GASTRIC TONOMETRY AND ENTERAL NUTRITION: A POSSIBLE CONFLICT IN CRITICAL CARE NURSING PRACTICE

By Andrea P. Marshall, RN, IC Cert, BN, MN (Research), and Sandra H. West, RM, CM, IntCareCert, BSc, PhD. From Department of Clinical Nursing, Faculty of Nursing, University of Sydney (APM, SHW), and Department of Critical Care, Manly Hospital, Manly, NSW, Australia (APM).

Background
Gastric tonometry is used to assess gastrointestinal mucosal perfusion in critically ill patients. However, enteral feeding is withheld during monitoring with gastric tonometry because enteral feeding is thought to influence tonometric measurements.

Objectives
To examine the effect of enteral feeding on the tonometric measurement of gastric mucosal carbon dioxide.

Methods
Gastric tonometers were placed in 20 critically ill patients, and the $P_{CO_2}$ of the gastric mucosa was measured in both the full and the empty stomach during a 48-hour period.

Results
The $P_{CO_2}$ measured by the tonometer increased after enteral feeding, and a significant difference in the $P_{CO_2}$ of the full versus the empty stomach was evident at 24 and 48 hours. $P_{CO_2}$ at 4, 24, and 48 hours differed significantly in the full stomach and in the empty stomach. However, the data did not reveal a significant difference in either the full stomach or the empty stomach between $P_{CO_2}$ at 24 hours and $P_{CO_2}$ at 48 hours.

Conclusion
After 24 hours of feeding, the initial increase in $P_{CO_2}$ observed at 4 hours was not evident, suggesting stabilization of the intragastric environment. However, a higher $P_{CO_2}$ was evident in the empty stomach, indicating that the presence of the feeding solution may reduce the diffusion of carbon dioxide into the tonometer balloon. Consequently, measurements of intragastric $P_{CO_2}$ obtained after 24 hours of feeding may be reliable if the stomach is emptied by aspiration via the tonometer immediately before measurement. (American Journal of Critical Care. 2003;12:349-356)

Critical illness can often be accompanied by alterations in gastrointestinal structure and function. The digestive functions of the gastrointestinal system are well understood, but the ability of this system to provide a protective barrier against microorganisms is of increasing interest because of the identification of links between gastrointestinal dysfunction and the development of multiple organ failure. Consequently, the ability to assess and maintain gastrointestinal function during episodes of critical illness has become an important aspect of critical care practice.

Enteral nutrition is the preferred method of nutritional support in critical illness. This method of nutritional support can provide nutrients, maintain the gastrointestinal mucosal barrier and immunological function, and improve blood flow in the gastrointestinal mucosa. Maintaining these normal physiological processes reduces bacterial translocation across the gastrointestinal wall and perhaps prevents the development of multiple organ failure.

Early enteral nutrition in critically ill patients has been widely advocated on the basis of these physiological benefits. Although provision of enteral nutrition promotes gastrointestinal function, early detection and management of gastrointestinal hypoperfusion may prevent gastrointestinal dysfunction and the development of multiple organ failure. Currently, gastric tonometry is...
Gastric tonometry is the only clinically available method for assessing gastrointestinal mucosal perfusion and oxygenation. Gastric tonometers are similar to traditional nasogastric tubes. Measurement of CO2 requires the sampling of fluid or air from a CO2-permeable balloon attached to the gastric tonometer. Theoretically, the CO2 of the mucosa should move from an area of higher partial pressure (the mucosa) to an area of lower partial pressure (the gastric lumen) until equilibrium is reached. Because the tonometer balloon is permeable to CO2, the gas continues to diffuse from the lumen and into the balloon, again until equilibrium is reached. Monitoring CO2 levels in the gastrointestinal mucosa can provide valuable information about regional perfusion. That is, when gastrointestinal mucosal perfusion is reduced, the time for diffusion is greater and thus the CO2 levels increase. Because few studies on gastric tonometry have been done in healthy adult patients, no values are widely accepted as normal for gastrointestinal CO2 levels measured by using a tonometer (Pt CO2). However, it is generally accepted that regional CO2 levels are marginally higher than those measured in arterial blood. For clinical evaluation, the difference between arterial and gastrointestinal mucosal levels of CO2 (CO2 gap) is often calculated; a difference of up to 10 mm Hg is considered acceptable.

Despite the ability to monitor gastrointestinal mucosal perfusion, monitoring and enteral feeding are not done concurrently during critical illness because it is thought that enteral nutritional solutions will influence the measurement of PtCO2. With the exception of small-bowel feeding, the withholding of enteral nutrition for a minimum of 1 hour is currently recommended for patients monitored via gastric tonometry. In many instances, this practice has led to delayed or inadequate provision of nutritional support.

**Methods**

The study was conducted during a 12-month period in a 9-bed general intensive care unit. Approval for the study was granted by the appropriate institutional review board, and the investigation was carried out in accordance with the ethical standards set forth in the Helsinki Declaration of 2000. For each patient, the study began after consent was obtained from the patient’s next of kin.

**Design and Sample**

An interrupted time series design was used to examine the influence of enteral feeding on the measurement of gastric mucosal CO2 levels. Patients were eligible for the study if they were between the ages of 18 and 75 years, required enteral nutrition via a naso-
gastric tube, and were being monitored with an intraarterial catheter. On the basis of these selection criteria, a convenience sample was acquired, and 20 critically ill patients who required nasogastric feeding with Jevity enteral feeding solution (Abbott Laboratories, Abbott Park, Ill) were enrolled in the study.

Data Collection
A TRIP NGS catheter (Tonometrics Division, Instrumentarium Corp, Helsinki, Finland) was inserted, and its position was confirmed by using chest radiography. The Tonocap TC-200 (Tonometrics Division, Instrumentarium Corp), which uses intermittent air tonometry, was used to measure PtCO2. The Tonocap is subject to less operator error, can measure PtCO2 more often, and is less time-consuming to use than manual establishment of each measurement. It also eliminates the limitations of tonometry with isotonic sodium chloride solution, such as the instability of the solution, and the bias of arterial blood gas analyzers. The precision of tonometric measurements obtained with the Tonocap compared with measurements obtained by using tonometry with isotonic sodium chloride solution has been established both in vitro and in vivo.

The tonometer was used to measure PtCO2 before feeding began and at 4, 24, and 48 hours after feeding began, when feeding formula was present in the stomach. The PtCO2 was also measured after aspiration of the gastric tonometer to empty the stomach at 4, 24, and 48 hours after feeding began. Triplicate readings of the PtCO2 were obtained at each time. The direct measurement of PtCO2 was used to calculate the gastric intramucosal pH (pHi) and the CO2 gap.

Data Analysis
Statistical analyses were performed to an α of .05 by using SPSS for Windows, Release 8.0.0 (SPSS Inc, Chicago, Ill). Descriptive statistics of the sample, including means, medians, and SDs, were calculated. Nonparametric statistical testing was used in this study because the clinical characteristics of critically ill patients resulted in data that were not normally distributed. The Wilcoxon signed rank test was used to determine if the measurements of PtCO2 obtained in the full stomach differed significantly from measurements obtained in the empty stomach at 4, 24, and 48 hours. The Kruskal-Wallis test was used to determine if measurements of PtCO2 obtained at 4, 24, or 48 hours differed significantly and if measurements obtained in the full stomach differed significantly from those obtained in the empty stomach.

Results
Characteristics of the Sample
A total of 10 women and 10 men were enrolled in the study. The mean age was 70.4 years (range 37-84 years), and the mean score on the Acute Physiology and Chronic Health Evaluation II at the time of admission to the study was 26. The diagnoses or reasons for admission varied and included pneumonia/sep-

![Figure 1](http://ajcc.aacnjournals.org/) Variation in enteral feeding rates during the 48 hours of data collection.
sis (n=10), general surgery (n=4), cardiovascular conditions (n=3), and other (n=3).

All patients were in a stable hemodynamic state during data collection, although some experienced transient periods of hypotension (>10 minutes) during the study period. Hemodynamic parameters were not precisely controlled, and strategies to support cardiovascular status, including the use of inotropic agents, were at the physician’s discretion.

Enteral feeding rates varied considerably between patients (Figure 1), and prokinetic agents, which were administered at the physician’s discretion, were not widely or consistently used. Enteral feeding formula was present in the stomach of all patients throughout the study period, and we did not think that alterations in the feeding patterns between patients would alter tonometric readings obtained in the fed stomach. Gastric residual volumes were measured throughout the study and were unpredictable. Although no distinct pattern was evident, the mean gastric residual volume did not increase substantially even when the rate of enteral feeding was at its highest (Table 1). Gastric aspirates were returned to a maximum of 150 mL. The mean pH of the gastric aspirate was 4.0 (SD 2.6) before feeding and 5.4 (SD 1.5) once feeding had begun. None of the patients received H2-receptor antagonists during the study period.

### Measurements Obtained With the Gastric Tonometer

The $P_{\text{CO}_2}$ increased after the administration of enteral feeding formula and remained elevated in the empty stomach throughout the study period (Table 2). Measurements obtained at 4 hours were similar for the full and the empty stomach (ie, the stomach in which feeding formula was aspirated via the tonometer). However, the $P_{\text{CO}_2}$ at 24 and 48 hours after feeding began was higher in the empty stomach than in the full stomach.

The $P_{\text{CO}_2}$ measured via the gastric tonometer was used to calculate the CO2 gap and pH. $P_{\text{CO}_2}$, CO2 gap, and pH for all patients followed a similar pattern during the 48-hour study period (Figure 2). Measurements obtained for patient 6 were clearly outside the range for the other patients and were also more than 3 SDs from the norm. Patient 6 also had a

### Overall, $P_{\text{CO}_2}$ in the full stomach differed significantly from $P_{\text{CO}_2}$ in the empty stomach. $P_{\text{CO}_2}$ increased when feeding was started and then appeared to stabilize at 24 and 48 hours after feeding began.

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Gastric residual volumes at each time for different rates of enteral feeding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rate of enteral feeding, mean, mL/h</td>
<td>Gastric residual volume, mL</td>
</tr>
<tr>
<td>Before feeding</td>
<td>0-1135</td>
</tr>
<tr>
<td>Hour 4</td>
<td>10-270</td>
</tr>
<tr>
<td>Hour 24</td>
<td>0-350</td>
</tr>
<tr>
<td>Hour 48</td>
<td>5-410</td>
</tr>
</tbody>
</table>

### Table 2 | $P_{\text{CO}_2}$ of the gastric mucosa obtained via tonometry |
<table>
<thead>
<tr>
<th>Time after enteral feeding begun, h</th>
<th>Feeding solution present in stomach</th>
<th>Mean of triplicate measurements of $P_{\text{CO}_2}$, mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No</td>
<td>38.8 ± 5.8</td>
</tr>
<tr>
<td>1</td>
<td>Yes</td>
<td>52.2 ± 9.8</td>
</tr>
<tr>
<td>4</td>
<td>Yes</td>
<td>49.1 ± 9.8</td>
</tr>
<tr>
<td>4</td>
<td>No</td>
<td>49.1 ± 7.6</td>
</tr>
<tr>
<td>24</td>
<td>Yes</td>
<td>39.7 ± 19.5</td>
</tr>
<tr>
<td>24</td>
<td>No</td>
<td>47.1 ± 18.0</td>
</tr>
<tr>
<td>48</td>
<td>Yes</td>
<td>38.6 ± 19.8</td>
</tr>
<tr>
<td>48</td>
<td>No</td>
<td>47.1 ± 18.3</td>
</tr>
</tbody>
</table>

### Table 3 | $P_{\text{CO}_2}$ of gastric mucosa obtained via tonometry, carbon dioxide gap, and gastric intramucosal pH in the presence of enteral feeding solution at each time |
<table>
<thead>
<tr>
<th>Parameter</th>
<th>Time after enteral feeding begun, hours</th>
<th>Mean rank</th>
<th>$\chi^2$</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$P_{\text{CO}_2}$, mm Hg</td>
<td>4</td>
<td>43</td>
<td>25.07</td>
<td>&lt;.001*</td>
</tr>
<tr>
<td>24</td>
<td>21</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>48</td>
<td>19</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carbon dioxide gap, mm Hg</td>
<td>4</td>
<td>43</td>
<td>26.42</td>
<td>&lt;.001*</td>
</tr>
<tr>
<td>24</td>
<td>22</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>48</td>
<td>18</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$P_{\text{H}_2}$</td>
<td>4</td>
<td>15</td>
<td>21.97</td>
<td>&lt;.001*</td>
</tr>
<tr>
<td>24</td>
<td>32</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>48</td>
<td>38</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Significant at $P \leq .05$. |
Figure 2  $\text{PCO}_2$ of the gastric mucosa obtained via tonometry, carbon dioxide gap, and gastric intramucosal pH for all patients. E indicates measurements obtained in the absence of enteral feeding solution (ie, the empty stomach); F, measurements obtained in the presence of enteral feeding solution (ie, the full stomach); #, measurements obtained for patient 6 at 24 and 48 hours.
greater severity of illness, as indicated by the score of the Acute Physiology and Chronic Evaluation II, and required a high level of inotropic support and fluid resuscitation to maintain hemodynamic stability. For these reasons, data collected for patient 6 were excluded from further data analyses.

**Statistical Analysis**

PtCO2 values at 4, 24, and 48 hours with food in the stomach differed significantly (Table 3). However, analysis of the mean ranks indicated that PtCO2, CO2 gap, and pH in the full stomachs did not differ significantly between 24 and 48 hours after the start of enteral feeding. After aspiration of the gastric tonometer (ie, with the stomach empty), the PtCO2, CO2 gap, and pH also differed significantly between 4, 24, and 48 hours (Table 4). Again, analysis of the mean ranks indicated that the significant difference was between measurements obtained at 4 hours and those obtained at 24 or 48 hours.

Although the PtCO2 appeared to stabilize after 24 hours of feeding, at 24 and 48 hours, measurements obtained in the presence of enteral feeding solution differed significantly ($z=3.58, P<.001$) from those obtained in the absence of feeding solution. These differences were also evident for the CO2 gap ($z=3.58, P<.001$) and pH (z = 3.58, P < .001) at these times. Measurements of PtCO2, CO2 gap, and pH obtained in the full stomach did not differ significantly from those obtained in the empty stomach after 4 hours.

**Discussion**

The Initial Response to Enteral Feeding

The purpose of this study was to investigate the influence of enteral feeding on the measurement of PtCO2 in critically ill patients. An increase in the PtCO2 after enteral feeding occurred and was matched by an increase in the CO2 gap and a decrease in the pH. This response is congruent with the small amount of reported data on the influence of enteral nutrition on the measurement of PtCO2.18,19

The initial increase in PtCO2 may be related to an increase in gastric mucosal blood flow associated with enteral feeding.27 With an increase in blood flow, CO2 may be flushed out of the mucosal microvasculature; however, this possibility has not been examined in experimental studies. The stimulation of hydrogen ion secretion during enteral feeding may also contribute to an increase in PtCO2. The subsequent buffering of hydrogen with bicarbonate ions may result in a production of CO2 that is unrelated to gastrointestinal mucosal blood flow.29,30 Although the use of H2-receptor antagonists results in a decrease in hydrogen ion secretion and may limit the increase in PtCO2 generated by the buffering of hydrogen with bicarbonate ions,29 this effect has not been observed in critically ill patients given enteral feedings.19 Consequently, most likely the increase in PtCO2 was not due to a lack of suppression of hydrogen ion secretion and may be due to enzymatic digestion of nutrients in the stomach, although further studies are required to specifically address this issue.

An increase in regional cellular metabolism and CO2 production may also result in an increase in PtCO2.16 Enteral feeding stimulates the digestive system,22 and the resulting increased metabolic rate may cause an increase in regional CO2 production and PtCO2 levels. Furthermore, in critically ill patients, the increased metabolic demand and regional oxygen requirements may not be met by a simultaneous increase in oxygen delivery.16 The consequences of this supply-demand mismatch could contribute to the development of hypoxia and anaerobic generation of energy,19 also resulting in an increase in CO2 production and thus measurable alteration in PtCO2.

Marik and Lorenzana19 reported that the initial increase in PtCO2 in patients given enteral feeding returned to baseline values within 1 hour after the feeding was stopped. On the basis of these data, they concluded that enteral feeding interfered with the measurement of PtCO2 and recommended that enteral feeding be stopped 1 hour before measurements are obtained with a gastric tonometer. Levy et al18 also found an increase in PtCO2 during enteral feeding, but in their study, the PtCO2 did not return to baseline once feeding was stopped. Nevertheless, Levy et al also recommended that enteral feeding be stopped before PtCO2 is measured.

Clinically, stopping enteral feeding for 1 hour to

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Time after enteral feeding begun, hours</th>
<th>Mean rank</th>
<th>$\chi^2$</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>PtCO2, mm Hg</td>
<td>4</td>
<td>35</td>
<td>6.59</td>
<td>.04*</td>
</tr>
<tr>
<td></td>
<td>24</td>
<td>23</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>48</td>
<td>26</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carbon dioxide gap</td>
<td>4</td>
<td>34</td>
<td>6.91</td>
<td>.03*</td>
</tr>
<tr>
<td></td>
<td>24</td>
<td>24</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>48</td>
<td>27</td>
<td></td>
<td></td>
</tr>
<tr>
<td>pH</td>
<td>4</td>
<td>21</td>
<td>6.79</td>
<td>.03*</td>
</tr>
<tr>
<td></td>
<td>24</td>
<td>29</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>48</td>
<td>35</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Significant at $P ≤ .05$.  

---

**Table 4** PtCO2 of gastric mucosa obtained via tonometry, carbon dioxide gap, and gastric intramucosal pH in the absence of enteral feeding solution at each time.
allow monitoring with gastric tonometry creates yet another barrier to the provision of adequate enteral nutrition, particularly if frequent tonometric readings are required. Furthermore, stopping enteral feeding does not necessarily ensure gastric emptying, particularly in critically ill patients, who often experience alterations in gastric motility. Therefore, gastric aspiration, which correlates with the degree of gastric emptying, most likely is a more effective means of emptying the stomach before PCO$_2$ is measured and will interfere less with the administration of enteral feeding solution.

The Response to Enteral Feeding Over Time

Examination of the response to enteral feeding over time revealed that the initial increase in PCO$_2$ is not sustained, and the PCO$_2$ appeared to stabilize after 24 hours of enteral feeding. Possibly, the initial increase in PCO$_2$ is related to a reversal of gastrointestinal mucosal vasoconstriction and increased gastric mucosal blood flow associated with the administration of enteral feeding solutions. Sustained administration of enteral feeding solution may maintain improved gastric mucosal blood flow, preventing the buildup of CO$_2$ in the mucosal vasculature. This possibility may explain why, in the absence of illness-related crises, the PCO$_2$ values at 24 and 48 hours in our study did not differ significantly.

Our finding that PCO$_2$ in the full and the empty stomach stabilized after 24 to 48 hours of enteral feeding suggests that the recommended practice of withholding enteral feeding for 1 hour may be unnecessary. Once enteral feeding is established for 24 hours, the PCO$_2$ could be obtained without stopping enteral feeding if the measurements are consistently obtained in the same intragastric environment, that is, with enteral feeding solution in the stomach or after aspiration of the gastric tonometer to empty the stomach. Possibly, PCO$_2$ could be measured reliably earlier than 24 hours after the start of feeding; however, further longitudinal studies are required to substantiate these findings.

Although PCO$_2$ appeared to stabilize after 24 hours of enteral feeding, at 24 and 48 hours, measurements obtained in the full stomach differed significantly from those obtained in the empty stomach. This finding suggests that the presence of enteral feeding solution does interfere with the measurement of PCO$_2$. The differences between PCO$_2$ in the full stomach and PCO$_2$ in the empty stomach may be related to the ability of CO$_2$ to diffuse into the tonometer balloon when enteral feeding solution is in the stomach. Enteral feeding solution in the stomach causes the tonometer balloon to move away from the gastric mucosa and may cause a decrease in measured PCO$_2$. Also, when enteral feeding solution is present in the stomach, the CO$_2$ must travel through the solution to the tonometer balloon. The ability of CO$_2$ to diffuse through this type of liquid is unknown but is an interesting area of research that could be addressed in the laboratory setting.

On the basis of our results, a change in clinical practice to allow continuation of enteral feeding in patients being monitored with gastric tonometry could be considered, provided the gastric tonometer is aspirated before the PCO$_2$ is measured. Aspiration of gastric contents through the appropriate port of the tonometer before PCO$_2$ is measured will limit the interference to diffusion of CO$_2$ created by the presence of enteral feeding solution. However, the ability to completely empty the stomach when this method is used is uncertain, because only minimal data indicate a strong correlation between measurements of gastric aspirates and the degree of gastric emptying. Aspiration of the tonometer would cause the balloon to be in close proximity to the gastric mucosa in that area of the stomach, even if the stomach was not completely empty.

Unlike the findings at 24 and 48 hours, at 4 hours after the start of enteral feeding, PCO$_2$ in the full stomach did not differ significantly from PCO$_2$ in the empty stomach, possibly because of a difference in the mean enteral feeding rate at these 3 times. At 4 hours, that mean volume of enteral feed delivered was 120 mL, which was considerably less than at 24 hours (172 mL) or 48 hours (240 mL). With a smaller volume of feeding solution in the stomach at 4 hours, the tonometer balloon may have been close to the gastric mucosa and the amount of feeding solution may have been insufficient to alter the diffusion of CO$_2$ from the gastric mucosa to the balloon. This possibility may explain why the measurements of PCO$_2$ obtained at 4 hours in the presence and absence of enteral feeding solution were similar. Again, further laboratory studies would be required to definitively explore this issue.

The recommendation that enteral feeding be withheld or stopped for 1 hour before tonometric measurement of Pco$_2$ is based on the premise that enteral feeding interferes with this measurement. Thus, a conflict is created between using a gastric tonometer to monitor gastric mucosal hypoperfusion and providing enteral nutrition. Although we found an initial increase in PCO$_2$, our data also indicate that after 24 hours of enteral feeding the PCO$_2$ stabilizes. Nevertheless, we found significant differences between measurements obtained in the presence and the absence of feeding solution. Consequently, PCO$_2$ should be measured in a
consistent intragastric environment, preferably after the stomach has been aspirated. This recommendation is
based on the apparent alteration in CO₂ diffusion that occurs when reasonable volumes of enteral feeding
solution are present and the clinical imperative of detecting increasing levels of PCO₂ as an indicator of
gastric mucosal perfusion.

The current recommendation to withhold enteral feeding for 1 hour is of questionable value. Although
further research is required, particularly research with a larger sample size and more tightly controlled clinical
parameters than those of our study, possibly tonometric measurements can be reliably obtained after aspiration
of the tonometer to empty the stomach rather than after a halt in feeding. A change in clinical practice that
allows enteral nutrition to continue during monitoring with gastric tonometry will enhance nutritional intake in
a group of patients susceptible to malnutrition and maximize the use of tonometric monitoring of early changes in regional perfusion.

ACKNOWLEDGMENTS
We acknowledge the support and assistance of Dr Keith Burgess, director, Department of Critical Care, and the staff of the intensive care unit at Manly Hospital, NSW, Australia. This research was funded by grants from the Northern Sydney Area Health Service Research Grants Program and the Australian College of Critical Care Nurses (formerly the Confederation of Australian Critical Care Nurses).

Commentary by Mary Jo Grap (see shaded boxes).

REFERENCES
Gastric Tonometry and Enteral Nutrition: a Possible Conflict in Critical Care Nursing Practice
Andrea P. Marshall and Sandra H. West
Am J Crit Care 2003;12 349-356
Copyright © 2003 by the American Association of Critical-Care Nurses
Published online http://ajcc.aacnjournals.org/

Personal use only. For copyright permission information:
http://ajcc.aacnjournals.org/cgi/external_ref?link_type=PERMISSIONDIRECT

Subscription Information
http://ajcc.aacnjournals.org/subscriptions/

Information for authors
http://ajcc.aacnjournals.org/misc/ifora.xhtml

Submit a manuscript
http://www.editorialmanager.com/ajcc

Email alerts
http://ajcc.aacnjournals.org/subscriptions/etoc.xhtml