domized clinical study to evaluate this hypothesis is justifiable.

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Combined mitral valve repair and the Cox maze procedure for mitral valve prolapse and regurgitation and associated atrial fibrillation

To the Editor:

I read with great interest the report by Handa and associates¹ on the early and intermediate term results of combined mitral valve repair and the Cox maze procedure for patients with severe mitral regurgitation and associated atrial fibrillation. It is gratifying to note that at the Mayo Clinic the addition of the Cox maze procedure to mitral valve repair not only is safe and effective but also decreases late complications.

One of the serious complications of mitral valve prolapse, which is otherwise a common² and benign³ condition, is mitral regurgitation.^{4,5} In fact, the commonest cause of mitral regurgitation in the United States is mitral valve prolapse.^{5,6} Mitral regurgitation in mitral valve prolapse may become progressive in some patients.⁵ One of the serious complications of progressive mitral regurgitation is atrial fibrillation, which usually persists even after successful corrective surgery of the mitral valve and often recurs after pharmacologic or electric cardioversion. Although postoperative atrial fibrillation can be successfully managed by antiarrhythmic drugs and long-term anticoagulant therapy to prevent thromboembolism, these therapeutic modalities are not without side-effects, torsades de pointes and bleeding, respectively. It is gratifying that the adjunctive Cox maze procedure at the Mayo Clinic increased the restoration of sinus rhythm to 82% of their patients.

Therefore, as the authors concluded, "Although the addition of the Cox maze procedure lengthens hospital stay in patients having mitral valve repair, overall medical costs may be decreased by reducing the need for long-term anticoagulation, as well as costs associated with stroke and bleeding complications."¹

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Should the bidirectional Glenn procedure be better performed through the support of cardiopulmonary bypass?

To the Editor:

We read with great interest the report of Jahangiri and associates¹ and the commentary by Jonas on the technique of bidirectional Glenn shunt through a thoracotomy without the use of cardiopulmonary bypass (CPB). We agree that clamping the superior vena cava (SVC) without decompressing the internal jugular system exposes the brain to the effects of reduced cerebral perfusion pressure. We have used electrophysiological indices of cerebral function (electroencephalography or evoked potentials) and transcranial Doppler during bidirectional Glenn shunts in patients with pulmonary atresia without support (n = 2) or through the use of CPB (n = 4). During clamping of the SVC without CPB, major reductions (>50%) in the diastolic, mean, and peak systolic blood flow velocities of the middle cerebral artery were identified, which were followed by mild electrocortical alterations as indicated by longer latencies of the cortically generated evoked potentials.² In contrast, this situation did not occur or was minimal in those cases in which clamping of the SVC was done with the support of CPB. We have learned that Doppler flow changes and electrocortical alterations may be expected as a result of acute SVC hypertension if the internal jugular venous system is not decompressed.³ In one of our cases without CPB, an intraoperative shunt into the SVC was indicated due to the presence of extremely low flow velocities and electrocortical alterations during SVC clamping. In this case, while the SVC shunt was patent, flow velocities were maintained and the latencies of evoked potentials returned to preclamping levels.⁴ In our institution, we routinely use CPB and intraoperative brain function monitoring by transcranial Doppler, near-infrared spectrophotometry, and electroencephalography for children undergoing these procedures.³ A note of caution should be made that the reported absence of gross neurologic deficits in Jahangiri and colleagues' cases is not an indicator that the brain is free of potential alterations during SVC clamping without CPB. Furthermore, under conditions of normothermic ischemia, the brain does not receive the protective benefit of hypothermia.⁵ Without brain protection, even short intervals of low cerebral perfusion could generate minor or subclinical deficits that might be detectable only through detailed cognitive testing. We agree with Jonas' comments that the demonstration of safety of this procedure will rest on the careful