Letters to the Editor

Rastelli repair for transposition of the great arteries

To the Editor:

I read with interest the letter from Lecompte¹ regarding the article "Twenty-five-year experience with Rastelli repair for transposition of the great arteries."² Although the letter was elegantly answered by my mentor in the Rastelli project, I believe there are a few points that need further clarification.

Lecompte claims that I am confused regarding the differences between a Lecompte maneuver and a Lecompte procedure. I consider that the concept that Rastelli brought into our field was a breakthrough discovery. The ventricular septal defect baffling from the left ventricle to the aorta is the main concept in the correction of transposition, ventricular septal defect, and pulmonary stenosis or atresia. I do not believe that resection of the conal septum or of the anterosuperior margin of the defect is of such importance to merit a new name of a procedure. Likewise, the way in which right ventricular-pulmonary arterial continuity is established can be another modification of the original Rastelli procedure. These are really accessories of the main concept: ventricular septal defect baffling from the left ventricle to the aorta and establishment of right ventricular-pulmonary artery continuity.

Like Lecompte, I believe that modifications or even more reappraisals of the original Rastelli technique for correction of tetralogy of Fallot and pulmonary atresia with autologous fresh pericardium³ can be and should be taken into consideration for repair of transposition, ventricular septal defect, and pulmonary stenosis or atresia. For example, our experience with Rastelli procedures with autologous fresh pericardial valved conduits was presented in several meetings and articles.^{4,5} The late results are spectacular, with a freedom from reoperation and a survival of more than 80% at 15 years.

This is another alternative to the "classic" Rastelli operation. I believe that the

operation proposed by Gian Carlo Rastelli is here to stay, with all the modifications that will allow improvements in survival and reoperation rates.

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Quinaprilat during cardioplegic arrest

To the Editor:

I read with interest the recent article by Korn and his coworkers¹ concerning quinaprilat during cardioplegic arrest to prevent ischemia-reperfusion injury. They have demonstrated that quinaprilat enhances recovery of the ischemic myocardium and suggests that its protective mechanism may be due to the increased coronary blood flow during the early reperfusion period. My group at the Boston Medical Center has recently published a study that helps to identify the mechanisms for the beneficial effects of quinaprilat.²

In an intact porcine model involving ischemia and reperfusion with cardioplegic arrest on cardiopulmonary bypass, hearts

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treated with the high-affinity tissue angiotensin-converting enzyme (ACE) inhibitor quinaprilat had significantly better recovery of regional wall motion, a lower incidence of ventricular arrhythmias, and a smaller infarct size than controls. When the low-affinity tissue ACE inhibitor enalaprilat was administered, the decrease in infarct size was less (24% enalaprilat vs 14% quinaprilat; $P < .0001$). The beneficial effects of quinaprilat's action could be explained by its superior preservation of endothelial function, since quinaprilat-treated hearts had significantly greater coronary arterial relaxation in response to bradykinin (66% quinaprilat vs 32% enalaprilat; $P < .0001$). When the bradykinin antagonist HOE140 was added to the quinaprilat-treated hearts, the protective effect on endothelial function was abolished and these hearts had a significant increase in infarct size (48% quinaprilat + HOE vs 14% quinaprilat; $P < .0001$).

I agree that ACE inhibitors enhance myocardial protection during cardioplegic arrest. However, ACE inhibitors are not all equally protective. ACE inhibitors with high tissue ACE inhibition such as quinaprilat result in better preservation of endothelial function, which decreases ischemic necrosis. This improvement in endothelial function appears to be bradykinin-mediated. I concur with Korn and coworkers that high-affinity tissue ACE inhibitors may play an important role in enhancing cardioplegic protection during coronary revascularization.^{2,3}

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Tracheal replacement

To the Editor:

It is encouraging that efforts continue to produce a "tissue-engineered trachea."¹ since this is about the only technique of the many attempts at tracheal replacement by a variety of approaches over more than 50 years that seems to offer any real promise.² I question, however, whether the experiments reported succeeded in forming "the structural component of a functional tracheal replacement."

Although visually resembling a trachea, the construction described failed to function (in sheep) over 5 and 7 days in 2 animals (with resultant malacia) or longer than 2 days in 4 animals (with stenosis). The authors' findings suggest that inflammatory response might be responsible for degeneration of the graft. Cell density and hydroxyproline content support this hypothesis. Conversion toward scar probably had begun, even with survival of some cartilage. The authors also allude to future advisability of assuring blood supply, which may indeed be a critical factor in this early deterioration of the implant. One of the keys to failure, even of autotransplanted free tracheal segments of any significant length, has been failure of blood supply, despite muscular or omental pedicles. Very short tracheal grafts, of course, are of no potential clinical use.

A subject that is deferred by the authors to later experimentation, perhaps with engineered epithelium, is the lack of epithelial coverage in these experiments. In time this would lead to graft obstruction by cicatrization. Epithelium in general has failed to recolonize any extended length of tracheal replacement by ingrowth from the normal tracheal ends alone.

I shall look forward with interest to future reports by this and other groups using tissue engineering in an effort to meet the challenge of fashioning a usable tracheal prosthesis for the rare occasion when it is indeed needed.

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Cardiac herniation after intrapericardial pneumonectomy

To the Editor:

The brief communication by Baisi and associates¹ reporting a case of cardiac herniation after intrapericardial pneumonectomy is almost identical with a case report in another journal.² Both reports fail to mention that chest tubes were deployed after the pneumonectomy. (They can clearly be seen in their respective illustrations.)

The previous case^{2,3} instigated a spirited correspondence^{4,5} centering on the role, if any, of chest tubes after pneumonectomy and their relation to cardiac herniation. The current case also fails to mention a chest tube in the left posterior thoracic gutter clearly seen on the computed tomographic scan (see Figure 1). No mention is made of its presence or mode of use. Rather the authors emphasize (without justification or substantiation in my mind) that the induction chemotherapy was to blame. Actually the initial closure of the postpneumonectomy pericardial defect failed because the sutures, placed under tension, cut through—a mechanical problem, not a neoplastic or chemotherapeutic problem.

The empty postpneumonectomy space is not to be equated with that containing variable amounts of lung tissue, which require chest tubes attached to suction. Using such continued suction in a postpneumonectomy space even for a short period of time is to court disaster produced by excessive mediastinal shift and, in some cases, cardiac herniation. Despite the opinion of Walker⁴ of the hazards of chest tubes after pneumonectomy, Cooley⁵ expressed it well when he said that he routinely used tubes after pneumonectomy but that "when not in use the tube should be clamped." [italics mine.]

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