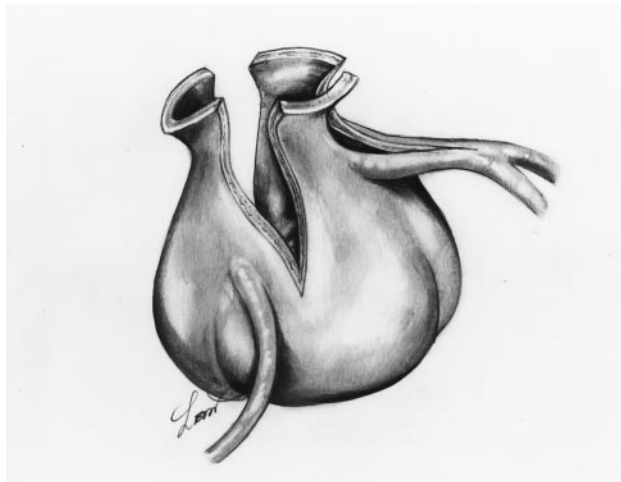
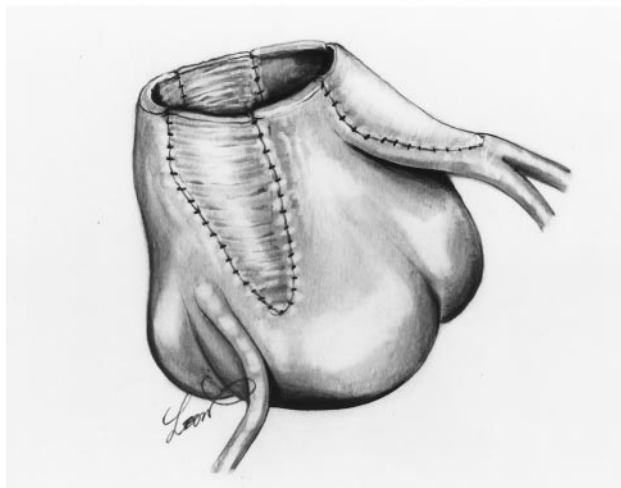




A



B



C

Fig 1. (A, B) An incision is made into each sinus across the stenotic ring. The incision in the left sinus is extended into the left main coronary artery as far as its bifurcation. (C) Three patches are sewn in to complete the repair.

aortic stenosis while restoring normal anatomic aortic root geometry.

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Cerebral Embolism Associated With Left Ventricular Assist Device Support and Successful Therapy With Intraarterial Urokinase

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A patient with a bioprosthetic aortic valve sustained a cerebral embolism during support with an implantable left ventricular assist device. This was lysed with intraarterial urokinase with complete resolution of the neurological deficit. Subsequently the patient underwent heart transplantation and remains neurologically intact. This case report is the first successful use of thrombolysis for cerebral embolism associated with a mechanical assist device.

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A 63-year-old male patient with dilated cardiomyopathy (left ventricular end diastolic dimension 6.6 cm and ejection fraction of 10%) who was inotrope dependent

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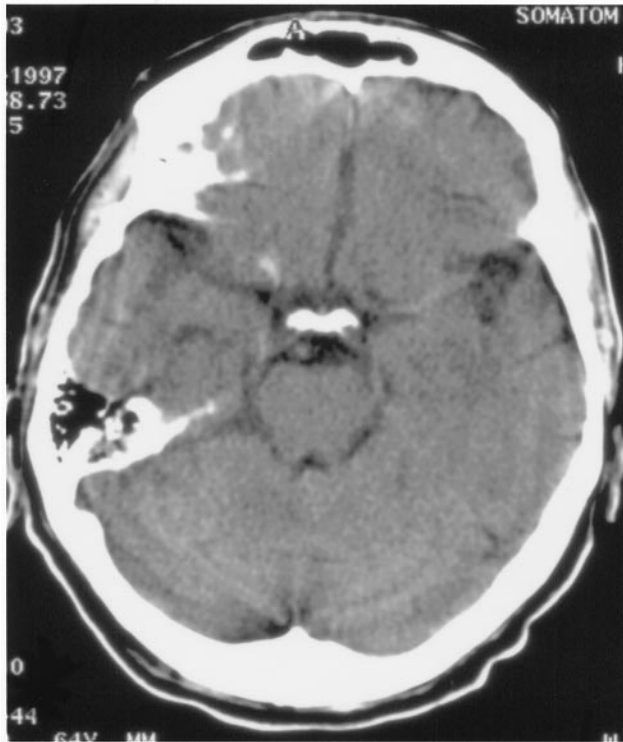


Fig 1. CT scan showing thrombus in left middle cerebral artery.

dent with severe mitral and tricuspid regurgitation and severe aortic valve stenosis (valve area of 0.7 cm²) underwent aortic valve replacement with 25 mm Carpentier Edwards pericardial valve, mitral valve repair with a 26 mm Cosgrove-Edwards ring and Alfieri stitch, partial left ventriculectomy, De Vega tricuspid valve repair, reverse saphenous vein graft to the left anterior descending artery and closure of the left atrial appendage. The patient was a transplant candidate and this procedure was done as a possible bridge or alternative to transplantation. Prior consent was obtained for implantation of a left ventricular assist system as a back up to this high-risk procedure. As the hemodynamics remained poor off bypass, the Novacor left ventricular assist system (Novacor LVAS) was implanted for circulatory support. The patient made an uneventful recovery and was transferred to an acute rehabilitation facility. Maintenance anticoagulation consisted of aspirin 325 mg once a day and Coumadin to maintain an international normalized ratio of 2.5-3.5. Fifty days post implantation of the Novacor LVAS he developed abrupt onset of dense right hemiplegia and aphasia. A CT scan showed thrombus in the left middle cerebral artery (Fig 1) and selective carotid angiography confirmed embolic occlusion in the left middle cerebral artery with retrograde flow through pial collaterals from the left anterior cerebral artery (Fig 2). Using a transfemoral arterial route, a 3.0 French micro catheter was placed into the thrombus in the left middle cerebral artery and 1.5 million units of urokinase were infused. Subsequent angiograms showed complete lysis

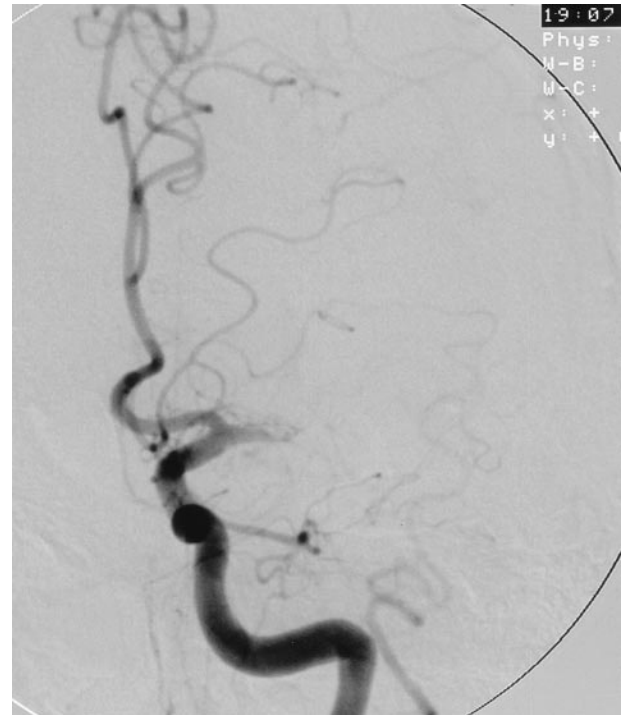


Fig 2. Selective carotid angiography confirms complete occlusion of left middle cerebral artery.

of the thrombus (Fig 3). Postprocedure the patient had mild left sided weakness that completely resolved in 24 hours. He underwent orthotic cardiac transplantation 5



Fig 3. Angiogram post urokinase infusion showing restoration of normal blood flow.

days later. A follow-up CT scan showed a small infarct in the left globus pallidus and internal capsule. At the time of transplantation, layered thrombus was noted on the ventricular side of the aortic valve. There was no thrombus in the left atrium or in the ventricular assist device or its cannulae.

Comments

The incidence of thromboembolic events associated with mechanical assist devices varies from 2% to 47% [1-6]. The presence of atrial and ventricular arrhythmias, a dilated left ventricle with preexisting thrombus, and the need for prosthetic valves in the assist devices makes it very difficult to identify the source of emboli. Infection also appears to increase the incidence of thromboembolic events [5]. With most devices, the use of an antiplatelet agent with or without anticoagulation is necessary. In spite of precautions, thromboembolic events and the resulting neurological sequelae remain one of the most devastating complications of mechanical circulatory support. The presence of a prosthetic valve in the aortic position is a risk factor for thromboembolic events [3]. During left ventricular support, the valve remains closed and may form thrombus on the aortic as well as the ventricular surfaces. Improvement in LV function or decreased pump output may result in ejection through this valve, showering the circulation with emboli. The use of a bioprosthetic valve or anticoagulation are not totally protective.

After routine CABG most CVAs are associated with atheroemboli, while strokes later in the postop period are thought to be cardiogenic in origin [7]. In the past, once patients developed neurologic symptoms in the postoperative period, the management consisted primarily of preventing recurrent embolic and minimizing complications from neurological deficits. In patients with dense deficits, such as the one reported, the prognosis for neurological recovery is poor. When severe deficits are present early after CABG, the mortality ranges from 20% to 60% [8]. Until recently there has been no direct treatment to reduce the extent of neurological injury. Thrombolytic recanalization of occluded arteries may reduce the degree of injury to the brain if it is done before the process of infarction has been completed. The National Institutes of Neurological Disorders and Stroke (NINDS) trial of intravenous recombinant tissue plasminogen activator showed despite a small increase in symptomatic intracerebral hemorrhage, treatment with tissue plasminogen activator within 3 hours of onset of ischemic stroke improved the clinical outcome at 3 months. There was no increase in mortality [9]. There were several other trials of intravenous thrombolytic use in acute ischemic stroke, the Australian Streptokinase (ASK) trial, the European Cooperative Acute Stroke Study (ECASS), the Multicenter Acute Stroke Trial-Italy (MAST-I) and the Multicenter Acute Stroke Trial-Europe (MAST-E). In these studies there appeared to be an increase in mortality in the treated groups, though the

survivors showed symptomatic improvement [10]. The reasons for this are unclear. However, in the NINDS trial, the time to institution of thrombolysis was less than 3 hours compared to the more than 4 to 6 hours in the other studies. Also the risk of intracerebral hemorrhage is increased in the presence of ischemia involving greater than one third of middle cerebral artery vascular territory on CT scan.

More recently the publication of PROACT, a controlled study of intraarterial local delivery of recombinant pro-urokinase demonstrate superior recanalisation of middle cerebral artery after thromboembolism when compared to placebo [11]. The theoretical yet unproven benefits of local delivery of drug is higher concentration of the drug at the site of embolus with lower systemic fibrinolysis.

A hopeless approach is no longer justified in postoperative patients suffering acute neurological events. As neuroradiologists develop an increasingly sophisticated array of devices to intervene in patients with acute strokes, our postoperative patients are ideally suited for rapid and timely intervention to reduce the mortality and disability of postoperative neurologic injury.

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