Electrographic seizure after neonatal and infant cardiac surgery

To the Editor:
We read with interest the article by Gaynor and associates, titled “Relationship of Postoperative Electrographic Seizure to Neurodevelopmental Outcome at 1 Year of Age After Neonatal and Infant Cardiac Surgery.” We appreciate the effort and hard work of the authors on this complex neurophysiologic phenomenon.

In this study postoperative electroencephalographic (EEG) seizures have been identified in a small number of patients (15/144, 13%), with absolutely no seizure activity reported in patients with transposition of the great arteries and ventricular septal defect. The latter are considered among the high-risk groups. This observation may be quite startling or may be purely coincidental.

The authors did not mention the preoperative seizure history or duration of seizure activity (which can significantly contribute to seizure-related neuronal death), and they did not discuss whether the EEG examiners were blinded to the fact that the child had a seizure in the postoperative period.

Gaynor and associates rightly mentioned that antiepileptic medication given to all the patients was not standardized and there was no control group, so it is not possible to determine conclusively whether treatment affected the neurodevelopmental outcome.

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References
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Reply to the Editor:
We appreciate the comments by Hashmi, Hanif, and O’Reilly concerning our recent article, titled “The Relationship of Postoperative Electrographic Seizures to Neurodevelopmental Outcome at 1 Year of Age After Neonatal and Infant Cardiac Surgery.” They raised several questions concerning including (1) preoperative seizure history; (2) the lack of seizure activity in patients with transposition of the great arteries (TGA) with a ventricular septal defect (VSD), previously considered a high-risk group for postoperative seizures; (3) duration of seizure activity; and (4) whether or not the electroencephalographic (EEG) examiners were blinded to the fact that the child had a seizure in the postoperative period.

The authors did not mention the preoperative seizure history or duration of seizure activity (which can significantly contribute to seizure-related neuronal death), and they did not discuss whether the EEG examiners were blinded to the fact that the child had a seizure in the postoperative period.

Gaynor and associates rightly mentioned that antiepileptic medication given to all the patients was not standardized and there was no control group, so it is not possible to determine conclusively whether treatment affected the neurodevelopmental outcome.

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without VSD). We speculated that the decreasing incidence of postoperative seizures compared with previous reports, particularly among children undergoing biventricular repair, was due to improved (intraoperative and perioperative) management strategies. It is likely that specific anatomic subgroups previously considered to be at high risk for postoperative seizure activity are no longer at increased risk. As Hashmi, Hanif, and O’Reilly note, we did not report duration of seizure activity. However, we did evaluate the number of seizures in the monitoring period as a risk factor for worse neurodevelopmental outcome. The number of seizures was not predictive of scores on either the Mental Developmental Index (MDI) or the Psychomotor Developmental Index (PDI) of the Bayley Scales of Infant Development-II.

Hashmi, Hanif, and O’Reilly also ask whether the EEG examiners were blinded to the fact that the child had a seizure in the postoperative period. As the recent study evaluated neurodevelopmental outcomes, we assume that they do not mean the EEG examiners, but rather the psychologists performing the 1-year neurodevelopmental evaluation, who were blinded to the child’s seizure status.

Internal thoracic artery grafts to right coronary system

To the Editor:

We read with interest the article by Sabik and coworkers,1 “Influence of Patient Characteristics and Arterial Grafts on Freedom From Coronary Reoperation.” We congratulate the team of surgeons from The Cleveland Clinic for coming up with another landmark article that will be of immense benefit to both surgeons and patients around the world.

We agree with Sabik and coworkers1 that aggressive risk factor modifications and more extensive use of arterial grafts are necessary for reduction of reoperations. They identified a reduced early hazard phase when there was incomplete right coronary artery (RCA) revascularization. During their discussion, they presented no valid reason for this apparently anomalous finding and remarked “It is hard to understand how incomplete revascularization to the RCA and elevated triglyceride level both lowered the early risk of reoperations.”

We have had similar experience with patients with a diffusely diseased RCA either bifurcating or trifurcating into small-caliber vessels, with none suitable for grafting. In this situation, not grafting the RCA would not disturb the native collaterals and would probably result in better intermediate postoperative results, as demonstrated by Sabik and coworkers.1 Endarterectomy in this situation would still result in incomplete revascularization, because the plaque cannot be removed completely from the small distal vessels, consistent with the poor early results seen with RCA revascularization.

In the later part of the study, internal thoracic artery grafting to RCA did reduce reoperation rates. Sabik and coworkers should specify what proportion of these patients in the early and later portions of the study had the posterior descending artery grafted in contrast to distal RCA grafts. Posterior descending artery grafts show significantly better patency rates and are associated with lower postoperative morbidity.2 We have observed fewer postoperative coronary events when the posterior descending artery is grafted instead of the distal RCA. We believe that the benefit observed in the later part of the study may have been due to a trend toward posterior descending artery grafting.

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References


Monobloc or separate aortic and mitral homografts?

To the Editor:

Recently, Obadia and associates1 reported their experience with monobloc aorto-mitral homograft for extensive endocarditis. Since 1994, we have performed combined aortic and mitral valve replacement with homografts in 6 patients (mean age: 31 ± 7 years). However, two separated valves were used for the reasons explained below. The etiology of the valve disease was rheumatic (n = 3), congenital (n = 1), or bacterial endocarditis with abscess of the aorto-mitral junction (n = 2). One patient was undergoing a third reoperation for prosthetic valve dehiscence. As in Obadia’s technique,1 the first step of the operation was fixation of the papillary muscles, whose exposure can be very difficult. It was anticipated that a bulky aorto-mitral monobloc, once lowered into position, would somewhat obstruct the access to the papillary muscles. In addition, the use of an aorto-mitral monobloc would necessarily have restricted the panel of available sizes, increasing the risk of mismatch, which has been shown as a factor of mitral homograft dysfunction.2 Thus, it was decided to implant two separate homografts. The aortic and the mitral valve were approached separately through the standard incisions. Part of the papillary muscle sutures were inserted through the aortic orifice. The mitral homograft was inserted according to a previously described technique, which invariably included ring annuloplasty.2 This latter technical detail is probably relevant.