Alternatively, is it better to give him or her autologous FFP at the beginning, and save the platelet-rich plasma for the end of the case? My personal preference is that it does not make much sense to give a known exposure up front to attempt to prevent a merely possible exposure later in the case, so I give the patient back his or her plasma, along with more heparin, and get another ACT before I will give autologous FFP. The article by Boldt et al reinforces this philosophy in my mind, as the evidence for the benefits of APP-PRP are at best marginal.

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Hypoxemia From Transient Right-to-Left Shunting During Atrial Septal Defect Repair Detected by Intraoperative Transesophageal Echocardiography

To the Editor:

We have detected by intraoperative transesophageal echocardiography (TEE), using pulsed-wave Doppler, several cases of intracardiac shunt reversal with resultant oxyhemoglobin desaturation occurring during manipulation of the heart prior to cannulation for cardiopulmonary bypass (CPB) for repair of atrial septal defects (ASD). We speculate that manipulation of the heart can sufficiently impede right ventricular filling to increase right atrial pressure leading to shunt reversal and transient hypoxemia. The following case illustrates our hypothesis.

A 25-year-old, ASA II, 75-kg woman was scheduled for a secundum ASD repair. She came to medical attention after developing dyspnea on exertion and shortness of breath when walking one block. She had a known heart murmur since age 12, but there was no history of cyanosis or congestive heart failure. Her physical examination was remarkable for a grade

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**Fig 1. Intraoperative transesophageal echocardiogram using pulsed-wave Doppler demonstrating the characteristic spectral flow velocity profile of left-to-right shunting at the atrial defect. The Doppler sample volume is placed along the right atrial septal surface adjacent to the defect, obtained from the four-chamber long-axis view.**
Fig 2. Reversal of interatrial shunt during manipulation of the heart prior to venous cannulation for cardiopulmonary bypass. Pulsed-wave Doppler demonstrates right-to-left shunting throughout the cardiac cycle.

III/VI holosystolic murmur with fixed splitting of S₂. Cardiac catheterization revealed a Qp/Qs of 2.7, pulmonary artery pressure of 41/15 mmHg (24 mmHg mean), and pulmonary artery occlusion pressure of 12 mmHg.

After placement of arterial and central venous lines, the patient was induced with fentanyl, thiopental, and doxacurium, and maintained with isoflurane and 100% oxygen. TEE confirmed the presence of a secundum ASD. Pulsed-wave Doppler demonstrated left-to-right shunting at the atrial level (Fig 1). The patient maintained an SaO₂ of 99% to 100% on an FIO₂ of 1.0 until the heart was manipulated for insertion of the bicaval cannulae for CPB. At that time, SaO₂ decreased to 87%, lasting for approximately 1 minute despite adequate ventilation, by direct observation of the lungs. Pulsed-wave Doppler demonstrated the presence of right-to-left shunting at the atrial level (Fig 2). After cannulation, the arterial saturation returned to the baseline value, and TEE showed the normal left-to-right flow profile. The remainder of the ASD repair was uneventful.

TEE has multiple intraoperative indications as both a diagnostic and monitoring tool. Because flow velocities across an ASD are relatively low, this lesion lends itself well to Doppler echocardiographic examination. With the exception of very small defects, the size of the ASD has less effect on the flow of blood across the defect than do the relative filling resistances in the ventricles. The direction of blood flow through the defect is related to differences in atrial pressures throughout the cardiac cycle, which are determined by the relative compliances of the respective ventricles. Transient right-to-left intracardiac shunting has been attributed to such factors as straining and coughing, release of high levels of PEEP, and positive-pressure ventilation. We believe that manipulation of the heart can significantly alter atrial pressures such that a right-to-left shunt with arterial desaturation may ensue, even in a patient without a history of cyanosis or right-to-left shunting. This case also demonstrates the utility of TEE in the assessment of intraoperative hypoxemia in a patient with intracardiac shunting.

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Epinephrine for Reversal of Atrioventricular Block Following Aortic Cross-Clamping and Potassium Cardioplegia

To the Editor:

In the anesthetized dog, epinephrine has proved to be most effective in attenuating atrioventricular (A-V) block following the calcium channel blocker diltiazem.¹ The present report shows that epinephrine can also attenuate the A-V block following resumption of coronary circulation after aortic cross-clamping and cold potassium cardioplegia in humans.

Two patients undergoing coronary artery bypass grafting, and one patient undergoing mitral valve replacement, are reported. Following initiation of cardiopulmonary bypass, the patients were cooled to 28-30°C, and the heart was arrested by aortic cross-clamping and cold potassium (20 mEq/L)-crystalloid cardioplegia. In the three patients, rewarming the patient and release of the aortic cross-clamp were followed by complete A-V block. A bolus of epinephrine, 1.5 µg/kg, followed by an infusion at a rate of 0.05-0.15 µg/kg/min were administered via the central venous line. The A-V conduction progressively improved, and normal sinus rhythm was restored. Figure 1 depicts the ECG response in one of the patients.

A-V dissociation arrhythmias after release of the aortic cross-clamp have been attributed to multiple factors such as hypoxic myocardium, continued hypothermia of the conduction system, and changes in the action potential of the cardiac membrane secondary to the high K⁺ of the cardioplegic solution.² The electrical and mechanical activity usually do not return for some time depending on the duration of the aortic cross-clamping and the volume of the cardioplegia used.

Epinephrine is the key endogenous catecholamine. Its potent actions on the heart are mediated by β₁-adrenoceptor activation. Myocardial β₂-adrenoceptors also exist, and it has been proposed that these innervated extrajunctional β₂-adrenoceptors may represent "hormonal" adrenoceptors that are responsive to the circulating blood-borne epinephrine.³ Thus, epinephrine acts on the β₁- and the β₂-adrenoceptors of the heart, resulting in a potent inotropic and chronotropic effect. It also speeds A-V conduction, and hence can decrease the grade of A-V block caused by disease, drugs, or vagal

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Fig 1. ECG tracings (V₁) in one of the patients depicting the effect of epinephrine on A-V block:
(A) The initial degree of A-V block. (B, C, D) ECG tracings 5, 7, and 10 minutes after the initiation of epinephrine infusion, showing the progressive attenuation of the A-V block. (E) ECG showing restoration of sinus rhythm, 15 minutes after the initiation of the epinephrine drip.