Esophageal \( \text{PCO}_2 \) as a monitor of perfusion failure during hemorrhagic shock

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Esophageal \( \text{PCO}_2 \) as a monitor of perfusion failure during hemorrhagic shock. J. Appl. Physiol. 82(2): 558–562, 1997.—Measurement of gastric wall \( \text{PCO}_2 \) (\( \text{PgCO}_2 \)) by tonometric method has emerged as an attractive option for estimating visceral perfusion during circulatory shock. However, gastric acid secretion obscures the tonometric measurement. We, therefore, investigated the option of measuring \( \text{PCO}_2 \) in the esophagus to minimize these restraints. Hemorrhagic shock was induced in five Sprague-Dawley rats, and five rats served as sham controls. \( \text{PgCO}_2 \) was measured with an ion-sensitive field effect transistor that was surgically implanted into the gastric wall. Esophageal luminal \( \text{PCO}_2 \) (\( \text{PeCO}_2 \)) was measured by a second ion-sensitive field effect transistor sensor. During hemorrhagic shock, mean aortic pressure declined from 150 to 50 mmHg. Gastric blood flow decreased from 58 to 12 ml·min\(^{-1}\)·100 g\(^{-1}\) (21% of preshock) and esophageal blood flow from 44 to 7 ml·min\(^{-1}\)·100 g\(^{-1}\) (16% of preshock). \( \text{PgCO}_2 \) simultaneously increased from 47 to 116 Torr and \( \text{PeCO}_2 \) from 47 to 127 Torr. The increases in \( \text{PgCO}_2 \) were highly correlated with increases in \( \text{PeCO}_2 \) (\( r = 0.90 \)). Esophageal tonometry may, therefore, serve as a practical alternative to gastric tonometry.

Measurement of gastric wall \( \text{PCO}_2 \) has emerged as an attractive option for estimating gastrointestinal ischemia during circulatory shock states (3, 20). Because \( \text{CO}_2 \) freely diffuses from gastric wall to gastric lumen, gastric wall \( \text{PCO}_2 \) may be estimated from measurements of gastric luminal \( \text{PCO}_2 \) by utilizing a gastric tonometer (7, 8, 15, 16). However, the \( \text{PCO}_2 \) of gastric juice may in some instances exceed that of the gastric wall and of gastric venous blood (19). These excesses of \( \text{PCO}_2 \) are generated from the action of acid gastric juice on bicarbonate contained either in the gastric juice itself or in the backflow of alkaline duodenal contents. The \( \text{CO}_2 \) so generated also backdiffuses into the gastric mucosa, which may further increase tissue \( \text{PCO}_2 \) independently of changes in mucosal blood flow (19). After \( \text{H}_2 \)-receptor blockade by cimetidine, \( \text{H}^+ \) production by the stomach is curtailed and the \( \text{PCO}_2 \) of gastric luminal fluid and that of gastric venous blood are approximately the same (11, 19). Accordingly, \( \text{H}_2 \)-receptor blockade is recommended as a routine to ensure reliability of conventional gastric tonometry (9, 11). Although \( \text{H}_2 \)-receptor blockade was previously a routine for prevention of stress ulceration in critically ill and injured patients, adverse effects, including increased risks of nosocomial pneumonia, prompted its more restrained use (4).

These limitations of gastric tonometry prompted our search for alternative sites for the measurement of visceral \( \text{PCO}_2 \). Initial trials in pigs demonstrated that there were significant increases in esophageal wall \( \text{PCO}_2 \) during hemorrhagic shock when these were directly measured with an ion-sensitive field-effect transistor (ISFET) \( \text{PCO}_2 \) sensor. Accordingly, we were attracted to the possibility that the incorporation of an ISFET or comparable \( \text{PCO}_2 \) sensor in the esophageal portion of the conventional gastric tube may serve as a competent monitor of visceral ischemia and, in turn, of the severity of perfusion failure (shock). Such esophageal measurements would potentially obviate the need for \( \text{H}_2 \)-receptor blockade. The present study was, therefore, undertaken to examine the relationship between gastric wall and esophageal luminal \( \text{PCO}_2 \) before, during, and after reversal of hemorrhagic shock.

METHODS

The experiments were performed in an established rodent model of hemorrhagic shock (15, 20–22). All animals received humane care in compliance with the Principles of Laboratory Animal Care formulated by the National Society for Medical Research and the National Institutes of Health (NIH) Guide for the Care and Use of Laboratory Animals (DHHS Publication No. 86-33, Revised 1985, Office of Science and Health Reports, Bethesda, MD 00892). Animal preparation. Ten Sprague-Dawley rats, weighing between 450 and 550 g, were fasted overnight except for free access to water. The animals were anesthetized by intraperitoneal injection of 45 mg/kg pentobarbital sodium supplemented with additional doses of 10 mg/kg at hourly intervals as needed. The trachea was surgically exposed at a site 2 cm caudal to the larynx. A 14-gauge cannula (Quick-Cath, Vicra Division, Travenol Laboratories, Dallas, TX) was then advanced into the trachea for a distance of 1 cm. Animals were breathing room air spontaneously. Through the left external jugular vein, a polyethylene catheter (PE-50; Becton-Dickinson, Franklin Lakes, N J) was advanced into the right atrium for measurement of right atrial pressures. This catheter was also used as a site for injection of indicator for measurement of cardiac output. Through the left carotid artery, a polyethylene catheter was advanced into the ascending aorta for measurement of aortic pressures and for sampling aortic blood. Aortic and right atrial pressures were measured with reference to the midchest with high-sensitivity transducers (model 42584–01, Abbott Critical Care Systems, North Chicago, IL). Through the left femoral artery and left femoral vein, polyethylene catheters were advanced into the thoracic aorta and into the inferior vena cava. The left femoral arterial...
catheter was connected to the barrel of a standard 20-ml plastic syringe that served as a reservoir for shed blood (21, 22). The left femoral vein catheter served as a source of sampling venous blood and as a site for transfusion of blood. For measurement of cardiac output, a 1.5-Fr thermodilution microcatheter (model 9030-12-D-30, Columbus Instruments, Columbus, OH) was advanced into the thoracic aorta through the right femoral artery by methods previously documented (2, 3, 17). Blood temperature was measured with this sensor and maintained between 36.5 and 37.5°C by utilizing infrared heating lamps.

For continuous measurement of esophageal luminal P\(_{\text{CO}}\)\(_2\), a 5-Fr cannula was advanced from the mouth into the proximal esophagus. An ISFET sensor (model CO-1035, Nihon Kohden, Tokyo, Japan) was then advanced through the esophageal cannula for a distance of 12.5 cm into the lower esophagus such that the tip of the sensor was positioned 1.5 cm proximal to the gastroesophageal junction. The cannula was then removed. A surgical incision was then made in the upper abdomen, and the stomach was exposed. An additional ISFET sensor was imbedded in the anterior wall of the gastric corpus to a depth of 5 mm for continuous measurement of gastric wall P\(_{\text{CO}}\)\(_2\), as previously described (3, 15, 20). This allowed for measurement of gastric wall P\(_{\text{CO}}\)\(_2\) without interference by the gastric secretions in the lumen of the stomach. The abdomen was then closed in one layer. The validity of ISFET measurements and comparisons with conventional gastric balloon tonometry have been previously reported (2, 15).

A conventional lead II electrocardiogram was continuously recorded, utilizing subcutaneous needle electrodes. Catheters were flushed intermittently with physiological salt solution containing 2.5 IU/ml of crystalline bovine heparin.

For measurement of the regional organ blood flow by utilizing the colored-microspheres technique, an additional polyethylene catheter was advanced through the right carotid artery into the left ventricle in an additional five animals, guided by the morphology of the pressure pulses during transit from the aorta to the left ventricle. This catheter served as the injection site of colored microspheres. For withdrawal of reference blood, an additional polyethylene catheter was advanced through the right femoral artery into the thoracic aorta in lieu of the thermodilution microcatheter.

Experimental procedure. The animals were randomized to serve as either hemorrhagic shock or sham controls by the sealed-envelope method. Blood from the left femoral arterial catheter was allowed to flow into the barrel of a 20-ml syringe serving as a plastic reservoir. The reservoir was pressurized to 80 mmHg for 10 min, 70 mmHg for 20 min, 60 mmHg for 20 min, and 50 mmHg for the ensuing 70 min. The reservoir pressure was controlled with a special device consisting of a piston that was manually adjusted with a lead screw, a bleeding valve, and a mercury manometer allowing for fine adjustments. After 2 h, the pressure in the reservoir was increased to 200 mmHg such that the blood in the reservoir was reintroduced over an interval of 10 min. Control animals were identical treated, except that no blood was allowed to flow from the femoral arterial catheter into the reservoir. An autopsy was routinely performed in all animals, with gross inspection of thoracic and abdominal organs to identify adverse effects of the interventions.

Organ blood flow was measured with an adaptation of the colored-microspheres technique as previously described by our group and by others (5, 10, 20). Approximately 5 x 10^5 colored microspheres with mean diameter of 15 ± 2 µm labeled with blue, orange, green, or red (E-Z TRAC, Los Angeles, CA) were suspended in 0.1 ml of normal saline and injected into the left ventricle of the five additional animals over an interval of 15 s. Measurements of organ blood flow were obtained before, 60 and 120 min after the start of hemorrhage, and 60 min after reinfusion of shed blood. The reference blood was withdrawn from the thoracic aorta with a syringe pump (model 940, Harvard Apparatus, South Natick, MA) at a rate of 1 ml/min over an interval of 4 min, beginning 10 s before microsphere injection. An equal amount of donor blood was simultaneously infused into the inferior vena cava by utilizing the same syringe pump.

Both the esophagus and stomach were harvested at the end of each experiment. The wet tissue weight of each was measured with an optical balance (MAGNI-GRAD type 21, Ainsworth & Sons, Denver, CO). The organs were digested by alkaline hydrolysis in a heated water bath maintained at 50°C for 18 h. The suspension was then centrifuged at 3,000 revolutions/min for 30 min with a standard centrifuge (MARA-THON 21K, Fisher Scientific, Pittsburgh, PA). The sediment containing microspheres was resuspended in the counting reagent supplied by E-Z TRAC and重新 centrifuged for 15 min. This sediment was then resuspended in the counting reagent such that the total volume was 0.3 ml. Aliquots of this suspension were then placed into a standard hemocytometer chamber for counting. The same procedures were applied to the reference samples of aortic blood.

Measurements. The electrocardiogram, aortic and right atrial pressures, gastric wall P\(_{\text{CO}}\)\(_2\), and esophageal luminal P\(_{\text{CO}}\)\(_2\) were continuously recorded with the aid of a computer (Deskpro 286, Compaq, Houston, TX) utilizing data-acquisition hardware and software (DATAQ Instruments, Akron, OH). Cardiac output was measured by the thermodilution technique after bolus injection of 200 µl of saline indicator maintained at a temperature of 15°C into the right atrium as previously described (2, 3, 17), and cardiac index was computed with the aid of a cardiac output computer (model CO-100, Institute of Critical Care Medicine, Palm Springs, CA). Aortic and right atrial blood pH, P\(_{\text{CO}}\)\(_2\), P\(_{\text{O}}\)\(_2\), and hemoglobin saturation were measured on a 0.7-ml sample of blood with the aid of an automated blood-gas analyzer and a CO-oximeter (models IL-1306 and IL-202, respectively, Instrumentation Laboratory, Lexington, MA). Aortic blood lactate concentrations were measured from the same sample with an electrode-based lactate analyzer (model 23L, Yellow Springs Instruments, Yellow Springs, OH). Measurements were obtained before hemorrhage (baseline measurements) and at 30, 60, 120, 150, and 180 min after the start of hemorrhage. After each sample blood had been withdrawn, an equal amount of blood from an anesthetized donor from the same litter was infused into the inferior vena cava.

Organ blood flows were computed as follows

\[
\dot{Q}_o (\text{ml/min}) = \frac{\text{Cr}_o \cdot \dot{Q}_{bw}}{\text{Cr}_b} \tag{1}
\]

in which \(\dot{Q}_o\) is organ blood flow, \(\text{Cr}_o\) is total microspheres in the organ, \(\dot{Q}_{bw}\) is flow of blood during withdrawal from the aorta (ml/min), and \(\text{Cr}_b\) is total counts of blood withdrawn from the aorta

\[
\dot{Q}_{ow} (\text{ml} \cdot \text{min}^{-1} \cdot 100 \text{ g}^{-1}) = \frac{\dot{Q}_o - 100}{\text{organ weight (g)}} \tag{2}
\]

in which \(\dot{Q}_{ow}\) is organ blood flow per 100 g of tissue.

Statistical analyses. Measurements are reported as means ± SD. Time-based measurements within groups were compared by analysis of variance repeated measurements. Time-coincident measurements of gastric wall P\(_{\text{CO}}\)\(_2\) and
Acid-base and metabolic measurements before, during, and after reversal of hemorrhagic shock

Baseline measurements of mean aortic pressure, cardiac index, aortic and right atrial blood pH, PaO₂, and PaCO₂ were examined for each animal by utilizing linear regression analysis, and the r values so obtained were averaged. A P value of <0.05 was regarded as significant.

RESULTS

Baseline measurements of mean aortic pressure, cardiac index, and blood pH were within the physiological ranges previously reported (2, 3, 15, 20). During the 120-min interval of hemorrhage, the mean aortic pressure decreased from an average of 150 to 51 mmHg and the cardiac index from 295 to 60 ml·min⁻¹·100 g⁻¹ (Fig. 1). These hemodynamic changes were accompanied by increases in aortic lactate concentration from 0.5 to 10.7 mmol/l and in arteriovenous gradients of PaCO₂ from 5 to 23 Torr (Table 1). Reinfusion of shed blood restored mean aortic pressure and cardiac index to ~85% of baseline values. Concurrently, arterial lactate and arteriovenous gradients of CO₂ declined.

During hemorrhage, gastric wall PaCO₂ increased from 47 to 116 Torr and esophageal luminal PaCO₂ increased from 47 to 127 Torr (Fig. 2). The differences between the two measurements were not significant. Gastric blood flow decreased from 58 to 20 ml·min⁻¹·100 g⁻¹ after 60 min of bleeding and to 12 ml·min⁻¹·100 g⁻¹ at the end of the 120-min interval before reinfusion (Table 2). There were corresponding decreases in esophageal blood flow from 44 to 12 ml·min⁻¹·100 g⁻¹ at 60 min and to 7 ml·min⁻¹·100 g⁻¹ at 120 min. Within 20 min after the start of reinfusion, both gastric wall and esophageal luminal PaCO₂ returned to baseline levels. The measurement of esophageal luminal PaCO₂ was highly correlated with that of gastric wall PaCO₂ for individual animals and yielded an r that ranged from

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Group</th>
<th>Baseline</th>
<th>Hemorrhage</th>
<th>Reinfusion</th>
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<tbody>
<tr>
<td>pH, units</td>
<td>C</td>
<td>7.40 ± 0.03</td>
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<td>H</td>
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<td>7.48 ± 0.10</td>
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<td>pH, units</td>
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<td>7.42 ± 0.03</td>
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<tr>
<td></td>
<td>H</td>
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<td>7.46 ± 0.06</td>
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<td>84 ± 2</td>
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<tr>
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<td>H</td>
<td>91 ± 6</td>
<td>115 ± 19*</td>
<td>123 ± 16*</td>
</tr>
<tr>
<td>PaO₂, Torr</td>
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<td>53 ± 7</td>
<td>47 ± 9</td>
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<td></td>
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<td>25 ± 5†</td>
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<td>41 ± 3</td>
<td>39 ± 4</td>
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<td>H</td>
<td>37 ± 4</td>
<td>28 ± 10*</td>
<td>20 ± 5†</td>
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<td>SvO₂, %</td>
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<td>43 ± 3</td>
<td>40 ± 5</td>
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<td></td>
<td>H</td>
<td>42 ± 4</td>
<td>33 ± 7†</td>
<td>29 ± 3†</td>
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<td>SvO₂, %</td>
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<td>92 ± 9</td>
<td>90 ± 1</td>
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<td>H</td>
<td>93 ± 3</td>
<td>95 ± 1*</td>
<td>96 ± 2*</td>
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<td>Lactate, mmol/l</td>
<td>C</td>
<td>0.5 ± 0.2</td>
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<td></td>
<td>H</td>
<td>0.5 ± 0.1</td>
<td>3.8 ± 1.5†</td>
<td>7.0 ± 1.1†</td>
</tr>
</tbody>
</table>

Values are means ± SD. C, control; H, hemorrhage; pH, arterial pH; pHe, venous pH; PaO₂, arterial PaO₂; PaCO₂, venous PaCO₂; PaO₂, arterial PaCO₂; Pvo₂, venous Pvo₂; Svo₂, venous O₂ saturation; SvO₂, venous O₂ saturation. *P < 0.05, **P < 0.01 vs. control.

![Fig. 1. Mean aortic pressure (MAP) and cardiac index (CI) before, during, and after hemorrhage. Values are means ± SD; n = 5 rats in each group. BL, baseline. **P < 0.01 vs. control](image1.png)

![Fig. 2. Comparison of changes in gastric wall PaCO₂ (PgCO₂) and esophageal luminal PaCO₂ (PeCO₂) during hemorrhagic shock. Values are means ± SD; n = 5 rats in each group. *P < 0.05, **P < 0.01 vs. BL](image2.png)
PCO$_2$ nor esophageal luminal PCO$_2$ was altered during anesthetized control animals. Neither gastric wall preshock level at 60 min after reinfusion of shed blood. Gastric and esophageal blood flow returned to and sepsis (3, 14, 15, 20). Gastric luminal PCO$_2$ measured with an implanted ISFET during hemorrhage were closely related to the decreases in aortic pressure, cardiac output, and gastric and esophageal blood flows. Hemorrhagic shock was characterized by increases in venoarterial PCO$_2$ and arterial lactate, changes that accompanied the decreases in cardiac output and oxygen delivery characteristic of hemorrhagic shock. The esophagus, therefore, was shown to be an appropriate alternative site in lieu of the gastric luminal site for measurements of visceral PCO$_2$ during hemorrhagic shock. The ISFET technology, which allowed for continuous measurement and display, had the additional benefit of rapidity and ease of measurement. It obviated the need for a gastric balloon from which saline is aspirated after a 60- to 90-min time interval for equilibration. It also obviated the need for simultaneous sampling of arterial blood for in vivo measurements on the aspirated saline and on the arterial blood (8).

Blood flow to the gastrointestinal viscera is sharply and disproportionately reduced during the low-flow states of hemorrhagic and obstructive shock (14, 18). Accordingly, the PCO$_2$ values of the organs perfused by the splanchnic circulation and especially the stomach are targeted as appropriate indicators of the adequacy of blood flow for measuring aerobic metabolism in these organs. To that extent, the gastrointestinal tract has been heralded as a canary of the body because canaries are used as the traditional monitors of hypoxia due to carbon monoxide intoxication in coal mining (1). Because the esophagus is primarily supplied by the systemic rather than by the splanchnic circuit, it was not targeted as a tissue PCO$_2$ monitor.

Because increases in esophageal PCO$_2$ were as great as those of the gastric wall in the present study, the question arose as to whether perfusion of the esophagus was comparably reduced. The earlier assumptions notwithstanding, such was the case. Accordingly, both increases in tissue PCO$_2$ and decreases in blood flow were approximately the same in the stomach and in the esophagus. Because the rapidity of bleeding and the severity of hemorrhagic shock were profound in the studies herein reported, we do not exclude the possibility that this close relationship in gastric and esophageal PCO$_2$ may not apply under conditions of lesser severity in other settings such as septic shock.

We previously demonstrated that ischemia of perfusion failure, which occurs early in the viscera, represents a dual phenomenon of tissue oxygen deficits and CO$_2$ excesses (12). This tissue hypercarbia is best explained by buffering of excesses of hydrogen ion by bicarbonate. The excess hydrogen ions are traced to anaerobically generated lactic acid and degeneration of high-energy phosphate compounds (13). This may also be related to delayed washout of metabolites during the low-flow states of circulatory shock (15, 20).

Initially, blood gases were measured on lightly anesthetized animals breathing room air spontaneously. Under these conditions, there were borderline decreases in arterial Po$_2$ and borderline increases in arterial PCO$_2$, compared with mechanically ventilated animals (15). However, these decreases did not alter baseline tissue PCO$_2$, nor was there an increase in gastric wall or esophageal luminal PCO$_2$ in control animals over the 3-h interval of measurements.

In the practical application of the esophageal luminal PCO$_2$ measurement, we do not exclude the possibility that either CO$_2$ generated in the gastric lumen or reflux of gastric juice into the esophagus may also alter the esophageal measurement. However, these factors were not in evidence in the course of the present studies during which gastric acid production was uninhibited.

<table>
<thead>
<tr>
<th>Organ</th>
<th>Baseline 60 min</th>
<th>Hemorrhage 60 min</th>
<th>Baseline 120 min</th>
<th>Hemorrhage 120 min</th>
<th>Baseline 180 min</th>
<th>Hemorrhage 180 min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stomach, ml·min$^{-1}$·100 g$^{-1}$</td>
<td>58 ± 18 (100)</td>
<td>20 ± 10† (34 ± 7)</td>
<td>12 ± 5 (21 ± 4)</td>
<td>36 ± 11† (62 ± 10)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Esophagus, ml·min$^{-1}$·100 g$^{-1}$</td>
<td>44 ± 17 (100)</td>
<td>12 ± 7† (27 ± 6)</td>
<td>7 ± 3† (16 ± 4)</td>
<td>26 ± 7† (59 ± 12)</td>
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</tbody>
</table>

Values are means ± SD; nos. in parentheses are percentages.*P < 0.05, †P < 0.01 vs. baseline.

DISCUSSION

Earlier studies demonstrated that increases in gastric wall PCO$_2$ serve as quantitators of onset and severity of perfusion failure during circulatory shock of diverse causes, including hemorrhage, anaphylaxis, and sepsis (3, 14, 15, 20). Gastric luminal PCO$_2$ measured with the conventional balloon tonometer underestimated relatively rapid increases in gastric wall PCO$_2$ measured with an implanted ISFET during hemorrhagic shock (15). Accordingly, the direct measurement of gastric wall PCO$_2$ with an implanted ISFET had greater reliability and sensitivity (15). We, therefore, utilized the implanted ISFET as a more stringent standard for comparison with esophageal luminal PCO$_2$.

The esophageal luminal PCO$_2$ corresponded closely to gastric wall PCO$_2$ (r = 0.90; P < 0.0001). This contrasted with an earlier observation in which gastric wall PCO$_2$ and tonometrically measured gastric luminal PCO$_2$ in this model yielded a lower correlation (r = 0.71; P < 0.01) (15). After acid secretion was blocked with ranitidine, the correlation increased only to 0.80 (P < 0.01) (15). The observed increases in both esophageal and gastric wall PCO$_2$ during hemorrhage were closely related to the decreases in aortic pressure, cardiac output, and gastric and esophageal blood flows. Hemorrhagic shock was characterized by increases in venoarterial PCO$_2$ and arterial lactate, changes that accompanied the decreases in cardiac output and oxygen delivery characteristic of hemorrhagic shock. The esophagus, therefore, was shown to be an appropriate alternative site in lieu of the gastric luminal site for measurements of visceral PCO$_2$ during hemorrhagic shock. The ISFET technology, which allowed for continuous measurement and display, had the additional benefit of rapidity and ease of measurement. It obviated the need for a gastric balloon from which saline is aspirated after a 60- to 90-min time interval for equilibration. It also obviated the need for simultaneous sampling of arterial blood for in vivo measurements on the aspirated saline and on the arterial blood (8).
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