of blood per minute. Although the anuria that had been present since cardiac arrest persisted, the pulmonary edema cleared, as indicated by plain chest x–ray and auscultation. We discontinued mechanical support after 4 days of continuous use, but the patient remained in a coma and died.[4]

At the beginning of 1966, Dr. DeBakey and I started implanting paracorporeal VADs at the Methodist Hospital. A patient from Mexico underwent a double valve replacement but could not be weaned from extracorporeal circulation. Then we implanted a paracorporeal VAD from the left atrium to the right subclavian artery. After support by the VAD for 10 days, at a flow rate up to 1,200 mL/min, the patient recovered, which made this the 1st successful use of a VAD for postcardiotomy shock.[5,6] Several years later, she died in Mexico in a car accident.

The 1966 paracorporeal VADs were lined with Dacron velour to create a neoendocardium that would deter thromboembolism.[7] Everything that followed this early clinical research in the field of assisted circulation is well known and need not be repeated here.

Domingo Liotta, MD,
Dean,
School of Medicine,
MoRón University,
Machado 914,
Buenos Aires (1708), Argentina

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Secondary Cross-Clamping and Blood Cardioplegia for Refractory Ventricular Fibrillation after Aortic Cross-Clamp Removal

To the Editor:

We wish to draw attention to a very rare phenomenon, refractory ventricular fibrillation (VF) after the removal of an aortic cross-clamp, which can be difficult to treat despite persistent defibrillation with direct-current (DC) countershock and the administration of multiple antiarrhythmic agents.

As an example, we present the cases of 3 of our patients who developed VF in this situation. Two patients with coronary artery disease and low ejection fractions (0.30) underwent coronary artery bypass grafting (CABG), and 1 patient with aortic stenosis (118-mmHg gradient) and advanced constrictive hypertrophy underwent mechanical valve replacement (#23, St. Jude Medical, Inc.; St. Paul, Minn). After the induction of anesthesia and administration of intravenous heparin, all patients were placed on cardiopulmonary bypass (CPB); mild hypothermia (33 °C) was induced during CPB. In all 3 patients, myocardial preservation was achieved by both antegrade and retrograde administration of hyperkalemic cold-blood cardioplegic solution. Lidocaine (100 mg) was administered intravenously before the aortic cross-clamp was removed. Rectal temperatures were above 36.6 °C after the removal of the cross-clamp.

In each case, the cardiac rhythm after cross-clamp removal was found to be VF. The rhythm could not be corrected despite repeated attempts to defibrillate with DC countershock (from 15-36 J). An additional bolus of lidocaine (100 mg) was administered intravenously, followed by continuous lidocaine infusion in each case. The pH, oxygenation, and electrolyte levels in the blood gas samples were all within normal limits. In each patient, we cross-clamped the aorta again and administered normothermic blood cardioplegia (1,000–1,700 cc). Sinus rhythm resumed when the cross-clamp was removed. An apical vent was placed in the left ventricle of the patient who underwent aortic valve replacement. This patient was supported by CPB for 60 minutes after the 2nd removal of the cross-clamp. One of the patients undergoing CABG required the administration of dual inotropic

agents (dopamine and dobutamine), but was weaned from CPB 26 minutes after the 2nd removal of the cross-clamp. The other patient undergoing CABG required triple inotropic agents (dopamine, dobutamine, and epinephrine). An intraaortic balloon pump (IABP) was placed, and his sternum was left open. Cardiopulmonary bypass was stopped 72 minutes after the 2nd cross-clamp removal. A temporary patch was placed over the open sternum, and the patient was taken to the intensive care unit. One day later, he was returned to the operating room, the sternum was closed, and the IABP was withdrawn. All 3 patients were discharged from the hospital with no further problems.

Ventricular fibrillation that is refractory after removal of the aortic cross-clamp, despite defibrillation and the administration of various antiarrhythmic agents, can be triggered by hypokalemia, acidosis, hypothermia, hypotension, or arrhythmogenic foci. Correction of these underlying conditions and the administration of antiarrhythmic agents generally ensure a successful return to sinus rhythm.

In some patients, however, the treatment of refractory VF is more difficult. Robicsek[1] reported the successful treatment of sustained VF in 6 patients by use of secondary aortic cross-clamping and the administration of crystalloid cardioplegic solution to induce ischemic arrest, followed by the introduction of a pacemaker that was set in the ventricular mode.

We successfully restored sinus rhythm in 3 patients, all of whom initially suffered from refractory ventricular fibrillation upon separation from cardiopulmonary bypass. Therefore, we advocate the use of secondary aortic cross-clamping and the administration of blood to induce cardioplegia when standard treatment options fail.

Ayda Turkoz, MD,
Huseyin I. Toprak, MD, and
M. Ozcan Ersoy, MD,
Department of Anesthesiology,
Inonu University Turgut Ozal Medicine Center;
and
Oner Gulcan, MD, and
Riza Turkoz, MD,
Department of Cardiovascular Surgery,
Inonu University Hospital;
Adana, Turkey

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Transaortic Double Valve Replacement

To the Editor:

I read with interest the case report[1] on transaortic mitral and aortic valve replacement with total preservation of the mitral valve chordae. Without any intent of detracting from the surgeons' well-deserved credit for having performed this procedure successfully in 4 cases, I wish to comment on a few issues.

First, their decision to replace the mitral valve has left me perplexed. Should mere thickening of the mitral valve, especially with only mild incompetence, have necessitated replacement? Second, the logic behind releasing adhesions to expose both the atria and the venae cavae also eludes comprehension, as these steps appear to be dispensable. Third, the decision to preserve all the chordae was noble; however, the availability of a St. Jude Medical mechanical valve for use in the mitral position should have enabled the implantation of that prosthesis with total preservation of the native mitral valve[2-4]—which might have been technically simpler. Last, in the presence of mitral stenosis with atrial fibrillation, the authors' approach precludes the detection of small, echocardiographically undetected left atrial thrombi and also the obliteration of the left atrial appendage.

Amit Banerjee, MCh,
Department of Cardiothoracic Surgery,
Govind Ballabh Pant Hospital,
New Delhi, India

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This letter was referred to Prof. Kumar, who replies in this manner:

The decision to replace a thickened, mildly regurgitant mitral valve was made because of previous open commissurotomy in this patient and to avoid a 3rd, possibly more hazardous, open-heart operation, in consideration of the young age of the patient.

We routinely mobilize the entire heart in redo surgery to provide good exposure, better topical cooling,