unlikely that the modest IABP-induced pulse pressure of only 24 ± 8 mm Hg would generate a dp/dt of 645 ± 64 mm Hg/s, particularly at the level of small cerebral arteries, and thus be enough to produce sufficient stretch to induce significant production of L-arginine in the small cerebral arteries. If the pulse pressure was not sufficient, then significant cerebral vasodilation would not be obtained to affect the $Sjvo_2$, and hence the IABP-induced pulsatility would be unable to ameliorate the impaired $Sjvo_2$. A similar study using a pulsatile system capable of producing a pulse pressure of 50 to 60 mm Hg⁶ to mimic that produced by the natural heart would be needed to be able to definitely exclude or include the role played by pulsatility. The study only demonstrates that the modest IABP-induced pulsatility was not effective but by no means excludes the role pulsatility may have.

3. It is widely known that during CPB the levels of circulating endogenous catecholamines including norepinephrine are increased. During systemic infusion of norepinephrine, increased electrical brain activity (consisting of increased low-voltage high-frequency waves and decreased high-voltage slow-frequency waves) suggestive of promotion of excitatory (*N*-methyl-D-aspartate?) receptor activation has been noted in nonischemic rabbits (unpublished observations of electroencephalograms concurrent to brain microdialysis studies), which may lead to Na^+ and Ca^{++} influx. To maintain the normal Na^+ and Ca^{++} transmembrane gradient, the demand for mechanisms to extrude them, which are highly oxygen consuming, will be increased. Therefore during late CPB the decreased $Sjvo_2$ might be a manifestation of that increased oxygen extraction not counteracted by the anesthetics, pressures, or pulsatility used by the authors. The exact effect of increased circulating catecholamines during CPB on the cerebral vasculature is not known, but if it was equivalent to increased sympathetic activity, then vasoconstriction of small cerebral vessels would result with consequent decreased flow.⁷ If both effects are operating, the development of decreased $Sjvo_2$ over the first 20 minutes of perfusion and the return to normal after CPB, also in 20 or 30 minutes, are easily explainable.

Norepinephrine determination with and without blockade in conjunction with metabolic studies across the brain in both groups might shed some light.

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12/8/104877

doi:10.1067/mtc.2000.104877

Reply to the Editor:

We appreciate the comments of Miyamoto and Miyamoto. As we described previously, it is not clear which type of pulsatile waveform has positive effects on cerebral circulation and improves outcome of patients.¹

The internal jugular venous oxygen saturation ($Sjvo_2$) value expressed in Fig 1 was the value measured in a blood sample drawn from a jugular venous catheter. At the same time, we continuously measured the $Sjvo_2$ value and regional cerebral oxygenation using an analysis system (Explorer system, Baxter Healthcare Corp, Irvine, Calif) and found that there were no changes in the $Sjvo_2$ and regional cerebral oxygenation values throughout the study. In our previous study,² we reported a close correlation between the oxygen saturation value measured by the optical catheter and the oxygen saturation value in sampled blood by the blood gas analyzer. Therefore we do not think that the change in $Sjvo_2$ would be observed if the value had been measured more frequently during cardiopulmonary bypass (CPB). It remains controversial whether cerebral autoregulation is intact during CPB.^{3,4} Newman and associates³ reported that an increase in cerebral blood flow was observed by an increase in mean arterial pressure. In contrast, Sadahiho and the associates⁴ reported that cerebral blood flow was constant when mean arterial pressure was higher than 50 mm Hg. At present, there is no solid agreement concerning the acceptable level of mean arterial pressure during CPB.

As we described previously, however, our pulse pressure was a physiologic pulse waveform. A higher pulse pressure (>40 mm Hg) might have had a positive effect of high-pressure intra-aortic balloon pumping (IABP) on cerebral circulation.¹

We did not measure the plasma catecholamine levels in the two groups; therefore it was not known whether plasma levels could have some effects on cerebral microcirculation in our study. Cerebral microcirculation might be affected by plasma catecholamine level, as they described. However,

other factors, such as renin-angiotensin and thromboxane A₂, might have effects on cerebral microcirculation.⁵

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12/8/104876

doi:10.1067/mtc.2000.104876

Vasodilatation after cardiac surgery

To the Editor:

Argenziano and associates,¹ in their article on management of vasodilatory shock after cardiac surgery, provide an excellent study of enormous clinical importance. Identification of low ejection fraction and angiotensin-converting enzyme (ACE) inhibitors as risk factors for vasodilatory shock after cardiopulmonary bypass (CPB) confirms the initial analysis of 2729 patients undergoing CPB in our center.

However, the study raises a number of important issues that were not mentioned:

While discussing the mechanisms for arginine vasopressin deficiency, which may be multifactorial, the authors failed to mention that angiotensin II decreases arginine vasopressin release from the neurohypophysis.² This implies that the newer selective angiotensin II inhibitors like losartan may also cause vasodilatory shock.

The majority of patients undergoing routine CPB, regardless of left ventricular function, have no sequelae if the ACE inhibitor is withheld on the morning of the operation. Withholding the ACE inhibitor also may decrease the chance of renal complications via the reduced efferent arterial tone caused by ACE inhibitors, which reduces glomerular filtration rate during CPB.³ Obviously, withholding the ACE inhibitor is not appropriate in patients awaiting cardiac transplantation or left ventricular assist device implantation, but the ACE inhibitor could be changed to a short-acting one from the long-acting one that most patients receive.

Amiodarone, which is a noncompetitive α -blocker and β -blocker, is commonly co-prescribed with ACE inhibitors in patients with end-stage cardiac failure before heart transplantation.⁴ Amiodarone has been shown to act synergistically with ACE inhibitors in causing vasodilatory shock via a two-hit mechanism.⁵ Unfortunately no mention was made of this.

Finally, nicorandil, a new potassium channel opener used in the treatment of angina, in our experience, has been associated with decreased systemic vascular resistance and increased need for vasoconstrictor treatment.⁶ This seems to be especially common when patients have taken a 10-mg dose within 12 hours of CPB.

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[Response declined]

12/8/105207

doi:10.1067/mtc.2000.105207

Posterior aortoventricular bleeding after aortic valve replacement with the stentless Cryolife-O'Brien valve

To the Editor

Mediratta, Sosnowski, and Galiñanes¹ have pointed out the possibility of posterior aortoventricular bleeding after supra-annular aortic valve replacement with a stentless prosthesis. Such complications are directly related to extensive decalcification of the aortic anulus (along the left coronary part of the anulus).

We have now implanted nearly 400 Cryolife-O'Brien valves (Cryolife International, Atlanta, Ga) in our hospital,