At our institution, most patients eligible for LVR undergo a preoperative EP study. In patients with spontaneous or inducible ventricular tachycardia (VT), we perform endocardial resection and cryoablation. In patients with preoperative clinical VT, we perform an EP study before hospital discharge, and in patients with inducible only VT, we perform an EP study 3 to 6 months after the operation. In case of postoperative clinical or inducible VT, we recommend ICD implantation. We have recently reported our experience in a series of 53 consecutive patients undergoing LVR and surgical intervention for VT. The success rate in terms of VT control was 90%. This finding is comparable to the results previously reported by Di Donato and colleagues and Mickleborough and associates.

**Treat the cause, not the symptoms.**

ICD firing is associated with a certain amount of discomfort for the patient. ICDs indisputably save lives, but the price can be high both in terms of money and patient well-being. Therefore the aim must be to eliminate the need for ICD. By adding specific antiarrhythmic surgical procedures, such as endocardectomy and cryoablation, in patients undergoing LVR, we have a potentially curative treatment option at our disposal. In our view an EP study is necessary after LVR, to identify surgical failures in which ICD therapy is warranted.

In our opinion patients scheduled for LVR should be assessed for ventricular arrhythmias, and if present, specific arrhythmia surgery should be performed concomitantly, and the postoperative result should be verified by means of EP studies. With this protocol, implantation of an ICD will not be needed in most patients after LVR including surgical intervention for VT.

**References**


**Reply to the Editor:**

As stated in the article, 30 patients had implantable cardioverter-defibrillators inserted preoperatively, and the indication for the majority of these patients was secondary prevention, having had either a documented ventricular arrhythmia or aborted sudden death. Of these 30 patients, 2 had aborted sudden cardiac death, 3 had sustained ventricular tachycardia, and the remainder presumably had positive electrophysiologic (EP) studies. For groups 2 and 3 of our series, we do not have accurate data on who underwent EP studies preoperatively.

Dr Sartipy’s group performs EP studies preoperatively. This approach is used to guide endocardial resection or cryoablation. Many of our patients (13%) underwent cryoablation for arrhythmias. However, the main indication for left ventricular reconstruction (LVR) was heart failure, rather than intractable arrhythmias.

LVR definitely has a role in the treatment of ventricular arrhythmias, but in patients with severe left ventricular dysfunction, border zones between scar and viable myocardium might provide arrhythmic substrate. In addition, patients in our series had evidence of marked left ventricular remodelling, with arrhythmic substrate in areas remote to the site of LVR.

Against that, the Coronary Artery Bypass Graft Patch Trial failed to show a reduction in mortality when patients with markers for increased risk of ventricular arrhythmia underwent implantable cardioverter-defibrillator implantation at the time of coronary artery bypass grafting. This has been attributed to a reduction in the risk of arrhythmic death as a result of revascularization. This indicates that perhaps the most important procedure to reduce arrhythmias is surgical revascularization.

Further prospective studies are required to elucidate the optimal strategy in this complex group of patients.

**Left ventricular assist device in heart failure**

**To the Editor:**

I read with great interest the article by Nicholas C. Dang and colleagues, wherein they report their experience with left ventricular assist device (LVAD) in patients with chronic congestive heart failure. There are various mechanical circulatory devices employed currently as a bridge to transplantation. The authors report their experience with the HeartMate (Thoratec Corp, Pleasanton, CA) device; however, the type of device engaged is not mentioned. It is pertinent to note that of HeartMate LVADs, the single-lead vented electrical devices have been linked with the best posttransplant survival rates. Even as vigilance for the predictive factors will help in patient selection, improved clinical outcome should also be sought by careful timing of transplantation following LVAD insertion. By instituting patient support and rehabilitation for at least a month following the implantation, significant normalization of end-organ func-
tation and improvement in physiologic status may be achieved to improve survival following the transplant.3

A possible scoring system for better selection of patient criteria is sought. In this context the Acute Physiology and Chronic Health Evaluation II (APACHE II) scoring system, a multiparameter, physiology-based predictor of outcome, might be helpful. It can aid in both selection and timing of LVAD implantation, particularly in patients not meeting normal hemodynamic criteria for LVAD usage.4

Development of right ventricular failure often causes poor results in patients with LVADs. It is important to take into consideration the predictive factors including the need for circulatory support, female gender, and nonischemic etiology, along with the hemodynamic alterations including low pulmonary artery pressure and low right ventricle stroke work index, that might indicate poor right ventricular outcome.5 Careful observation of the above would assist both in patient selection and clinical handling of isolated LVAD implants.

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References

Reply to the Editor:
We thank Dr Ashraf for his comments on our recent article concerning the use of left ventricular assist devices (LVADs) in patients with chronic congestive heart failure.1 His letter invites us to discuss several important points.

First, to clarify the device type used predominantly at our center and used exclusively in our study, we favor the HeartMate XVE (single-lead vented electric) LVAD (Thoratec Corp, Pleasanton, CA) for its relative ease of implantation, durability, and lack of need for systemic anticoagulation. Our long-term experience with this device has paralleled an evolution in design, resulting in improved bridge-to-transplant and posttransplant survival rates.2

Next, we could not agree more with Dr Ashraf’s observation that the timing of transplantation following LVAD insertion plays a critical role in determining survival. Our own unpublished data show near normalization of blood urea nitrogen, creatinine, and liver function values at approximately 3 months of support time, bolstering the concept of enhanced end-organ perfusion by the LVAD. Moreover, the smooth transition to cardiac rehabilitation and nutritional optimization throughout the recovery period are of critical importance.

Although we do not employ the Acute Physiology and Chronic Health Evaluation II (APACHE II) scoring system ourselves, we use similar clinical and laboratory-based parameters to select LVAD candidates. All patients referred for LVAD are generally refractory to maximal medical therapy, which often includes the use of intravenous inotropes, vasopressors, and intra-aortic balloon pumps. Exclusion is therefore done on the basis of such factors as ventilatory status, elevated pulmonary pressures, and protracted prothrombin time.

Despite the physiologic benefits of LVADs, even as they apply to the right ventricle, right heart failure (RHF) occurs in approximately 15% to 20% of patients postoperatively.3,4 Multiple studies have sought to identify demographic and hemodynamic risk factors predictive of the development of RHF, but in practice, these parameters often exhibit variable outcomes. Although the best treatment for RHF is avoidance, when it does become manifest, a low threshold should be maintained to promptly start inotropic (ie, milrinone) and pulmonary vasodilator (ie, nitric oxide) therapy, with a right ventricular assist device close at hand.

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References

Apoptosis in ischemic spinal cord injury
To the Editor:
We read the article of Suzuki and associates1 titled “Experimental study on the protective effects of edaravone against ischemic spinal cord injury” with great interest. They studied the effect of a free radical scavenger named “edaravone” in a rabbit model of transient aortic occlusion and claimed its protective effect on the ischemia-reperfusion injury of spinal cord by suppressing the level of reactive oxygen species (ROS). We congratulate Suzuki and associates for their excellent study. We think that the introduction of microdialysis method to determine the production of ROS in the neuronal tissue after transient ischemia for the first time in the literature by the authors is a great contribution to our current knowledge.

Recently, data has accumulated that programmed cell death or apoptosis of motor neurons in spinal cord after transient ischemia is an outstanding mechanism of postoperative paraplegia or paraparesis.2,3 The neuronal injury following transient aortic occlusion occurs in 2 phases, namely, early and delayed. Ischemic insult in spinal cord

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