Increasing perfusion pressure does not improve gastric tissue blood flow after experimental gastric tube reconstruction

Leakage and stenosis of a gastro-oesophageal anastomosis are important complications of oesophagectomy with gastric tube reconstruction. Insufficient local blood flow is considered a major contributing factor. In the current study, the authors tested the hypothesis that increasing perfusion pressure could improve gastric tube blood flow.

Microvascular blood flow was measured by laser speckle imaging. Additionally, videothermography was used as a second, although indirect, measure of tissue blood flow. All experiments were performed in properly volume resuscitated pigs. Because of important differences in anatomy, a gastric pull-up with restoration of continuity in the neck was not performed and all measurements were made with the gastric tube positioned on top of the abdominal content. Blood flow was measured at the top of the gastric tube, at the virtual anastomotic site, at the medial part and at the base. Mean arterial blood pressure was increased in six steps from 50 to 110 mm Hg using norepinephrine.

During the increase in MAP, all other haemodynamic parameters remained constant. Blood flow and temperature were always lower at the top than in the medial part and base of the gastric tube. Increasing MAP did not increase blood flow or temperature in any part of the gastric tube. As expected, blood flow and temperature were strongly correlated ($r = 0.84$).

This is a properly executed study, investigating an important clinical problem. The study shows that increasing perfusion pressure does not result in an increase in blood flow at the anastomotic site of the gastric tube. Importantly, this study also suggests that the use of vasopressors does not jeopardise gastric tube blood flow if the subject is properly fluid loaded. Increasing MAP to supranormal levels is unlikely to prevent anastomotic leakage after oesophagectomy.


The effect of sildenafil in patients with ARDS

Acute Respiratory Distress Syndrome (ARDS) is characterized by pulmonary oedema due to increased vascular permeability, reduced compliance and pulmonary hypertension. Inhaled nitric oxide (NO) is frequently used as a rescue therapy to decrease pulmonary vascular resistance and improve ventilation-perfusion matching, thereby increasing oxygenation. Inhaled NO does not improve mortality and may increase the incidence of renal failure. Cornet et al. investigated the effects of an alternative pulmonary vasodilator sildenafil, a selective phosphodiesterase (PDE)-5 inhibitor.

The authors included 10 patients within one week after establishing the diagnosis ARDS. Diagnosis was based on consensus criteria. A pulmonary artery catheter was inserted in all patients. All patients received one tablet of 50 mg sildenafil orally. Haemodynamic and respiratory parameters were measured at baseline and 30, 60, 90, 120, 150, 180, 210, 240 and 360 minutes after administration. Sildenafil and desmethylsildenafil levels were measured using liquid-gas chromatography mass-spectrometry. MPAP decreased after 30 minutes from 25 to 22 mmHg. This effect was not selective for the pulmonary circulation as the MAP also decreased from 81 to 75 mmHg. Cardiac index did not change over time. Sildenafil tended to worsen oxygenation after 30 followed by a slow return to baseline levels. Shunt fraction significantly increased after 30 minutes from 24 to 31%. Maximal levels of sildenafil and desmethylsildenafil did not correlate with hemodynamic or oxygenation parameters.

This physiological study raises serious concerns about the routine administration of sildenafil in all patients with ARDS. The decrease in systemic blood pressure and the increase in shunt are potentially serious side effects. On the other hand, sildenafil could be useful in patients with more severe forms of right ventricular failure although the decrease in systemic blood pressure may still be an important limitation.

Cornet AD, Hofstra JJ, Swart EL, Girbes AR, Juffermans NP. Sildenafil attenuates pulmonary arterial pressure but does not improve oxygenation during ARDS. Intensive Care Med. 2010;36:758-64.