1-1-2000

Sublingual capnometry: In search of its holy grail

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Recommended Citation
Yee J, Susanto I. Sublingual capnometry: In search of its holy grail. Chest 2000; 118(4):894-896.

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Sublingual Capnometry

In Search of Its Holy Grail

In the climactic scene from the adventure film *Indiana Jones and the Last Crusade*, the protagonist must choose, from a number of bedazzling chalices, the Holy Grail of Arthurian legend. The knight whose destiny is to guard the grail cautions Jones to "choose wisely," and Jones rightly ignores the fanciful alternatives and chooses the cup of a laborer, simple and practically suited to its task. Challenged to find one of the "holy grails" of medicine, that is, a simplified technique that can detect early tissue hypoperfusion during circulatory shock, Povoas and colleagues, in this issue of *CHEST* (see page 1127), may have approached one step closer to the goal with the technique of sublingual capnometry.

Tissue hypoperfusion and the ensuing ischemia represent a common underlying theme in various states of circulatory shock. Intracellular acidosis, by virtue of anaerobic metabolism of glucose, hydrolysis of high-energy phosphates, and lipolysis, is accompanied by elevations of the partial pressure of venous and tissue carbon dioxide.[1] The measurement of visceral intramucosal pH, most specifically of the stomach, has been posited to reflect the net balance of aerobic and anaerobic cellular metabolism.[2,3] However, ascertainment of this exacting parameter has been fraught with setbacks and essentially abandoned. Part of the failure has been attributed to an inability to determine accurately the mucosal bicarbonate concentration.[4,5] Following the abandonment of gastric mucosal pH measurement, another surrogate for tissue hypoperfusion was embraced, visceral hypercarbia or mucosal P_{CO_2}.

Elevations of visceral P_{CO_2}, particularly those of gastric P_{CO_2}, herald the heightened bicarbonate buffering of hydrogen ions generated from intracellular acidosis, concomitant with a diminution of CO_2 washout (Povoas and colleagues).[1,6] Indeed, tissue hypercarbia has been shown to reflect splanchnic organ dysoxia during states of circulatory shock, and gastric mucosal capnometry has established a relationship between advancing visceral hypoperfusion and increasing mucosal P_{CO_2}. However, gastric tonometry has been encumbered by the requisite and prolonged period of equilibration between the mucosal wall and the fluid-filled measuring device (Povoas and colleagues). Despite the invention of more rapidly equilibrating gas-filled devices, the technique has nonetheless suffered from confounding errors in measurement introduced by enteral feeding and hydrochloric acid secretion, the latter of which can be obviated by pharmacologic gastric acid suppression (Povoas and colleagues).

The notion that the viscera represented the "canary of the body" was subsequently refuted by observations in nonvisceral organs, including kidney and tongue, which afforded information equal in value to that of gastric capnometry (Povoas and colleagues).[7] To evaluate this finding further, Povoas and colleagues compared gastric tonometry with sublingual capnometry in a porcine model of hypovolemic shock. During 120 min of observation, following induction of hemorrhage and hypotension, gastric and sublingual P_{CO_2} rose commensurately and early (within 10 min of induction of hemorrhage), and presaged increases in venoarterial P_{CO_2} gradients and arterial lactate formation. Reconstitution of circulatory volume by reinfusion of shed blood rapidly reversed the sublingual tissue hypercarbia, which preceded the slower reversal of arterial lactate to the normal range. These results confirmed

that the information obtained from tissue capnometry of the GI tract proximal to the stomach compared favorably with that of the stomach.[8,9] Furthermore, sublingual capnometry is more facilely imposed on the critically ill patient than its gastric counterpart, and its ease of insertion is a powerful inducement to its application. Last, Weil and colleagues[10] previously demonstrated that this methodology, in a limited number of patients, predicted survival among those who had incurred different states of circulatory shock.

The question of whether the conclusions of this investigation and the cumulative data therein warrant widespread clinical application of sublingual tonometry must be confronted. Several issues should be considered. First, tissue hypercarbia is produced by a combination of increased production and decreased removal of CO_2. In addition, one must be mindful that the readily diffusible nature of CO_2 creates a dynamic balance between systemic and local tissue P_{CO_2}, and that the measurement of mucosal P_{CO_2} does not represent an absolutely fail-safe measure. For example, during induced respiratory acidosis and alkalosis in hemodynamically stable experimental animals, significant alterations of gastric and ileal mucosal P_{CO_2} may occur, thereby creating the potential for misinterpretation of tissue perfusion.[11] This issue is of minor importance in most instances of either pure hemorrhagic or hypovolemic shock. However, when circulatory shock is associated with ARDS and permissive hypercapnia is utilized, tissue hypercarbia must be more cautiously interpreted.

Second, although sublingual capnometry sensor placement entails considerably less effort than gastric capnometry, is the technique always so easily applied to the critically ill patient? Failure of the sensor to equilibrate with sublingual P_{CO_2} may occur when there is inability to properly maintain sensor position beneath the tongue of a patient.[10] Although it appears to be technically trivial, the ability of the sublingual capnometry sensor to equilibrate and deliver the information should be reproduced and substantiated by others in the busy and less well-controlled critical care setting, especially given that the majority of studies regarding sublingual capnometry represent the work of Povoas and colleagues.

Third, additional data from prospective studies are needed to demonstrate whether sublingual capnometry is uniformly applicable to other types of shock. Measurements of gastric P_{CO_2} during ovalbumin-induced anaphylactoid shock and sublingual P_{CO_2} in staphylococcal-imposed septic shock suggest that this technology may be applied more universally.[12,13]

Fourth, as with other interventions indigenous to the critical care environment, the ultimate test of a new intervention is its ability to predict or improve patient survival in a manner superior to that of existing methodologies. If, in hemorrhagic shock, sublingual capnometry reveals the earliest sentinel event of trouble (a rise in sublingual P_{CO_2}), but not its cause, does it justify its expense? Does knowing that trouble is brewing earlier have an impact on patient outcomes? The same question may be raised regarding the potential applicability of this technique in other circulatory abnormalities.

Alas, maybe the greatest lesson imparted by the quest for the Holy Grail is that simplicity represents the highest form of elegance. Povoas and colleagues have chosen wisely and pioneered a simple noninvasive methodology that monitors the critically ill patient. However, whether this technology will augment the care of the severely ill shall remain unanswered until the gauntlet laid down by Povoas and colleagues is picked up by those who desire to pen the next chapter of the adventure. The clinical utility and predictive accuracy of sublingual capnometry in direct comparisons with conventional technologies in hypovolemic and other forms of circulatory shock will remain eagerly anticipated until such times!

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