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*(Accepted for publication January 28, 2009.)*

Anesthesiology 2009; 110:1198 

*In Reply:*—We thank Dr. Navarro-Martinez and colleagues for their letter concerning our review on perioperative fluid management. In general, we would like to point out that our article was targeted on perioperative fluid therapy in patients who primarily have a steady state concerning their fluid compartments.[1] In these patients an intact vascular barrier function ensures that, despite a positive pressure within the circulatory space, plasma constituents are not distributed evenly across the whole extracellular compartment.[2,3] Rather, under normal physiologic conditions, they are predominantly retained where they are needed to maintain a sufficient cardiac preload. A small residual flow towards the interstitial space is managed by an intact lymphatic system.[1] In this situation, requirement-adapted fluid handling might limit tissue edema by considering physiologic and pathologic shifting, provided that the vascular barrier is primarily fully functioning.[1]

The septic patient, undergoing surgery or not, does not present such a steady state.[4] The normally accompanying capillary leakage syndrome, as a result of an insufficient vascular barrier, leads to a barely calculable shift of fluid and macromolecules (such as proteins and colloids) towards the interstitial space, representing a primary problem during sepsis.[5] Recent evidence suggested a deterioration of the endothelial glycocalyx by inflammatory mediators to be an important part of the underlying pathomechanism.[6,7] Therefore, a careful differential indication between crystalloids and colloids as suggested for the perioperative steady state might not only be insufficient in this context, but in vain.[1] Until today, we only know that we have to give enough, irrespective of the kind of fluid, to improve outcome of patients suffering from severe sepsis and septic shock.[8]

We support most of the interesting considerations by Dr. Navarro-Martinez and colleagues. However, septic patients were not the focus of our rational approach.

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*(Accepted for publication January 28, 2009.)*

Anesthesiology 2009; 110:1198–9 

# Nitric Oxide Metabolites, Platelet Activation, and Myocardial Ischemia Reperfusion Injury

*To the Editor:*—We read with great interest the research article by Nagasaka *et al.* demonstrating a role of nitric oxide and its metabolites in the systemic circulation.[1] As the authors impressively demonstrate, active nitric oxide metabolites are carried into the systemic circulation where they accumulate in the blood and in the heart, and as a result have significant impact on the extent of myocardial ischemia reperfusion injury.

Nitric oxide induces cyclic guanosine phosphatase activation and increases cyclic guanosine monophosphate levels in several tissues, including platelets. Several investigators have demonstrated that this is caused not only by endogenous nitric oxide with significant impact on platelet activity, but also by inhalative nitric oxide.[2,3] The crucial importance of platelets and the activity state of platelets on the extent of myocardial ischemia reperfusion injury was outlined by previous investigations.[3–5] A downstream target of cyclic guanosine phosphatase activation in platelets is vasodilator-stimulated phosphoprotein (VASP), a central cytoskeletal binding protein.[6] The intracellular increase in cyclic guanosine phosphatase results in a phosphorylation of VASP, which is a crucial step in the control of platelet activity. Clini-

The above letter was sent to the authors of the referenced report. The authors did not feel that a response was required. —James C. Eisenach, M.D., Editor-in-Chief.

cally, the extent of VASP phosphorylation is used to monitor the success of an anticoagulatory therapy, as during clopidogrel therapy, and reflects the success of platelet activation during an anticoagulatory therapy.[7]

Therefore we were wondering whether the authors think that VASP phosphorylation could be a useful monitor in their experimental system, and whether the monitoring the extent of VASP phosphorylation could be a valuable tool to monitor the clinical effectiveness if an inhaled nitric oxide therapy.

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*(Accepted for publication February 2, 2009.)*

Anesthesiology 2009; 110:1199–200 

# Continuous Positive Airway Pressure Applied Through a Bronchial Blocker as a Treatment for Hypoxemia due to Stenosis of the Left Main Bronchus

*To the Editor:*—We have developed a simple, lightweight continuous positive airway pressure (CPAP) delivery device for the nondependent lung during one-lung ventilation,[1] and we applied this device to a patient with urgent hypoxemia caused by acute airway obstruction where mechanical ventilation before installation of the stent device[2,3] did not improve the hypoxemia sufficiently. To our knowledge, this is the first report of selective CPAP delivery to a derecruited lung in which a bronchial blocker tube treated hypoxemia caused by severe stenosis of the main bronchus.

A 58-yr-old male who received an esophagostomy for esophageal cancer 7 yr ago began coughing. Two weeks later, chest computed tomographic imaging showed that his left main bronchus was narrowed by a submucous tumor, which was considered a recurrence of the esophageal cancer. While plans were being made for radiation therapy and chemotherapy to reduce the size of the tumor, the patient suddenly experienced severe dyspnea in the ward and was transferred to the intensive care unit. Even with the administration of oxygen of over 15l/min with an oxygen facemask, his peripheral saturation of oxygen ($Spo_2$) did not improve and stayed at around 70%, accompanied by tachypnea, tachycardia, and perspiration. No sound could be heard from the left lung through a stethoscope. To treat the hypoxemia, we intubated the patient's trachea to administer mechanical ventilation with 100% oxygen, but conventional mechanical ventilation did not improve the hypoxemia sufficiently (results of arterial blood analysis with 100% $O_2$: pH 7.41, $Paco_2$ 41 torr, $Pao_2$ 50 torr, $Spo_2$ around 75 to 85%, heart rate 100 to 110 beats/min). The bronchoscopy after the unsuccessful conventional ventilation revealed that the left main bronchus seemed to be totally occluded.

During bronchoscopy after moderate hypoxemia that continued for 10 h after the tracheal intubation we found that a fiberscope head tip of 2.8 mm diameter could be wedged through the severe stenosis, which had previously appeared as total occlusion at the left main bronchus. To recruit the left lung to treat the hypoxemia, we replaced the endotracheal tube with a Univent tube (Fuji System Corp., Tokyo, Japan), and placement of the bronchial blocker tube beyond the severe stenosis was easily achieved without bleeding. CPAP was applied by supplying oxygen to the left lung through a CPAP-delivery device composed of a three-way stopcock (Connecta Plus 3; Becton Dickinson, Helsingborg, Sweden) whose side port works as a pressure relief port.[1]

With the CPAP delivery device, we performed a recruitment maneuver of the lower lobe of the left lung, which had been isolated and derecruited by the severe stenosis caused by the tumor, by using a finger to temporarily occlude the side port of the three-way stopcock while carefully monitoring the blood pressure and heart rate to avoid excessive overinflation of the left lung. The maneuver improved $Spo_2$ to 100% immediately, and the partial recruitment of the lower lobe of the left lung was confirmed by aeration in the chest radiograph.

The relationship between CPAP produced with the device and oxygen flow rate was confirmed before the application, where 5l/min and 10l/min of oxygen flow rate created 4 cm $H_2O$ and 14 cm $H_2O$, respectively. When we decreased the oxygen flow rate to 5l/min while the right lung was ventilated with a fractional inspired oxygen tension of 0.4, $Spo_2$ fell under 90%, and therefore the oxygen flow rate was raised again to 10l/min and $Spo_2$ stayed at over 99%. After oxygenation was stabilized, the right lung was ventilated by pressure support to assist the patient's spontaneous ventilation, while CPAP was applied to the left lung. To avoid accidental overinflation that could cause barotrauma and pneumothorax of the left lung during CPAP application, we used a piece of adhesive tape to hold the device's stopcock fully open in three directions, and we took care not to obstruct the side port of the stopcock.

On the third day of the patient's stay in the intensive care unit, with CPAP delivery to the left lung under mechanical ventilation with 100% oxygen to the right lung, pulmonary gas exchange was stable (results of arterial blood gas analysis: pH 7.48, $Paco_2$ 35 torr, and $Pao_2$ 367 torr) and a chest radiograph showed total recruitment of the left lower lobe. The installation of a self-expanding stent device (Ultraflex; Boston Scientific, Natick, MA) at the stenosis was conducted successfully, and both lungs were ventilated with the Univent tube with the bronchial blocker withdrawn. The patient was tracheally extubated without