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Reliable gastric tonometry after coronary artery surgery: need for acid secretion suppression despite transient failure of acid secretion

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Abstract *Objective*: To study the need for suppression of gastric acid secretion for reliable intragastric partial pressure of carbon dioxide (PCO_2) tonometry by evaluating the effect of an oral dose of sodium bicarbonate before and after administration of the H_2-blocker ranitidine to mimic CO_2 generation following the buffering of acid by bicarbonate in patients after cardiac surgery.
Design: Prospective, open, non-randomized clinical study.
Setting: Cardiothoracic intensive care unit at a university hospital.
Patients: 10 patients after elective coronary artery bypass surgery.
Interventions: An oral dose of 500 mg sodium bicarbonate before and after acid secretion suppression by 100 mg ranitidine as an intravenous bolus given at ≈ 3 h after surgery (day 0) and on the first postoperative day (day 1).
Measurements and results: Intragastric PCO_2 ($iPCO_2$; tonometry), gastric juice pH (aspirate) and arterial blood gas values were measured. On day 0, the $iPCO_2$ was 25 ± 5 mmHg before and 31 ± 5 mmHg after the bicarbonate dose, 29 ± 5 mmHg after ranitidine infusion, and 31 ± 5 mmHg after the bicarbonate dose following the ranitidine infusion (NS). On day 1, the basal $iPCO_2$ was 32 ± 4 mmHg and it increased to 56 ± 25 mmHg following bicarbonate ($p < 0.01$). After ranitidine, the $iPCO_2$ was 33 ± 4 mmHg before and 40 ± 14 mmHg after bicarbonate (NS). Basal gastric juice pH was > 4 in nine of ten patients on day 0 and > 4 in seven of ten patients on day 1.
Conclusions: Pharmacological suppression of gastric acid secretion is mandatory for reliable $iPCO_2$ tonometry after cardiopulmonary bypass surgery, even when gastric acid secretion is transiently inhibited. In fact, gastric acid secretion was inhibited immediately after surgery, but returned on the first postoperative day in most patients, as judged from the bicarbonate back titration of gastric acid, even when gastric juice pH was relatively high.

Key words Tonometry · H_2-blocking agents · Acid secretion suppression · Gastric pHi · Back-diffusion

Introduction

Tonometry of the intragastric partial pressure of carbon dioxide ($iPCO_2$) and calculation of the gastric intramucosal pH (pHi) from $iPCO_2$ and the bicarbonate content of the blood, as measures of the adequacy of splanchnic perfusion, are increasingly used in intensive care patients. An elevated $iPCO_2$ or decreased pHi may indicate gastrointestinal hypoperfusion and may predict an adverse outcome [1–5]. Tonometry may

thus guide treatment and thereby improve outcome [1–5].

Suppression of gastric acid secretion is recommended since acid secretion may result in a false increase in $iPCO_2$, independently of mucosal perfusion and transmucosal diffusion, following intraluminal CO_2 production when gastric acid is buffered by intragastric bicarbonate derived from either non-parietal cells or entering the stomach via the esophagus or duodenum [6, 7]. The generation of CO_2 after administration of bicarbonate in the stomach to mimic bicarbonate secretion is circumvented, at least in healthy volunteers, by prior administration of H_2 antagonists such as ranitidine suppressing gastric acid secretion [7]. However, it is not clear whether there is a need to suppress acid secretion for reliable $iPCO_2$ tonometry in critically ill patients [5, 6, 8]. In fact, critically ill patients may have a low gastric acid secretion rate, which may result from gastric mucosal hypoperfusion, so that suppression of acid to prevent bicarbonate buffering of acid and a spurious elevation in $iPCO_2$, may not be necessary [8–10]. After major (cardiopulmonary bypass) surgery, gastric acid secretion may be reduced only in the immediate postoperative period and may return during recovery [9, 11]. Moreover suppressing acid secretion is not without risk and may be associated with the development of nosocomial pneumonia [12, 13]. Some studies on the pHi after cardiac surgery have utilized prior H_2 blockade [14–16], while others have not [4, 16–20]. This makes it difficult to interpret the transient postoperative decrease in pHi that accompanies postoperative complications in some patients in these studies [15–20]. In addition, there is the uncertainty that mucosal bicarbonate equals the blood bicarbonate [5].

We therefore studied gastric acidity, $iPCO_2$ before and after an oral dose of bicarbonate and before and after ranitidine administration, to evaluate the need to suppress acid secretion for reliable tonometry at two times, directly after coronary artery surgery and on the first postoperative day. Bicarbonate was administered intragastrically to mimic the buffering effects of bicarbonate from gastric mucosa, saliva, or duodenal fluid in the stomach [7].

Patients and methods

The study was approved by the Ethics Committee of the State University Groningen and informed consent was obtained from each patient. Ten consecutive patients (six males, four females) with coronary artery disease, New York Heart Association class III-IV, who were undergoing coronary artery surgery were studied. The exclusion criteria for surgery were: other sites of severe atherosclerotic disease, active peptic ulcer disease or a history of peptic ulcer disease, the use of gastric acid secretion inhibiting drugs, coagulation disturbances (thrombocyte count $< 50 \times 10^9$/l, thrombin time > 1.50 international reference unit), or cardiopulmonary failure. Prior to the operation, pulmonary and radial artery catheters were inserted for routine pressure monitoring (Uniflow, Baxter Critical Care Division, Irvine, Calif., USA) after calibration and zeroing to mid-chest level with the patient in the supine position. Additionally, a nasogastric tonometer (TRIP Gastric tonometer, Tonometrics, Bethesda, Md., USA) was inserted, and the gastric position was checked by fluoroscopy. Environmental losses of gastric gas were prevented by closing the lumen of the tonometer. Anesthesia was induced with midazolam, sufentanil, and pancuronium. Cardiopulmonary bypass was performed under hypothermia (29 ± 1 °C, mean ± SD) using non-pulsatile flow. All patients underwent coronary artery bypass grafting, with a mean of three (range 1 to 6) anastomoses; the time of aortic occlusion was 61 ± 24 min. Hydroxyethylstarch 10% was used as a plasma expander: 500 ± 250 ml was given during the operation and another 600 ± 150 ml in the first 24 h postoperatively.

The study started 3 h (range 2.5–4 h) after the end of surgery. The patients fasted throughout the study period. Each measurement period took 30 min and started with an infusion of 2.5 ml of saline into the tonometer balloon for a measurement interval of exactly 30 min. In period 1 (t = 0 to 30 min), baseline measurements were done. At the start of period 2 (t = 30 to 60 min), 500 mg sodium bicarbonate (6.25 mEq) dissolved in 50 ml water was introduced into the stomach through the tonometer lumen. At t = 60 min, 100 mg of the H_2 receptor antagonist ranitidine was administered intravenously to suppress gastric acid secretion. One hour later, measurements were repeated (period 3, t = 120 to 150 min). At the start of period 4 (t = 150 to 180 min), 500 mg bicarbonate was administered intragastrically again. Twenty-four hours later (day 1), the measurement sequence was repeated. At day 1, all patients were off the ventilator and the pulmonary artery catheters had been removed.

At the end of each measurement interval, the central venous pressure (CVP), pulmonary capillary wedge pressure (PCWP), heart rate (HR), and mean arterial blood pressure (MAP) were measured. Cardiac output (CO) was measured by the thermodilution technique, using the mean of triplicate injections of 10 ml saline at room temperature (Marquette Electronics, Milwaukee, Wis., USA). At the end of each measurement interval, all of the saline was aspirated from the tonometer; after 1 ml was discarded, representing the dead space volume, the remaining 1.5 ml was used for measurements in a blood gas analyzer. At the same time, an arterial blood sample was drawn from the radial artery and a gastric juice sample from the nasogastric tube. The gastric juice pH was determined using litmus paper [21]. Gasometric values in saline and blood samples were measured with a Radiometer type 330 blood gas analyzer (Radiometer, Copenhagen, Denmark) within 10 min of sampling. In the arterial blood sample, the pH (pHa) and PCO_2 ($PaCO_2$) were measured and the standard bicarbonate content was calculated; in the saline sample, the PCO_2 was measured ($iPCO_2$). The steady state $iPCO_2$ was calculated by multiplying the measured PCO_2 by 1.24, thereby correcting for incomplete equilibration after the 30 min measurement interval [22] and the 18% underestimation of PCO_2 measured in saline with this type of blood gas analyzer [23]. The pHi was calculated using a modified Henderson-Hasselbalch equation as previously described [1, 5]. The differences between pHi and pHa (ΔpH) and between $iPCO_2$ and $PaCO_2$ (ΔPCO_2) were calculated.

The hypothesis that bicarbonate and ranitidine changed the $iPCO_2$ was tested by analysis of variance for repeated measurements, and, since significance was achieved, by a paired Student's *t*-test. Changes in hemodynamic parameters, blood gas values, and gastric juice pH were evaluated by similar techniques. A $p < 0.05$ was considered statistically significant. Values are mean ± standard deviation.

Table 1 $iPCO_2$ and pHi in different study periods. Values are mean ± SD ($iPCO_2$ intramucosal PCO_2, ΔPCO_2 difference between intramucosal and arterial PCO_2, *pHi* gastric intramucosal pH, ΔpH difference between intramucosal and arterial pH)

Study period	Ranitidine	Bicarbonate	$iPCO_2$ (mm Hg)	ΔPCO_2 (mm Hg)	pHi	ΔpH
Day 0						
1	–	–	25 ± 5	–9 ± 5	7.47 ± 0.08	0.05 ± 0.06
2	–	+	31 ± 5	–2 ± 6	7.35 ± 0.07	–0.07 ± 0.06
3	+	–	29 ± 5	–4 ± 6	7.37 ± 0.08	–0.03 ± 0.09
4	+	+	31 ± 5	–5 ± 5	7.37 ± 0.06	–0.03 ± 0.06
Day 1						
1	–	–	32 ± 4	–4 ± 4	7.37 ± 0.06	–0.03 ± 0.05
2	–	+	56 ± 25*	20 ± 26*	7.16 ± 0.16*	–0.25 ± 0.15*
3	+	–	33 ± 4	–2 ± 4	7.35 ± 0.05	–0.06 ± 0.04
4	+	+	40 ± 14	5 ± 14	7.29 ± 0.11	–0.13 ± 0.10

* $p < 0.001$ versus period 1

Results

HR, MAP, and CVP did not change during the study periods. The mean HR for all periods was 79 ± 12 beats/min, for mean MAP 83 ± 7 mmHg, and mean CVP 8 ± 3 mmHg. The PCWP and CO on day 0 did not differ between periods and averaged 15 ± 4 mmHg and 6.1 ± 1.3 l/min, respectively. On day 1, CO and PCWP were not measured since the pulmonary catheter had been removed from all patients.

On day 0, basal gastric juice pH was 5.3 ± 1.4. Only one patient had a basal pH < 4. In the remaining periods, the pH was 6. On day 1 the basal pH was 4.9 ± 1.7. In three patients it was < 4. In periods 2 and 4, the pH was 6. In period 3, it was 5.7 ± 0.9. In one patient it remained below 4 despite administration of ranitidine.

The pHa remained unchanged during the study and averaged 7.41 ± 0.03 on days 0 and 1. The $PaCO_2$ on day 0 averaged 34 ± 4 mmHg compared to 36 ± 3 mmHg on day 1 ($p < 0.05$). The bicarbonate content averaged 20 ± 1 mmol/L on day 0, compared to 22 ± 1 mmol/L on day 1 ($p < 0.01$).

On day 0, $iPCO_2$ was unchanged despite bicarbonate administration and ranitidine infusion, and ranitidine did not have a significant effect either (Table 1). The basal $iPCO_2$ was below 47 mmHg (taken as the upper limit of normal) in all patients (Fig. 1). The ΔPCO_2 on day 0 did not differ among periods. It was above 5 mmHg (taken as the upper limit of normal) in only one of ten patients. On day 1, bicarbonate administration increased the $iPCO_2$ by 23 ± 22 mmHg ($p < 0.0001$) and the ΔPCO_2 by 25 ± 20 mmHg ($p < 0.0001$) over baseline values before, but not after, ranitidine infusion. The basal $iPCO_2$ was below 47 mmHg in nine of the ten patients. In one patient, administration of bicarbonate after ranitidine on day 1 increased $iPCO_2$ and ΔPCO_2 to 82 (Fig. 1) and 44 mmHg, respectively. In this patient, gastric juice pH was < 4 immediately prior to the bicarbonate dose.

The pattern of pHi and ΔpH in the various study periods was opposite to that observed in $iPCO_2$ and ΔPCO_2 (Table 1), but the absolute pHi and ΔpH in period 3 (the normal baseline period during acid suppression) was more often abnormal than the $iPCO_2$ and ΔPCO_2. The pHi was below normal (taken as < 7.35) in one patient on day 0 and in four others on day 1. The ΔpH was above normal (taken as > 0.05) in five patients on day 0 and in eight patients on day 1.

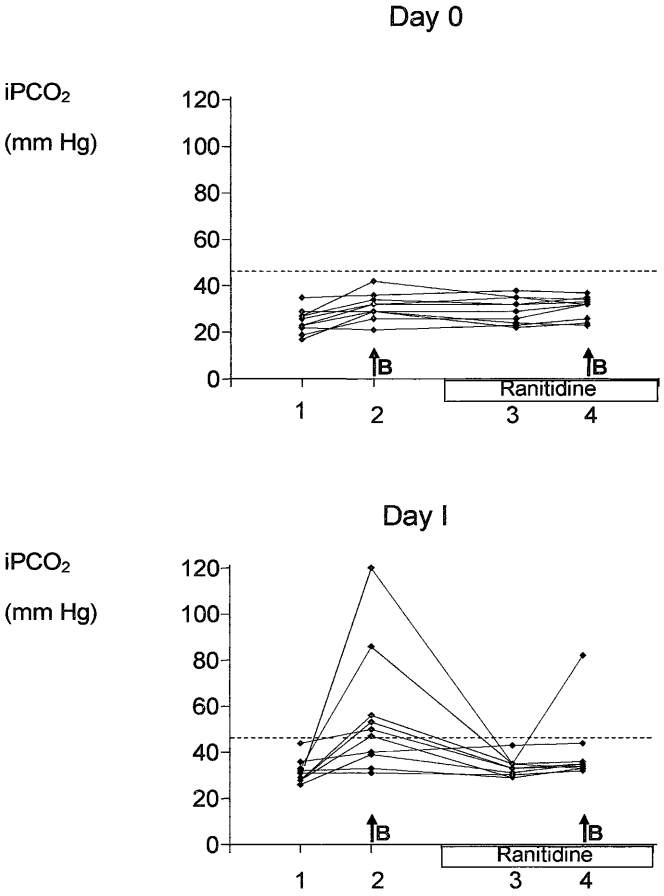

Fig. 1 The individual $iPCO_2$ in ten postoperative coronary artery surgery patients on days 0 and 1. Period *1* basal, *2* after oral dose of bicarbonate *B*, *3* after start of ranitidine infusion, *4* after oral dose of bicarbonate *B* following ranitidine infusion. The dotted line represents the upper level

Discussion

This study suggests that gastric acid secretion is diminished in the immediate postoperative period following coronary artery surgery, even in hemodynamically stable patients. The return of acid secretion was on day 1 was associated with a marked increase in $iPCO_2$ and a decrease in pHi following oral bicarbonate administration, which prevented by prior administration of H_2 receptor blocking agents. The artifactually elevated PCO_2 decreases the specificity of gastric tonometry for mucosal hypoperfusion.

A similar protocol was followed in healthy volunteers [7]. Gastric acid output on day 1 in our coronary artery surgery patients seems lower than the acid secretion in healthy volunteers [7], as judged from the higher gastric juice pH and the lower $iPCO_2$ increase with intragastric bicarbonate in our patients. The gastric juice pH is a measure of the proton concentration per se but not of the amount of gastric acid [24]. The $iPCO_2$ increase after the bicarbonate dose, however, is a function of the total amount of protons present in the stomach, since the bicarbonate will neutralize gastric acid by generating CO_2, which subsequently diffuses into the tonometer balloon. Thus, administration of 500 mg bicarbonate results in back-titration of gastric acid, which can be measured as an increase in $iPCO_2$.

In six of our ten patients, the $iPCO_2$ increased to supranormal levels after the bicarbonate dose on day 1, with a mean increase of 20 mmHg. Three of these six patients had a basal gastric juice pH < 4. In healthy volunteers we found a basal pH < 4 but an $iPCO_2$ increase (by a mean of 81 mmHg) to supranormal levels in all ten patients [7]. Therefore, gastric acid secretion is diminished after coronary artery surgery and seems to return in 60 % of patients by day 1, albeit not to levels observed in healthy volunteers.

The efficacy of ranitidine administration in preventing an elevated $iPCO_2$ following an intragastric dose of bicarbonate is in accordance with studies in healthy volunteers [6–8]. However, the dose of bicarbonate after the ranitidine infusion still caused an increase in $iPCO_2$ in one patient on day 1, who had a basal gastric juice pH of 3, which remained at 3 after ranitidine infusion. This may be explained by a time lag between the infusion of ranitidine and the rise in gastric juice pH, which may sometimes exceed 60 min or by an individual variation in response to ranitidine [25]. The need for gastric acid secretion suppression for reliable tonometry has been questioned before [8], even though the $iPCO_2$ was lower and the pHi was higher in healthy volunteers after administration of H_2-blockers, suggesting that, even at rest, intragastric buffering of acid by non-parietal cell-derived bicarbonate may spuriously elevate intraluminal PCO_2 independently of $iPCO_2$ [6–8]. The current study indicates that, even if gastric acid and perhaps bicarbonate secretion are inhibited in critically ill patients, H_2-blockers should be administered prior to $iPCO_2$ tonometry to achieve reliability. Our findings would not only apply to conventional saline tonometry at hospitals where semicontinuous air tonometry (a new and apparently accurate automated technique [26] which circumvents many sources of error inherent in manual saline tonometry) is not yet available, but also to air tonometry itself. A transient $iPCO_2$ increase during air tonometry could result, in the absence of H_2-blockade, from duodenogastric reflux and from transient gastric mucosal hypoperfusion, supporting the use of H_2-blockers prior to air tonometry as well, in order to eliminate CO_2 generation via the former mechanism.

A discrepancy was found between the presumed prevalence of gastric mucosal hypoperfusion based on ΔpH or ΔPCO_2. The ΔpH was supranormal in 50 % of patients on day 0, suggesting hypoperfusion, which is in accordance with previous studies showing a transient decrease in pHi in the first day after cardiopulmonary bypass surgery [14–20]. However, the ΔPCO_2 was supranormal in only 10 % of patients on day 0. If it is indeed true that the ΔPCO_2 is a more specific indicator of gastric mucosal hypoperfusion than the pHi and ΔpH, this may indicate that the prevalence of mucosal hypoperfusion following cardiac surgery may have been overestimated in previous reports [5]. The disparity between ΔPCO_2 and ΔpH was caused by a decrease in arterial bicarbonate from about 25 mmol/l before surgery to 20 mmol/l at the end of surgery. It has been shown previously that rapid changes in the blood bicarbonate content result in spurious pHi changes, since changes in blood bicarbonate may not parallel changes in mucosal bicarbonate content [27, 28]. That the ΔPCO_2 may be a more specific indicator of mucosal hypoperfusion is supported by the uneventful recovery in our patients, suggesting the absence of hypoperfusion, since a low pHi has been associated with adverse events after cardiopulmonary bypass surgery [1–5, 15–20]. Second, among the six subjects in whom gastric acid secretion had resumed, four showed an abnormal ΔpH and two an abnormal pHi, whereas none showed an abnormal $iPCO_2$ or ΔPCO_2. It is unlikely that these subjects had gastric mucosal hypoperfusion, in the absence of complete anacidity [10, 11, 29]. Nevertheless, more studies are needed to evaluate the value of ΔPCO_2 and ΔpH over the pHi in detecting gastrointestinal hypoperfusion [15, 16, 19]. Otherwise, the ΔPCO_2 was negative at times, contrary to theoretical expectation [5], so that we cannot exclude a slight but systematic underestimation of the $iPCO_2$. The cause of transiently diminished gastric acid secretion after surgery is not clear from this study, in the absence of overt tonometric signs of gastric hypoperfusion. We cannot, however, exclude the possibility that gastric hypoperfusion may have occurred during the operation, in the absence of intraoperative $iPCO_2$ and pHi measurements.

We are not aware of any studies of the time course of parietal cell recovery after hypoperfusion. Therefore, we cannot overlook the possibility that the transiently diminished gastric acid secretion after the operation was caused by gastric hypoperfusion during the operation.

In conclusion, the results of the current study suggest that H_2 receptor blockade is mandatory for reliable $iPCO_2$ tonometry after cardiopulmonary bypass surgery.

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