remarkable from 1 week after apex-sacrificing VRS but not in apex-sparing VRS. Drs Lunkenheimer and Anderson described in their letter that resection of any part of LV wall, including the septum, is tolerated without impairing LV function when the left ventricle is dilated. However, septal anterior ventricular exclusion or pacopexy reduces the septal and anterior wall without amputating the LV apex and yields good clinical results.[13,14] We think those results are compatible with our study.

In addition, results of some clinical cases that underwent the apex-sparing VRS in our unit have been excellent so far. Thus we believe that it is important to reduce the LV volume in dilated left ventricle with severe LV dysfunction. Apex-sparing VRS may improve the clinical results.

Although the hypothesis by Dr Torrent-Guasp and colleagues is very interesting,[15] there is no scientific proof so far. Therefore the relationship between cause and result in the improved LV function after the apex-sparing VRS and preservation of single muscle band is still unknown, and further study will be required.

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Study of warm perfusion rather than cardioplegia

To the Editor:

I read the article by Mallidi and colleagues,[1] "The Short-term and Long-term Effects of Warm or Tepid Cardioplegia," and raise the following concern. The title is not descriptive of the protocols. Rather than isolated cold or warm cardioplegia, the article really describes cold and warm total-body and cardiac perfusion strategies. The article states, "In the warm or tepid blood cardioplegia group, the systemic temperature was maintained at 33°C to 37°C, and the blood cardioplegia was delivered at a temperature of 37°C. In the tepid cardioplegia group, the systemic temperature was permitted to drift passively during the operation to 32°C to 34°C. The temperature of the cardioplegia was 28° to 30°C. In the cold cardioplegia group, the systemic temperature was actively cooled to 25°C to 32°C, and the blood cardioplegia was actively cooled to a temperature of 5°C to 8°C."

Other combinations may have similar results, for example a warm corporeal perfusion strategy (drifting without active cooling) and cold cardioplegia. The data do not preclude such a result.

This group has done a nice job scientifically studying and promoting warm perfusion and protection strategies. I think that describing their technique as "warm blood cardioplegia" does not describe the strategy adequately, and "warm perfusion strategy" might be more accurate.

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Reply to the Editor:

Dr Savage correctly points out that the article published in the March issue of the Journal by our group was not simply a comparison of cold versus warm or tepid cardioplegia, but rather a comparison of the strategy of warm or tepid cardioplegia versus cold cardioplegia. The three cardioplegic and systemic perfusion strategies used in patients undergoing isolated coronary artery grafting surgery at our institution were described in detail in the article. Other possible cardioplegic and systemic perfusion strategies (such as warm systemic perfusion with cold cardioplegia, tepid systemic perfusion with cold cardioplegia, systemic hypothermia with warm or tepid cardioplegia, and so on) were not used in our institution.

One potential reason for avoiding strategies that have mixed cardioplegia and systemic perfusion temperatures (cold for one with warm or tepid for the other) is the concern that the myocardial temperature in the mixed temperatures situation would likely result in highly variable and inconsistent myocardial temperatures.[1] It has been demonstrated that the delivery of cold cardioplegia results in decreased subepicardial and midwall ventricular perfusion. This datum, coupled with information that myocardial temperature is variable with cold cardioplegia, suggests that myocardial protection may be compromised by such a mixed temperature environment.[2]

However, others have shown no increase in the release of myocardial injury markers (troponin I and T) after normothermic systemic perfusion relative to hypothermic systemic perfusion when a regimen of cold cardioplegia with topical cooling was used for myocardial protection, and acceptable clinical results are possible.[3] Furthermore, acceptable clinical results have been reported with the technique of cold cardioplegia with warm systemic perfusion.[4] Thus considerable controversy continues to exist regarding whether cold cardioplegia with warm systemic perfusion is harmful; however, there are no studies demonstrating its superiority to the strategy of warm cardioplegia with tepid systemic perfusion.

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Nickel allergy to the percutaneous patent foramen ovale occluder and subsequent systemic nickel allergy

To the Editor:

We read with great interest the article "Systemic Allergic Reaction to the Percutaneous Patent Foramen Ovale Occluder" by Fukahara and colleagues.[1] In that article, Fukahara and colleagues[1] described the case of a 37-year-old patient who underwent patent foramen ovale closure with the Star device (Cardia Inc, Burnsville, Minn). In the following 2 months, the patient began demonstrating hypersensitivity to the device, confirmed by skin patch testing as an allergy to nitinol. Removal of the device ultimately led to this patient's recovery.

In October 2001, we treated an 11-year-old boy who had undergone transcatheter closure of a secundum type atrial septal defect with the HELEX Septal Occluder (W.L. Gore and Associates, Flagstaff, Ariz).[2] Similar to Fukahara and colleagues' patient, our patient had a similar reaction to the device; he ultimately had positive skin patch test results for nickel allergy. This patient underwent uneventful removal of the device as well but also required subsequent removal of the sternal wires, which had trace amounts of nickel. This patient was initially unavailable for follow-up at our institution. To our knowledge, however, he has had an otherwise uneventful recovery.

We thank Fukahara and colleagues[1] for their report on the subject. We encourage the cardiothoracic community at large to report such events to determine objectively the incidence of adverse outcomes that may be associated with these emerging technologies.

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Activated prothrombin complex concentrates and recombinant factor VIIa in the bleeding patient: Are they appropriate and safe?

To the Editor:

I read with great interest the article of Bui and colleagues,[1] "Fatal Thrombosis After Administration of Activated Prothrombin Complex Concentrates in a Patient Supported by Extracorporeal Membrane Oxygenation Who Had Received Activated Recombinant Factor VII," in the October 2002 Journal. Many issues related to the administration of activated prothrombin complex concentrates presented by this case were too simply discussed.

First, the patient had evidence of disseminated intravascular coagulation both before the cessation of heparin and after its discontinuation, with prolonged prothrombin time and partial thromboplastin time. No data are available regarding the patient's platelet count before or after surgical intervention. Both recombinant factor VIIa and factor VIII bypassing activity (FEIBA) were used off-label, about which the authors made little comment. Cardiac surgery with extracorporeal membrane oxygenation is known to activate clotting, and death might well have occurred even without either biologic agent. The cardiac death may not have been from thrombus, but of another origin; because no autopsy was performed, however, all this is pure speculation.

The authors stated with great conviction that thrombosis with recombinant factor VIIa is less common than with FEIBA. Continued hemorrhage itself is a cause of activation of coagulation. Agents known to aid in stopping hemorrhage in patients with coagulation defects have caused thrombotic episodes.

Almost all the life-threatening thrombotic events with FEIBA have occurred with doses exceeding those recommended.[2] Bui and colleagues[1] provided no data regarding the dose of FEIBA given. Thrombotic events, including myocardial