behind the heart. Cardiopulmonary bypass was re instituted, and the heart was carefully lifted to find out the site of bleeding. No evidence of any bleeder could be found in spite of multiple efforts; specifically, there was no hematoma around the coronary sinus or the cardiac veins or anywhere on the posterolateral surface of the left ventricle. We were apprehensive about manipulating the heart too much with a mitral prosthesis in place. The ventricular mass seemed to be oozing from all over.

The posterior surface of the heart was packed with a large sponge, the patient weaned off cardiopulmonary bypass, and protamine was given. The bleeding slowly reduced, although when the sponge was taken out a continuous flow again was encountered. A fresh sponge was packed in and fresh blood products given, although no coagulation disorder was found on investigation. The bleeding looked manageable after some time and the skin was closed after mediastinal drains were put in; the sternum could not be approximated because the heart was pushed up by the sponge. The patient drained just 200 mL over the next 12 hours and was hemodynamically stable. She was taken back to the operating room and the sponge was removed with no collection of blood. The sternum could be closed easily. She drained just 150 mL over the next 12 hours and was extubated. She made an uneventful recovery thereafter.

Our case confirms the efficacy of a seemingly gross technique to control frustrating “impossible” bleeders.

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Brain Protection During Cerebral Perfusion
To the Editor:

I read with interest the report by Dr Safi and colleagues [1] regarding the clinical use of retrograde cerebral perfusion during aortic arch aneurysm repair. Although some experimental studies have suggested that this may be a useful method for brain protection [2], other studies have revealed nonhomogeneous distribution of flow with a consequent risk of localized brain injury [3, 4]. Therefore, I was disappointed that Safi and colleagues did not describe comprehensive preoperative and postoperative neurologic testing as well as preoperative and postoperative psychometric testing to detect subtle deterioration of motor or cognitive function. In addition, it would have been of interest for these patients to have undergone preoperative and postoperative cranial magnetic resonance imaging so that it would be possible to determine specifically if obvious gross neurologic insults, such as the stroke that was described in 1 patient, were indeed embolic in origin as was inferred by Safi and colleagues. Although Safi and colleagues were “encouraged by the way all patients looked and behaved after their operations,” many previous studies in which neuropsychometric testing has been performed after exposure to cardiopulmonary bypass alone without the additional potential deleterious effects of circulatory arrest or atherosclerotic emboli from removal of an arch aneurysm have revealed important deterioration in cerebral function. Our own experience in undertaking a prospective analysis of 171 patients randomized to either circulatory arrest or continuous bypass has confirmed that comprehensive testing by psychologists and neurologists exposes evidence of neurologic injury not apparent to surgeons [5]. I am hopeful that in the prospective study of retrograde cerebral perfusion that Safi and colleagues state they are currently undertaking, all patients will be studied in an appropriately comprehensive fashion, both preoperatively and postoperatively.

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References

Sodium Nitroprusside During Aortic Cross-clamping
To the Editor:

We were pleased to see that Dr Cernaianu and colleagues [1] have addressed the issue of using sodium nitroprusside to control proximal hypertension during aortic cross-clamping. In our earlier study [2] 4 years ago a similar protocol (except for one significant difference, the method of anesthesia) was followed and identical results were reported.

Several questions arise from Cernaianu and colleagues’ report. How were somatosensory evoked potentials measured, ie, where were electrodes placed? Was the effect of halogenated anesthetics considered? Halothane and enflurane anesthetic agents generally are avoided in clinical situations because they interfere with the recording of evoked cortical potentials [3]. Isoflurane has been shown to progressively affect cortical responses significantly at 1% to 2% concentrations [4, 5]. Cernaianu and colleagues used concentrations of 0.25% to 2%.

In our experience, times associated with the initial loss of somatosensory evoked potentials, either the time for a 10% increase in latency or the time for total loss of somatosensory evoked potentials, are critical determinants of ischemic injury [6]. Do Cernaianu and colleagues have such data? We also have found (as did Cernaianu and colleagues) that time to somatosensory evoked potential return after removal of the cross-clamp correlated well with the neurologic state at recovery. If normal spinal cord conduction did not return within 30 minutes, animals suffered spastic paraplegia, whereas if morphologically intact somatosensory evoked potential tracings returned early, they were spared [7].

Was any attempt made to measure spinal cord perfusion pressure? It has been shown that the reduction or reversal of spinal cord perfusion pressure is one of the most significant predictors of neurologic injury [2, 6]. In our study sodium nitroprusside caused a reversal of spinal cord perfusion pressure. Were
any measurements taken of spinal cord blood flow? We noted differences during cross-clamping that were not present at baseline, or after reperfusion.

We commend Cernaianu and his associates for restating the dangers of indiscriminate use of sodium nitroprusside. Although it might not be the best solution, our studies indicate that exsanguination and volume control to moderate proximal hypertension is a viable alternative [8]. If proximal pressures are kept within the normal range, autoregulation of the spinal cord will not be compromised.


Woloszyń TT, Marini CP, Coons MS, et al. Partial exsanguina

Reply
To the Editor:

We appreciate Drs Marini and Nathan’s kind comments on the importance of our work [1]. We used an animal protocol very similar to that of Marini and co-workers [2] to demonstrate our concerns regarding the use of a hypotensive agent, sodium nitroprusside, during aortic procedures involving spinal cord ischemia.

Although we are aware of the effect of halogenated anesthetic compounds on the recording of somatosensory evoked potentials, we did not encounter any specific problems related to their use. Moreover, we used halothane anesthesia for both the control and the treated group, and whatever possible minimal suppressive effect the halogenated agent may have had, it did not affect our measurements or our results. Other investigators [3] also have used halogenated agents for maintenance of anesthesia during somatosensory evoked potential measurements. The somatosen-
sory evoked potential recording is susceptible to anesthetic influ-

ence only when the drug is administered in high doses. The actual recommendation of the American Society of Anesthesiologists regarding the anesthetic regimen for intraoperative monitoring of somatosensory evoked potentials during operation on the thoracic aorta is continuous intravenous infusion of a narcotic (fentanyl or sufentanil) supplemented with low-dose inhaled anesthetic (isoflurane or halothane) or with nitrous oxide [4].

In our study, the somatosensory evoked potentials were measured in a standard fashion as described by Laschinger and associates [5] in 1987. This method also was used by Marini and associates [2] in their study. Our study did not attempt to measure spinal cord perfusion pressure or spinal cord blood flow. Howev-

er, we commend Dr Marini and his colleagues for presenting such data in their article. Finally, we agree with Drs Marini and Nathan’s statement that other solutions such as exsanguination and volume control may attenuate the need for pharmacologic manipulation of hypertension during operations involving aortic cross-clamping.

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References


Postoperative Atrial Arrhythmias
To the Editor:

We read with interest the article entitled “Hazards of Postoperative Atrial Arrhythmias” by Creswell and associates [1] and the editorial comment by Dr Cox [2]. These articles offer a useful and comprehensive review of atrial fibrillation occurring in postoperative cardiac patients. We totally agree with Dr Creswell and his colleagues regarding the hazards effects of atrial fibrillation, especially its correlation with the increase in the incidence of stroke. Atrial ischemia is suggested by Dr Creswell and colleagues as the most important cause or stimulus that generates vulnerability to atrial fibrillation. It is well known that the atrial fibrillation develops most commonly 2 or 3 days after the operation. Also, in some cases, chronic atrial fibrillation converts to sinus rhythm intraoperatively and persists for hours or even days before it returns to the fibrillation status. A different mechanism that occurs postoperatively is required to explain these observations. We were surprised that despite such a good review of the

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