Communications to the Editor

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Anemia and COPD

To the Editor:

John and colleagues reported in a recent issue of CHEST (March 2005) a relatively high frequency of anemia in patients with COPD. However, they did not mention whether the studied population was in treatment with angiotensin-converting enzyme (ACE) inhibitors or angiotensin II type 1 (AT1) receptor blockers.

Several observations suggested that an intact and activated renin-angiotensin system may be an important determinant of erythropoiesis, including a variety of clinical conditions such as hypertension, chronic renal insufficiency, heart failure, and COPD. Consequently, the assumption of ACE inhibitors and AT1 receptor blockers has been shown to reduce hemoglobin concentrations both in healthy subjects and in those with pathologic conditions. Thus, therapy with ACE inhibitors and AT1 receptor blockers might have been a confounder in this study.

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To the Editor:

The author of this comment mentioned that therapy with angiotensin-converting enzyme (ACE) inhibitors might be a confounder in interpreting the results of our study. In this patient population, 23 patients of the nonanemic group (88 patients) were treated with ACE inhibitors or angiotensin type-1 (AT1) receptor blockers. In the anemic group (13 patients), 3 patients received cotherapy with ACE inhibitors or receptor blockers. The $\chi^2$ test revealed no difference in the occurrence of ACE inhibitor therapy between both groups (p = 0.813). Therefore, we conclude that the reported prevalence of anemia in COPD patients in our study is not influenced by an ACE inhibitor or AT1 receptor blocker medication. Overall, it is an interesting issue that should be taken into consideration for future studies.

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Diverse Influences on Blood Glucose Measurements in the ICU Setting

To the Editor:

We thank Finkielman et al (May 2005) for addressing the important issue of the comparability of whole blood glucose
measurements in the ICU setting to glucose levels measured in the clinical laboratory. The authors concluded that while, on average, bedside whole blood glucose measurements (SureStepFlexx; Lifescan; Milpitas, CA) provide an adequate measurement of laboratory-measured plasma glucose, the two measurements (bedside and laboratory measured) are quite different on an individual basis. The noncomparability is due to at least two major factors: susceptibility to interference by many currently available bedside glucose meters, and the authors’ incorrect assumption that venous blood glucose and capillary blood glucose are interchangeable.

The whole blood milieu of the average ICU patient can be very different from that of the average patient with diabetes: ICU hematocrits can be very low, levels of P₀₂ can be extremely high or low, and many patients have acid-base abnormalities. These three variables can significantly affect the whole blood glucose measurements by currently available analyzers. In fact, Lifescan cautions in its SureStepFlexx product literature that low hematocrits (< 25%) will render the measured glucose inaccurate. As the SureStepFlexx meter uses glucose oxidase to measure glucose, its operation is dependent on the P₀₂ of the blood. To prevent this P₀₂ dependence, at least one meter manufacturer uses glucose dehydrogenase rather than glucose oxidase. Unfortunately, glucose dehydrogenase reacts with maltose, a metabolite of icodextrin, a substance used in peritoneal dialysis. This reaction will result in artifically increased glucose in patients receiving this type of dialysis. If the authors have access to the patients’ hematocrit and P₀₂, can they provide some analysis to demonstrate the glucose-hematocrit and glucose-P₀₂ dependencies?

The study was comprised of two types of glucose comparisons grouped together in the results and conclusion. In one type of comparison, the same sample, drawn from either an arterial or central vein catheter, was measured at the bedside and in the laboratory. In the second comparison, bedside measurements from a capillary (finger-stick) origin were correlated to laboratory plasma measurements taken from a peripheral vein. Venous-capillary glucose can vary considerably for up to 4 hours after a meal or after a patient has received insulin. Figure 1 in their article, the graph of differences between the laboratory-measured and bedside-measured glucose, does not distinguish the source of the samples compared. Without this information, it may be reasonable to conclude that some of the larger differences represent the nonidentical capillary and venous samples. Can these venous capillary comparisons be excluded and the Figure redrawn?

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To the Editor:

We thank Drs. Binette and Cembrowski for their important and valid comments about our study (May 2005). As they correctly pointed out, the whole blood milieu of the average ICU patient can be very different from that of the average patient with diabetes. Although glucose measurements can be affected by several critical care variables, few studies have looked at the performance of point-of-care glucose testing in the ICU setting. Although hypotension was not associated with a difference between bedside and plasma glucose values in our study, Atkin et al found that finger-stick glucose testing does not accurately represent venous glucose in patients with severe hypotension. Maser et al also noted that the glucose concentration in arterial serum samples was significantly higher than the corresponding capillary whole blood (finger-stick) glucose by 9 mg/dL and 21 mg/dL, unadjusted and adjusted to the hematocrit, respectively. Additionally, high (> 40%) and low (< 30%) hematocrits negatively and positively biased point-of-care glucose testing devices; high arterial oxygen tension (> 250 mm Hg) produced a negative bias, while pH 6.80 to 7.53 did not affect glucose measurements substantially. Unfortunately, we are unable to perform the analyses suggested by Drs. Binette and Cembrowski since we did not obtain the hematocrit and arterial oxygen tension values at the time the blood samples were obtained in our study population.

Drs. Binette and Cembrowski speculate that the large differences between the bedside blood and plasma glucose measurements seen in our study were due to nonidentical capillary and venous samples. However, Ray et al reported results similar to ours. In a small prospective study of 105 arterial blood samples from 10 critically ill adults, they measured glucose at the bedside (One Touch Profile Blood Glucose Monitoring System; LifeScan; Milpitas, CA) and in the core chemistry laboratory and found the mean difference between the two methods to be −0.72 mg/dL with the 95% limits of agreement between +41 mg/dL and −40 mg/dL.

In spite of the limitations highlighted by Drs. Binette and Cembrowski, we believe that our conclusions are true and supported by our data. Although, on average, bedside glucose measurement provides a reasonable estimation of laboratory-measured plasma glucose, the former method gives an unreliable estimate of the latter in individual patients. This fact has an important clinical implication, particularly in this era of tight glucose control. A definitive prospective study on point-of-care glucose testing in the ICU setting seems warranted.

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A New Tracheostomy Procedure

To the Editor:

In a recent issue of CHEST (July 2004),1 Ferraro et al suggested using a pediatric uncuffed endotracheal tube that was 4 mm in the inner diameter to ventilate patients during either percutaneous dilatational tracheostomy (PDT) according to Ciaglia et al2 or translaryngeal tracheostomy according to Fantoni and Ripamonti.3 This suggestion limits the risk of hypventilation and allows the whole procedure to proceed under direct vision using a flexible bronchoscope that is inserted parallel to the pediatric tube. Having used PDT for several years in our respiratory ICU, and despite the favorable results presented in this interesting report, we are not prone to adopt the procedure reported by Ferraro et al for the following reasons.

The suggested procedure requires the extubation of the patient before PDT or translaryngeal tracheostomy, which may be unsafe in some patients with respiratory failure and may be associated with an additional and unacceptable risk of difficult reintubation,4 even if ventilating the tube exchanger might help.5 Moreover, the suggested procedure uses an uncuffed tracheal tube, which may promote the inhalation of pharyngeal secretions and gastric content. Finally, hypventilation is not strictly avoided, as attested in Table 2 in the article by Ferraro et al.

In our respiratory ICU, we routinely use the PDT procedure under intermittent endoscopic monitoring and have observed that the technique of Ciaglia et al2 using a one-step dilator is always possible without extubating the patient. This requires a careful transillumination of the tip of the cuffed tracheal tube with a flexible bronchoscope that is briefly inserted into the tube. This localisation may be repeated during the procedure and allows a safe puncture of the trachea without puncturing the cuff and without a significant risk of hypventilation.

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We appreciate the interest of Dr. Cuvelier and coworkers in our study that was published in CHEST (July 2004).1 We agree that the use of transillumination by the bronchoscope may generally allow correct needle placement for percutaneous tracheostomy; but this is not in contrast with our technique.

Since 1985, when Ciaglia introduced his percutaneous dilatational tracheostomy technique, a separate operator has been used to maintain the placement of the withdrawn endotracheal tube with the cuff just above the vocal cords. This way of airway management is risky because accidental extubation, transfixation of the tube, and deflation of the cuff can happen. Serious complications, including emphysema, pneumothorax, and death, can result from the loss of the airway.2,4,6 but this technique has been used successfully by Cuvelier et al. Further, there is much evidence in the literature about the dangers of interference between bronchoscopy and ventilation.5,7,8 For this reason, Cuvelier et al use “intermittent endoscopic monitoring” to protect against hypventilation. There is then a real risk of tracheal damage during the dilation phase and tracheostomy tube placement phase, which are both carried out without the aid of an endoscopic view.

Our technique maintains continuous airway control (ie, a patent and secure airway by orotracheal intubation at the level of the carina), a continuous endoscopic view (independent from ventilation), and continuous gas passage from the glottis (translaryngeal open ventilation in pressure-controlled ventilation tube) during the entire procedure. Our technique avoids airway loss, paratracheal cannulation, posterior tracheal wall damage, emphysema (subcutaneous and/or mediastinal), hypoxia, and the inhalation of biological liquid (eg, blood or pharyngeal secretions). To accomplish this, we never extubate the patient, and we change the endotracheal tube only by means of the correct and safe use of a tube exchanger without laryngoscopy.1

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