<td>Brain stem reflexes</td>
</tr>
<tr>
<td>4 = Pupil and corneal reflexes present</td>
</tr>
<tr>
<td>3 = One pupil wide and fixed</td>
</tr>
<tr>
<td>2 = Pupil or corneal reflexes absent</td>
</tr>
<tr>
<td>1 = Pupil and corneal reflexes absent</td>
</tr>
<tr>
<td>0 = Absent pupil, corneal, and cough reflexes</td>
</tr>
<tr>
<td>Respirations</td>
</tr>
<tr>
<td>4 = Not intubated, regular breathing pattern</td>
</tr>
<tr>
<td>3 = Not intubated, Cheyne-Stokes breathing pattern</td>
</tr>
<tr>
<td>2 = Not intubated, irregular breathing</td>
</tr>
<tr>
<td>1 = Respirations greater than ventilator rate</td>
</tr>
<tr>
<td>0 = Respirations at ventilator rate or apnea</td>
</tr>
</tbody>
</table>

a Based on data from Lieberman et al,162 and Wijdicks et al.163

Spheres assessed by the GCS (Table 7). In patients with these limitations, the GCS score may provide inaccurate data on consciousness, motor function, and arousal.

The effectiveness of the GCS depends on the ability of a patient to respond and interact with a clinician. Optimal neurological assessment will indicate clinical states that interfere with and limit the efficacy of the GCS. Deep sedation/analgesia produces a drug-induced depression of consciousness, arousal, and cognitive ability, making the GCS ineffective. In this setting, a sedation assessment tool may be highly appropriate. Neuromuscular blockade, in a dose-related manner, produces skeletal muscle relaxation in which a patient may potentially be awake but appears to be poorly responsive solely because of drug effects on neuromuscular transmission, not brain function. In each instance, optimal evaluation of the central nervous system is facilitated by using electrophysiological monitoring.

Predictive Value of GCS Scores
In clinical practice, the GCS score is used for multiple purposes, including guiding therapeutic decisions, predicting outcomes, and evaluating patients after they have ingested a toxin155 (Table 8).

Available research supports the predictive value of GCS scores for clinical outcomes, particularly when the scores are used in context with other neurological data. Patients with a GCS score of 3 and fixed, dilated pupils after brain trauma have no reasonable chance for survival.160 Aggressive resuscitation and physiological support may be
agreement between experienced and novice nurses from the neuroscience ICU and other nurses.\textsuperscript{164} Moreover, it adds brain stem and respiratory assessment and provides additional information beyond that provided by the GCS.\textsuperscript{163,164} It can assist in detecting disorders such as uncal herniation and locked-in syndrome\textsuperscript{166} and in predicting in-hospital mortality.\textsuperscript{163,164}

**EBP Recommendations**

Best clinical practice for neurological assessment includes optimal and consistent use of the GCS plus inclusion of other neurological data such as assessment of brain stem reflexes; eye examination, including pupil reactivity and extraocular movement; vital signs; and respiratory rate, depth, and pattern. Best practice also demands that the neurological evaluation include consideration of clinical state, concurrent injury, and drug therapy. A neurological evaluation compromised by depression of consciousness or concurrent drug therapy should be supplemented by neuroimaging\textsuperscript{165} or neurophysiological (electroencephalographic)\textsuperscript{166,167} evaluation.

**Management of Intracranial Hypertension**

Intracranial pressure is the total pressure produced within the skull by cerebrospinal fluid, blood, and brain.\textsuperscript{153,168-170} In order to maintain stable intracranial pressure, an increase in volume of one component must be balanced by a decrease in volume of one or both of the other components.\textsuperscript{153,168,169} Selective manipulation of these components is a mainstay of therapy for intracranial hypertension, and in attempts to improve patients’ outcomes, each component is the subject of ongoing research.

**Modulation of Volume of Cerebrospinal Fluid: Intraventricular Drain**

Drainage of cerebrospinal fluid is a hallmark of aggressive management of intracranial hypertension and is indicated for sustained elevations of intracranial pressure greater than 20 mm Hg.\textsuperscript{171} Further indications include depressed level of consciousness such as a GCS score of 8 or lower.\textsuperscript{170,172} Drainage of cerebrospinal fluid improves management of intracranial pressure and cerebral perfusion pressure (CPP) as well as clinical and neurological outcomes, particularly for younger patients in whom coordinated, mechanism-based management is used, with interventions tailored more specifically to the underlying pathophysiological changes.\textsuperscript{173} An example is drainage of cerebrospinal fluid in the management of hydrocephalus due to subarachnoid hemorrhage. Drainage of cerebrospinal fluid will remain a mainstay of therapy after traumatic brain injury\textsuperscript{174} and after other clinical states such as hydrocephalus after subarachnoid hemorrhage. Such drainage may have predictable effects in decreasing intracranial pressure and increasing CPP when done in a controlled, protocol-directed manner.\textsuperscript{174} In patients with intracranial hypertension, initially refractory elevations of intracranial pressure, drainage of cerebrospinal fluid was effective and was associated with improved functional outcome and lower mortality 6 months after injury.\textsuperscript{175} Drainage of cerebrospinal fluid also is not associated with marked risks to other body systems as is drug-induced coma or hypothermia.\textsuperscript{176} Available evidence strongly supports drainage of cerebrospinal fluid as an effective monitoring and therapeutic technique.

**Modulation of Brain Volume: Mannitol and Hypertonic Saline**

Modulating brain volume (80% of intracranial volume) is a focus for aggressive intervention. Osmotherapy with an agent such as mannitol to reduce brain volume works by 2 mechanisms. First, agents such as mannitol produce an osmotic gradient that draws water out from otherwise swollen brain tissue. Second, agents such as mannitol reduce blood viscosity and hematocrit and augment cerebral blood flow.\textsuperscript{168,170,176-178}

Mannitol has been used extensively as an osmotic diuretic for many years.\textsuperscript{168} It can be administered as a bolus or as an infusion.\textsuperscript{168,170} Recent studies\textsuperscript{170,177} suggest earlier use of high-dose mannitol (eg, 1.4 g/kg) may be more effective than standard-dose therapy in improving intracranial pressure and outcomes. Bolus dosing of mannitol is generally more effective than continuous infusion.\textsuperscript{168} Limitations of mannitol include hyperosmolality and volume loss from osmotic diuresis. Also, with longer duration such as several days of therapy, rebound elevation of intracranial pressure may occur.\textsuperscript{178}

Hypertonic saline, an osmotic agent with a concentration of sodium chloride that exceeds that of physiological saline (0.9%),\textsuperscript{178,179} has more recently been studied and used to manage intracranial hypertension. Concentrations of hypertonic saline used to manage intracranial hypertension include 2%, 3%, 5%, 7%,
In addition to reducing intracranial pressure, hypertonic saline augments hemodynamic stability and intravascular volume. In some studies and some patients, hypertonic saline has been more effective than mannitol for treatment of elevated intracranial pressure. When mannitol and hypertonic saline were compared in patients who had brain swelling and intracranial hypertension after ischemic stroke, hypertonic saline was more effective in reducing intracranial pressure and supporting CPP. Hypertonic saline is also effective in managing intracranial pressure in patients refractory to mannitol, including patients who have had brain trauma and ischemic stroke. Optimal concentration, volume, bolus vs infusion dosing, and timing/duration of therapy with hypertonic saline and targeted clinical state have not yet been determined. Hypertonic saline may be most effective with patient-specific titration of therapy, including volume, dosing interval, and concentration targeted to specific clinical goals, including CPP, intracranial pressure, and other monitored parameters.

**Metabolic Suppression: Therapeutic Hypothermia**

Therapeutic hypothermia is the controlled depression of body temperature to 36°C or lower. Goals of therapeutic hypothermia include controlling refractory elevations of intracranial pressure and modulating effects of secondary brain injury. Multiple factors are associated with secondary brain injury, including release of excitatory neurotransmitters, calcium release, hyperemia, inflammatory response, brain edema, and intracranial hypertension. Many of these consequences are temperature dependent and potential targets for therapeutic hypothermia. Therapeutic hypothermia improves neurological outcomes after cardiac arrest. Therapeutic hypothermia is also effective in controlling dangerous refractory elevations of intracranial pressure.

In patients with hepatic failure, neurophysiological changes such as brain edema, cerebral hyperemia, loss of autoregulation, and intracranial hypertension are, among others, risk factors for poor clinical and neurological outcomes. Of particular concern are elevations in intracranial pressure exceeding 30 to 50 mm Hg, which are associated with severe hepatic failure. Mortality due to intracranial hypertension in patients with acute liver failure is approximately 20%. Mild to moderate therapeutic hypothermia with core temperature approximately 32°C to 34°C is safe and effective for controlling elevations in intracranial pressure refractory to other therapies in the ICU immediately before and during liver transplantation.

One review of multiple studies concluded that therapeutic hypothermia after traumatic brain injury was effective in reducing intracranial pressure and may reduce risks of mortality and poor neurological outcome. Multiple aspects of therapeutic hypothermia have been researched, such as duration of therapy. Long-term therapy (5 days vs 2 days) was associated with improved outcomes such as control of intracranial pressure. In children, preliminary data from a study of 48 patients suggested that therapeutic hypothermia is most likely safe, effective for control of intracranial pressure, and associated with a potential trend toward improved functional outcomes 3 to 6 months after injury. In a study of patients with severe head injury, patients treated with hypothermia had significantly higher CPP than did patients in the normothermic and hyperthermic subgroups. In another investigation, optimal body temperature for reducing intracranial hypertension in patients with severe brain injury was between 35.0°C and 35.5°C.

Therapeutic hypothermia potentially can have marked effects on multiple body systems. Risks include coagulopathy, cardiovascular instability, and increased risk of infection. Optimal use of therapeutic hypothermia may ultimately be best when titrated as a patient-specific and mechanism-based therapy to desired core and brain temperature. Best practices for duration of therapy, rate of temperature decrease and rewarming, and target temperature as well as optimal selection of patients are not yet determined. Therapeutic hypothermia may be used as an option on an individual basis for refractory intracranial hypertension. The currently available evidence does not support routine use of therapeutic hypothermia after traumatic brain injury.

**Modulating Cerebral Blood Volume: Controlled Hyperventilation**

Decreasing arterial carbon dioxide levels via controlled hyperventilation has long been used to control intracranial pressure by reducing cerebral blood flow. Hyperventilation...
reduces elevated intracranial pressure but risks ischemic injury. Reduction in intracranial pressure is also transient for a given degree of hypocapnia. Because of the risks of brain ischemia, particularly during the first 24 hours after brain trauma, when cerebral blood flow is already compromised, prolonged hyperventilation (PaCO₂ 25-30 mm Hg) for more than a few hours pending optimal use of definitive therapy for control of intracranial pressure may cause global or localized cerebral ischemia. Reductions in cerebral blood flow may last longer than reductions in intracranial pressure during controlled hyperventilation. Effects of controlled hypocapnia in reducing intracranial pressure are well established, but the risks associated with this treatment include brain ischemia and poor outcomes. Because of its effects on cerebral hemodynamics, blood flow, and ischemic risk, long-term use of hyperventilation is not supported by the available evidence. The use of hyperventilation in patients with traumatic brain injury best supported by the evidence is in management of acute elevations in intracranial pressure pending aggressive use of definitive therapies specific to the cause of the elevation, such as optimal use of osmotic or metabolic suppression therapies. Longer term application of hyperventilation may have a role when cerebral metabolic parameters such as brain tissue oxygenation are monitored, permitting real-time titration of therapy to a patientspecific metabolic state.

**Summary**

Many of the studies used to develop the EBP recommendations described here were nursing research. Nurses asked questions about practice and, using the scientific process, undertook the challenge to find the answers. Once evidence is discovered, it is left to nurses at the bedside to implement the appropriate change. Sometimes the change actually makes practice easier. For example, not icing cardiac output solution was easy to implement and saved nursing time but it took almost a decade before it became common practice. It is estimated that 30% to 40% of patients do not receive care consistent with the current scientific evidence. In a self-review of use of EBP in their unit during a 1-year period, Ilan et al found that they implemented their own approved protocols only 50% of the time. Ironically, they discovered that it was the “sickest patients” who were least likely to receive commonly recommended best practices. Pravikoff et al examined nurses’ perceptions about readiness for EBP implementation and found that, after lack of time, the second highest barrier was a “lack of value for research in practice.” Larrabee et al found that a nurse’s “attitude” about research was a key factor as to whether the nurse was likely to be supportive of EBP changes. Plost and Nelson implemented 9 EBP protocols in their 35-bed ICU, and after 3 years, they found that the use of protocols simplified processes, standardized care, facilitated patients’ safety, and reduced costs.

Two major challenges are before us. We must continue to answer clinical questions with research, and we must implement the EBP recommendations that will assist us in providing best practice. As noted by Titler et al, “Although education is necessary to change practice, alone it is not sufficient.” If it took only posting an article in the bathroom or a poster in the back room to change practice, this article would not have been necessary. It will take the dedication of advanced practice and bedside nurses to evaluate their own practice and the needs of their patients and a continued vigilance to ask, Are we doing what is best for our patients with the current evidence available to us?

“The most cost-effective opportunity to improve the quality of care will not come from discovering new therapies, but from discovering how to deliver therapies that are known to be effective.”

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Now that you’ve read the article, create or contribute to an online discussion about this topic using eLetters. Just visit http://ccn.aacnjournals.org and click “Respond to This Article” in either the full-text or PDF view of the article.

**Financial Disclosures**
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CCN Fast Facts

Seven Evidence-Based Practice Habits: Putting Some Sacred Cows Out to Pasture

Facts

According to estimates, 30% to 40% of patients do not receive care consistent with current scientific evidence. Advanced practice and bedside nurses must evaluate their own practice and the needs of their patients and ask, Are we doing what is best for our patients with the current evidence available to us?

Instillation of Normal Saline Before Endotracheal Suctioning: Helpful or Harmful?

• Do not instill normal saline (physiological salt solution) before endotracheal suctioning.

Best Way to Verify Proper Placement of Nasogastric and Pyloric Tubes

• Use chest radiography to confirm correct placement of nasogastric tubes.

Accurate Measurements of Blood Pressure

• For accurate noninvasive measurement of blood pressure, choose the correct size cuff and position the patient’s arm at the level of the heart. Validated evidence-based algorithms are available to optimize use of invasive arterial pressure monitoring systems.

Selection of Electrocardiographic Leads

• Attention to correct placement of electrocardiography leads is imperative. Wide-complex tachycardia is best assessed with leads V1 and V6.

Mobility

• Match the right time of illness to the right positioning and mobility strategy.

• If a patient experiences consolidated pneumonia in one lung, positioning with the good lung down will result in better oxygenation.

• Progressive mobilization to dangling legs, standing, and walking is safe for intubated patients.

• Patients breathe better and experience improved oxygenation with higher elevations of the head of the bed if their hemodynamic status is such that they can tolerate the elevation.

• Turning critically ill patients every 2 hours may not be enough to preserve the oxygenating ability of the lungs or to prevent healthcare-acquired pneumonia.

• Kinetic and continuous lateral rotation therapy reduces the risk of ventilator-associated pneumonia in patients receiving mechanical ventilation. Optimal benefit depends on early placement and more than 18 hours of rotation per day.

The Glasgow Coma Scale in Neurological Assessment

• For neurological assessment, use the Glasgow Coma Scale in combination with other evaluation of brain stem reflexes; eye examination, including pupil reactivity and extraocular movement; vital signs; and evaluation of respiratory rate, depth, and pattern. Neurological evaluation compromised by depression of consciousness or concurrent drug therapy should be supplemented by neuroimaging or neurophysiological (electroencephalographic) evaluation.

Management of Intracranial Hypertension

• To manage intracranial hypertension, balance an increase in the volume of any 1 of the 3 components of total intracranial pressure (cerebrospinal fluid, blood, brain) by a decrease in the volume of the other 2 components.

• Drainage of cerebrospinal fluid is indicated for sustained elevations of intracranial pressure greater than 20 mm Hg and in patients with a score of 8 or lower on the Glasgow Coma Scale.

• In some patients, hypertonic saline is more effective than mannitol in treating intracranial hypertension.

• Currently available evidence does not support routine use of therapeutic hypothermia after traumatic brain injury.

• Long-term use of controlled hyperventilation to modulate cerebral blood volume is not supported by the available evidence.


This article and an online version of the CE test may be found online at http://ccn.aacnjournals.org.