

Commentary

Tonometry of partial carbon dioxide tension in gastric mucosa: use of saline, buffer solutions, gastric juice or air

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Received: 23 May 2000
Accepted: 30 May 2000
Published: 20 June 2000

Crit Care 2000, 4:201–203

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Abstract

Tonometry of gastric mucosal partial carbon dioxide tension (PCO_2) has been forwarded as a clinically applicable tool to monitor regional perfusion adequacy during circulatory failure. The technique is still not used widely, partly because of methodological problems. Current measurement principles are reviewed, with help of the report on PCO_2 measurements in gastric juice and tonometer in this issue.

Keywords: clinical use of tonometers, gastric partial carbon dioxide tension, gastric pH, tonometry technique

Gastric mucosal PCO_2 tonometry has evolved as a useful adjunctive monitoring technique that is physiologically sound, simple and noninvasive, even though the precise methodology and interpretation of the results continue to be debated [1]. A supranormal gastric mucosal to arterial blood PCO_2 has been regarded as indirect evidence for mucosal hypoperfusion [1]. Among the controversies are the issues of the need for gastric acid secretion suppression and the best PCO_2 measurement medium for gastric tonometry [1,2]. Administration of H_2 -blockers before intraluminal gastric tonometry has been advised to obviate erroneously high gastric PCO_2 following carbon dioxide production by bicarbonate buffering of actively secreted gastric acid.

For the original tonometric technique, a balloon-tipped saline-filled tonometer is used that necessitates *ex vivo* PCO_2 measurements with a blood gas machine, after a

certain dwell time and after aspiration of the saline [1]. It has been recognized, however, that saline may yield erroneous PCO_2 values, and that the bias may depend on the solution and the type of blood gas machine used to measure PCO_2 [1,3–5]. Indeed, the bias may be buffer- and pH-dependent [1,3,5]. Various buffer solutions have been proposed to circumvent PCO_2 losses during handling and measurement in the blood gas machine and to circumvent underestimation of PCO_2 in saline [1,3,5].

Some of these problems may be avoided with air tonometry, in which surrounding intraluminal gastric carbon dioxide is allowed to rapidly equilibrate with the air-filled tonometer balloon PCO_2 [4,6]. Various types have been proposed, depending on the mode of recirculation of air in the catheter between measurement intervals, and one is commercially available (Tonocap; Datex, Helsinki, Finland) [1,5,6]. This automated semicontinuous technique (involving an *ex vivo*

infrared sensor) may both over- and underestimate saline PCO_2 tonometry, depending, among other reasons, on correction factors for incomplete equilibration times and the bias of the blood gas machine used for the latter [1,3,4,6,7].

Other authors have determined gastric air or juice PCO_2 using blood gas machines, with the presumption that mucosal PCO_2 is in equilibrium with gastric air and juice PCO_2 at steady state; this thereby circumvents the need for equilibration with saline in a balloon tonometer [1,6,8]. 'Balloonless' gastric air PCO_2 tonometry has been compared in dogs with saline balloon PCO_2 tonometry by Salzman *et al* [8], who found that these methods were equivalent under controlled circumstances after full acid secretion suppression with H_2 -blockade. The presumption that gastric air is in equilibrium with gastric mucosa may not be true in the presence of active secretion of acid and bicarbonate by the stomach wall. Indeed, at low gastric juice pH in the stomach without acid secretion suppression, gastric juice PCO_2 exceeds that in the mucosa and supplying blood, whereas after H_2 -blockade both measures may be at equilibrium [1,9]. The former can be explained by buffering of bicarbonate by gastric acid and intraluminal gastric production of carbon dioxide independently of transmucosal diffusion [9]. Indeed, duodenal PCO_2 in humans may exceed 100 mmHg, even in the absence of mucosal hypoperfusion, after buffering of gastric acid entering the duodenum by actively secreted bicarbonate [10]. Gastric juice/air and saline balloon PCO_2 tonometry may thus yield systematic and accidental differences, depending on the pH of the measurement media and the blood gas machine used [3,5,6].

Carbon dioxide production after bicarbonate buffering of gastric juice might explain why Dubin *et al* [11], as reported in this issue, found a higher PCO_2 in gastric juice than in concomitantly aspirated tonometer balloon saline, particularly at high PCO_2 , even though the pH of the juice aspirated was not given. Indeed, the regimen of H_2 -blockade they used in their patients may not have fully prevented active gastric acid secretion, because interindividual differences in the response to ranitidine are great and unpredictable. Nevertheless, the observation agrees with others showing a greater PCO_2 in gastric air than in tonometer balloon saline. However, others have shown that balloon air tonometry results in lower PCO_2 values than saline balloon PCO_2 tonometry, particularly at high PCO_2 , with blood gas analyzer bias and erroneous correction factors potentially responsible for the under- and overestimation by saline balloon tonometry, respectively [1,3,4,6–8,12]. Because the same machine was used and the propensity for carbon dioxide losses during sample handling and determination may be greater at low than at high pH [3,5], an underestimation of balloon saline versus gastric juice PCO_2 tonometry as an explanation for the results obtained by Dubin *et al* [11] is unlikely.

How gastric juice PCO_2 tonometry compares with a gold standard of intraluminal gastric PCO_2 tonometry (infrared PCO_2 tonometry in air?) would be of interest because, if these were interchangeable, then the former could replace the latter and render the technique relatively simple, albeit persistently dependent on a blood gas machine and subject to potential errors. Mohsenifar *et al* [13] used gastric juice PCO_2 tonometry and found that high values during weaning from mechanical ventilation predicted weaning failure and death, possibly associated with redirection of blood flow from the gastrointestinal tract to respiratory muscles associated with increased work of breathing. Even though not validated in that study, gastric juice PCO_2 tonometry could thus help the clinician in guiding therapeutic interventions. Otherwise, another gold standard may be afforded by the fiberoptic sensor technique (Paratrend®; Biomedical Sensors, High Wycombe, UK) [6], which has been used by Knichwitz *et al* [5] to yield more rapid and accurate information than saline balloon tonometry. Otherwise, another clue to suspect measurement error may be a negative mucosal to arterial blood PCO_2 gradient, but this may also occur after swallowing of environmental air.

Another type of measurement medium may be the gastric mucosal tissue itself, circumventing the confounding factors influencing intraluminal PCO_2 . In fact, this direct mucosal measurement could be done by electrodes incorporated into gastric catheters. Indeed, the ion-sensitive field effect transistor sensor has been successfully used for this purpose [14]. The disadvantage of such sensors over intraluminal methods may be that they will provide only regional information, whereas intraluminal techniques will provide more overall gastric mucosal information, at steady state. However, rapid, noninvasive, clinically applicable direct tissue PCO_2 measurement techniques are not yet available.

In the presence of increasing evidence of the value of gastric mucosal PCO_2 tonometry in clinical practice, there is increasing need for simple, rapid and accurate techniques, which are reproducible and validated against each other, and automated semicontinuous air tonometry may be the first step in that direction [1,4,6–8,12]. The trend is towards *in situ* sensors in the gastric lumen, and may evolve to direct tissue measurements. Concomitant gastric pH and arterial PCO_2 measurements may still help us to recognize and interpret some causes of error.

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