The splanchnic circulation contains about 30% of total blood volume and receives about 30% of the cardiac output. These proportions reveal the great influence the splanchnic vasculature and its regulation have on the systemic vascular behaviour in normal physiological conditions and even more so in states of haemodynamic instability and shock [1].

In fact, a typical response of the splanchnic circulation to different situations, characterised by hypovolaemic and/or low cardiac output states, is an intense vascular spasm disproportionate to that of the systemic vasculature. This vasoconstriction results in an increase of mesenteric arterial resistance and a decrease in mesenteric flow two to five fold greater than that in the peripheral circulation. These changes reflect selective splanchnic vasospasm.

This profound splanchnic vasoconstriction is mediated by an increase in sympathetic nervous system activity, vasopressin and activation of the renin-angiotensin system [2, 3].

The distinctive behaviour of splanchnic circulation is also characterised by its precocity. Splanchnic vasospasm is one of the first phenomena that can be seen when hypovolaemic or cardiogenic shock occurs. In septic states, although pin an early charge, the behaviour of the splanchnic vasculature is quite different. Vasodilatation and increase in blood flow are common findings. However, even though blood flow to the whole intestine may be increased, mucosal flow is in most cases impaired with subsequent mucosal ischaemia [4].

As has already been stated, intestinal ischaemia is a precocious manifestation of different forms of haemodynamic disturbances. Some authors have christened this phenomenon as the “canary” of the body (D. Dantzker), resembling the employment of canaries in coal mines to detect toxic gases before any harm could occur to miners. Therefore, detection of intestinal ischaemia would be of great importance in prognosis and probably in treatment in the critical care setting.

Measurement of pH$i$

Gastric tonometry is a method designed to monitor gut mucosa oxygenation status.
Experiments published several years ago [5, 6] showed that gas tension in the lumen of a hollow viscus (alimentary tract, gall bladder, and urinary bladder) is the same as that in the superficial layers of their mucosa. This knowledge was employed for the first time in 1959 by Boda et al. [7]. The authors used gastric tonometry to estimate arterial PCO₂, measuring PCO₂ in saline maintained for a certain equilibration period in a gastric balloon. They found a close correlation between values of gastric PCO₂ and end tidal PCO₂ with the exception of the values in patients with “haemodynamic instability”!% In them, tonometered PCO₂ was much higher than end tidal PCO₂ values.

Based upon information available [8, 9], it can be considered that PCO₂ in gastric air or fluid is in equilibrium with PCO₂ in the internal lining of the stomach (gastric mucosa). If luminal PCO₂ is measured by tonometry and mucosal bicarbonate concentration estimated (assuming that bicarbonate concentrations in mucosa and arterial blood are the same), the intramucosal pH (pHi) can be calculated using the Henderson-Hasselbalch equation.

To measure gastric tonometered PCO₂ (PtCO₂), a gastric tonometer is necessary. Although gastric juice has been used [10] further validation is needed. A device commonly used consists of a modified nasogastric tube (TRIP-NGS catheter, Tonometrics Inc, Worcester, MA, USA). It has two common sumping ports and a third one, which is connected through a gas impermeable tube to a silicone balloon placed on the end of the nasogastric device. This third port allows the balloon to be filled with saline solution and for samples to be taken for PtCO₂ measurements. The balloon is gas permeable and allows a time dependent equilibration of PCO₂ between saline solution and surrounding tissue and fluid.

The first step towards getting reliable pHi measurements is the X-ray confirmation of the balloon’s position in the lumen of the stomach. Then, 2.5 cc of 0.9% saline solution should be injected into the tonometer. After an equilibration period (not less than 20 minutes), the saline solution is sampled, having discarded the first 1 cc that fills the dead space of the tube. The PCO₂ of saline sampled is measured in a blood gas machine together with a simultaneous arterial blood sample. Finally, pHi is calculated as follows:

\[
pHi = 6.1 + \log \left( \frac{\text{arterial }{\text{HCO}_3^-}}{(F \times 0.03 \times \text{tonometered saline PCO}_2)} \right) 
\]

(Where F is a time dependent factor for fully or even partially equilibrated samples and is provided by the manufacturer).

One theoretical concern about the tonometric pHi calculation is the assumption that bicarbonate concentration in gastric mucosa is in equilibrium with bicarbonate concentration in arterial blood. However, the equilibrium is dependent on dynamic conditions and concentration differences could be related to the rate of change. For instance, fast administration of sodium bicarbonate could invalidate tonometric pHi calculation because bicarbonate equilibration between blood and gastric mucosa takes more time than that employed to make the measurement [11-13].