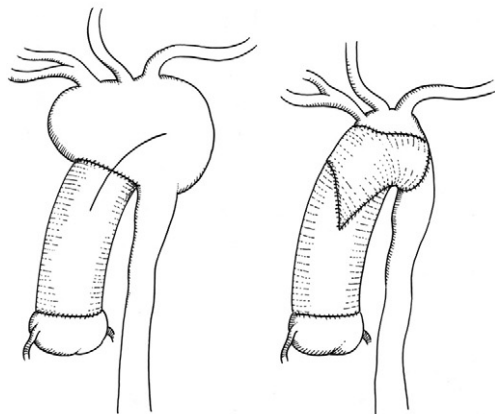


A



B

Fig 3. (A) Postoperative cardiac catheterization (26 months after first surgery) revealed severely dilated aortic arch (upper), with the aortic arch branches and descending aorta of normal diameter (lower). (B) Schematic representation showing the (left) aneurysmal dilatation of the aortic arch distal to the previous ascending aortic graft, and (right) the operative technique of prosthetic replacement of the previously replaced distal ascending aorta (proximal anastomosis) and replacement of the aortic arch until the isthmus region just before the proximal descending aorta (distal anastomosis).

phy, provides good mid-term results without the risk of aneurysm recurrence [6]. Long-term results of ascending aorta replacement in children have not been reported yet.

In conclusion, this report demonstrates that aneurysm of the ascending aorta and aortic arch can be isolated and progressive in infants and may be successfully treated by their replacement with an adult-sized synthetic graft.

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Reversible Unilateral Brain Edema Presenting With Major Neurologic Deficit After Valve Repair

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Brain edema after cardiac surgery is unusual and often asymptomatic. We encountered a 34-year-old man who had postoperative left flaccid hemiplegia and anosognosia after undergoing composite root replacement and closure of a patent foramen ovale. Computed tomographic and magnetic resonance imaging (MRI) scans showed profound white matter changes indicative of brain edema predominantly in the right hemisphere. His symptoms resolved spontaneously within 3 days with

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resolution of MRI abnormality. No evidence of cerebral infarction was documented on diffusion-weighted imaging and apparent diffusion coefficient mapping, or on the follow-up MRI obtained 25 days after presentation. The cause for the unilateral brain edema is unknown, but the patient's clinical course and imaging are supportive for a variant of a hyperperfusion syndrome or reversible encephalopathy. The outcome was excellent.

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Neurologic complications after valve surgery typically involve ischemic strokes [1-5]. Patients may have localized findings, such as a new speech abnormality or hemiparesis and rarely postoperative stupor from multiple hemispheric infarctions [1]. Brain edema after normothermic cardiopulmonary bypass has been described, but these patients have remained asymptomatic and changes were only detected with serial MRI scans [3]. We describe a patient with strikingly unilateral brain edema with complete resolution but presenting with a major neurologic deficit suggesting an ischemic stroke. We believe that this presentation has not been previously reported.

A 34-year-old man underwent uneventful composite root replacement and closure of patent foramen ovale under moderate hypothermia (34°C) with central aortic cannulation using a Terumo Capiox SX25 membrane oxygenator (Terumo Cardiovascular Systems, Ann Arbor, MI) and cardioplegic arrest. The patient had a history of prior graft replacement of the ascending aorta for acute DeBakey type I dissection, reoperation for aneurysmal dilatation of the aortic arch, and subsequent repair of Crawford extent II thoracoabdominal aortic aneurysm. The latter two procedures were performed at our institution under profound hypothermia with circulatory arrest, with the last having been performed 3 months previously.

Postoperatively, the patient was noted to have a left hemiparesis immediately on withdrawal of sedation. This was preceded by vigorous shaking of his leg. On examination, he did not open his eyes to pain but followed commands, had intact brainstem reflexes, a spontaneous clonus on the left side, and flaccid left arm and left leg weakness. There was no forced eye deviation or any facial asymmetry. A marked left-sided anosognosia was noted. Initial computed tomographic scan of the brain obtained on the day of surgery showed no clear abnormalities. An MRI scan of the brain (Fig 1) obtained the following day showed a striking pattern of signal change with diffuse areas of increased T2-signal throughout the

subcortical and deep white matter of the right cerebral hemisphere, and only minimal signal change within the left temporal and occipital lobes. There was no evidence of restricted diffusion to suggest acute infarction. The apparent diffusion coefficient map showed increased diffusion corresponding to the right hemispheric signal changes. The T2 signal change did not follow a specific vascular territory. The follow-up computed tomographic scan on postoperative day 3 showed areas of hypointensity in a similar distribution to that seen on the prior MRI scan. There was no evidence for intracranial hemorrhage. Twenty-four hours later, he was markedly improved, and no neglect was detected. His left arm was still weak, but he was able to lift it against gravity and above his head. When he was examined 48-hours later, all neurologic deficits did resolve. An MRI of the brain obtained on postoperative day 5 demonstrated development of some restricted diffusion within the splenium of the corpus callosum, and white matter within the mid and posterior right centrum semiovale. No gadolinium enhancement was noted. An MRI performed 25 days after the presentation showed complete resolution of the abnormalities without evidence for encephalomalacia or gliosis.

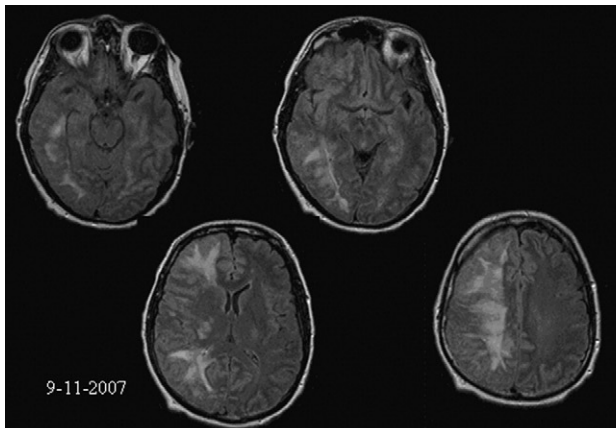
During his postoperative stay, he was on nitroprusside infusion, but at no point was there evidence of postoperative hypertension. He initially complained of postoperative headaches, but they resolved spontaneously. The carotid ultrasound studies were normal. Intraoperative transesophageal echocardiogram was unrevealing.

Comment

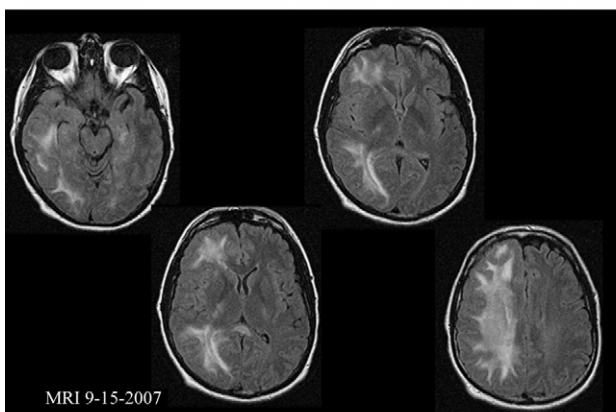
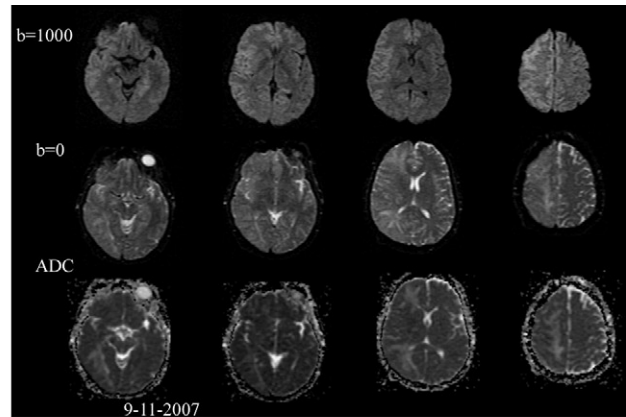
We present here for the first time unilateral brain edema after aortic valve replacement. Most remarkably, our patient presented with hemibody neglect, and a profound left hemiparesis suggesting an infarction in the middle cerebral artery territory. However, computed tomographic and MRI scans showed a transient emergence of brain edema in the white matter that was essentially unilateral and completely reversible. The MRI (diffusion weighted images with apparent diffusion coefficient mapping) was helpful in excluding ischemic infarction as the cause of these changes.

We can only surmise this abnormality represents a variant of hyperperfusion syndrome or reversible encephalopathy. Sudden forced dilatation of cerebral arteries may disrupt the blood brain barrier and cause extravasation of fluid and resulting edema. It remains puzzling in our case example that the blood brain barrier was breached on one side. Hypertensive surges causing a reversible encephalopathy syndrome known to present unilaterally in some cases can be considered, but the patient blood pressures were well controlled with a vasodilator [6]. Whether relative hypotension caused ischemia in the white matter, a known watershed area, is unlikely because again MRI characteristics of ischemia were absent. Inadvertently directing too much flow from the aortic cannula to the innominate artery can be considered as a possible mechanism, but a straight-tipped cannula was inserted into the Dacron neo-aorta (DuPont,

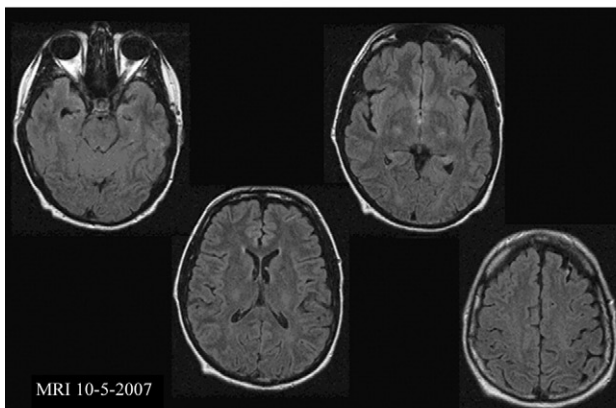
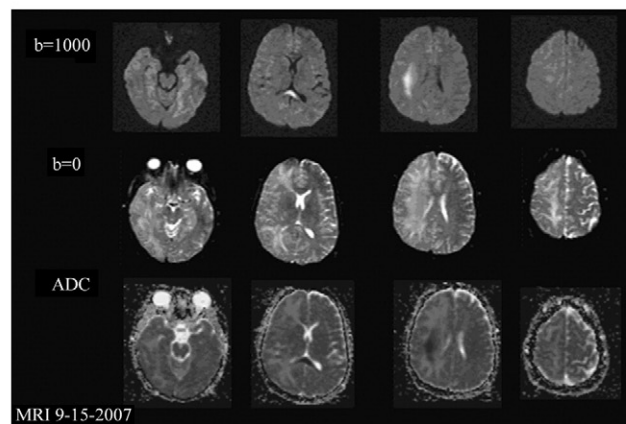
Dr Sundt discloses that he has a financial relationship with Atricute Inc, Bolton Medical, Jarvik Heart Inc, Medtronic Inc, Sorin Group/Carbomedics, St. Jude Medical, Thoratec Corporation, W. L. Gore & Associates Inc, and Boston Scientific Corp.



A



B



C

Fig 1. Serial magnetic resonance imaging (MRI) examinations of the brain showing the development of reversible signal change predominantly involving the right cerebral hemisphere. (A) An MRI obtained on the day after surgery demonstrates diffuse areas of increased T2 signal within the right hemispheric white matter. Diffusion-weighted images including ADC map are negative for acute ischemia, and show increased diffusion in the areas of signal change consistent with edema. (B) An MRI obtained on postoperative day 5 demonstrates development of restricted diffusion signal (confirmed on the ADC map) within the splenium of the corpus callosum and the right centrum semiovale. No pathologic enhancement is noted after administration of the gadolinium. The nonenhancing areas of T2 signal change in the right hemisphere have increased slightly and become more confluent since the prior MRI. (C) All changes are resolved on the MRI study obtained on postoperative day 25 without evidence for developing encephalomalacia or gliosis.

Wilmington, DE) in this patient and this makes maldistribution of flow to one brachiocephalic vessel unlikely.

Diffusion-weighted MRI scans after cardiac surgery are rarely performed. The largest study published from John Hopkins University involved 14 patients who underwent diffusion-weighted MRI scans, but concentrated largely on ischemic deficits. Diffusion-weighted MRI scan was more sensitive in documenting ischemic changes than a computed tomographic scan. Two patients had a fluctuating deficit, but the diffusion-weighted MRI scan

showed a clear diffusion-weighted abnormality suggesting an infarction [5]. Other studies have noted that aortic valve replacement is associated with high incidences of postoperative diffusion-weighted image lesions, but again, all of these abnormalities are ischemic in origin.

Reversible encephalopathy syndrome can have atypical distributions and manifestations, as noted in two recent large series [7, 8]. The distribution of reversible encephalopathy syndrome is not limited to the posterior cerebral hemispheres and has been shown to involve the

remainder of the cerebral hemispheres, brainstem, pons, and cerebellum. These patient series have also shown that reversible encephalopathy syndrome is not limited to white matter, but can involve cortex and deep nuclei. Awareness of variations in the appearance of reversible encephalopathy syndrome is important.

The true mechanism of the reversible white matter changes in this patient after aortic valve surgery remains unclear. The clinical course and imaging are supportive for a variant of a hyperperfusion syndrome-reversible encephalopathy. This case is of clinical interest due to the complete resolution of profound neurologic and MRI abnormalities that initially may have been clinically confounded as cerebral infarction.

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Percutaneous Ventricular Assist Device Support During Off-Pump Surgical Coronary Revascularization

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Intraaortic balloon pump counterpulsation has been used for mechanical circulatory support in cardiogenic shock

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patients, but percutaneous left ventricular assist devices can provide superior circulatory support in the same group of patients. We describe the case of a patient in cardiogenic shock after a myocardial infarction. A percutaneous ventricular assist device was used to provide immediate active hemodynamic support, and, because the patient's condition necessitated surgical revascularization, percutaneous left ventricular assist device support was continued during off-pump coronary artery bypass.

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Cardiogenic shock can occur as a consequence of various conditions and diseases, including acute myocardial infarction, decompensated chronic heart failure, fulminant myocarditis, acute valvular disease, or postcardiotomy injury. The current in-hospital mortality for cardiogenic shock ranges between 40% and 80% [1]. Emergency revascularization in patients who have had an acute myocardial infarction has been shown to improve their long-term survival; however, no study to date has demonstrated a successful strategy for improving short-term survival in this patient population [2, 3].

Traditionally, intraaortic balloon pump (IABP) counterpulsation has been used for mechanical circulatory support in cardiogenic shock patients, but IABP support can only modestly increase cardiac output and coronary blood flow, and it cannot provide total circulatory support in the setting of hemodynamic collapse [4].

Percutaneous left ventricular assist devices (pVADs) can provide superior circulatory support compared with the IABP in patients with cardiogenic shock [5]. They may be used for hemodynamic support of patients undergoing high-risk percutaneous coronary interventions [6]. They have also been used as bridges to recovery in the setting of acute fulminant myocarditis and as short-term bridges to transplantation in patients with acute decompensated heart failure [7].

We describe the case of a patient in cardiogenic shock after myocardial infarction. A pVAD was used to provide immediate active hemodynamic support, and, because the patient's condition necessitated surgical revascularization, pVAD support was continued during off-pump coronary artery bypass.

A 59-year-old man with a history of poorly controlled diabetes mellitus, hypertension, dyslipidemia, and tobacco use was admitted to another hospital for acute-onset chest pain. He had already experienced an ST-segment-elevation myocardial infarction of the anteroseptal region, complicated by acute pulmonary edema. In the cardiac catheterization laboratory, rapid decompensation necessitated intubation and IABP insertion. A coronary angiogram revealed critical 95% stenosis of the distal left main coronary artery and occlusion of the proximal right coronary artery. A left ventriculogram revealed inferior akinesis and severe anterior hypokinesis. Emergency coronary revascularization was considered, but his rapid