

suffice for those with failure of the Fontan circulation [4]. Because of the passive flow of blood through the pulmonary vascular bed, inadequate preload is often the underlying cause of diminished cardiac output in the failing Fontan circulation. A systemic VAD does not address this fundamental issue and will not necessarily reduce the systemic venous pressure. Indeed, filling and function of the VAD is likely to be limited by inadequate preload.

Because of poor results with isolated systemic VADs for the failing Fontan circulation, some have constructed biventricular assist devices for use in patients in functional SV [4]. There is also a single report of a Fontan takedown and placement of a sub-pulmonary VAD [5], but this strategy is subject to significant challenges related to matching of programmed pulmonary blood flow to systemic cardiac output and has not been adopted in broader clinical practice. Providing biventricular support in this clinical setting has the advantage of reducing systemic venous pressure while augmenting cardiac output. This strategy addresses the fundamental difficulty with the failing Fontan circulation and, as in this case, may allow for the resolution of symptoms and end-organ dysfunction associated with failing Fontan physiology, thus improving suitability for transplantation. It is important to note that once stable on the TAH, our patient had a complete resolution of his plastic bronchitis prior to his transplant. By providing normal cardiac output and low systemic venous pressures, the TAH may allow for resolution of the myriad manifestations of the failing Fontan circulation, thereby facilitating rehabilitation and improving suitability for eventual transplantation.

The 70-cc TAH was utilized in our patient. This device is recommended for patients with a body surface area of 1.7 m² or greater. While many younger patients with failing Fontan circulations are too small to be eligible for this device, the Food and Drug Administration recently approved a 50-cc device suitable for smaller patients. As demonstrated in this report, these novel devices are well suited for the cohort of children and young adults with failing Fontan physiology and may become an increasing important tool in the long-term care of this unique and growing population.

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Ventricular Assist Devices as Rescue Therapy in Cardiogenic Shock After Subarachnoid Hemorrhage

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We review the journey to myocardial and neurologic recovery of a 42-year-old mother with severe acute cardiogenic shock and multiorgan failure after extensive subarachnoid hemorrhage, who was salvaged successfully using a CentriMag short-term biventricular assist device.

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Cardiac abnormalities occur in as many as 50% of patients with subarachnoid hemorrhage (SAH) [1]. Possible hypotheses include multivessel coronary spasm, microvascular dysfunction, and catecholamine-mediated injury [1]. The most severe form of cardiac abnormality, neurogenic stressed (or stunned) myocardium, occurs in 20-30% of patients [2]. Left ventricular (LV) dysfunction limits tolerance to hypervolemic, hypertensive, and hemodilutional ("triple-H") therapy that is classically instituted to reverse ischemic deficits from SAH-induced cerebral vasospasm [2, 3]. Inotropic therapy and intra-aortic balloon pump (IABP) counterpulsation have been implemented in this setting with variable success [4] and the management of cardiogenic shock, refractory to such measures, remains problematic. Ventricular assist devices (VADs) are now an established therapy for cardiogenic shock but their use in the SAH setting has been contraindicated owing to the need for systemic anticoagulation. We present the first successful salvage strategy using VADs in SAH-induced cardiogenic shock.

A 42-year-old mother, 4 months post partum, was found unresponsive in the community experiencing tonic-clonic seizures. Cardiorespiratory arrest (pulseless electrical activity) rapidly ensued, and return of spontaneous circulation was achieved after 4 minutes of cardiopulmonary resuscitation. The patient was airlifted to a

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secondary-care facility, where cranial computed tomography (CT) revealed extensive SAH (Fig 1). Further cranial imaging and interventions were not possible because of the patient's progressive hemodynamic instability. Transthoracic echocardiography (TTE) confirmed severe LV systolic dysfunction, prompting transfer to our center for consideration of IABP counterpulsation. On arrival, physical examination revealed a moribund, comatose (Glasgow Coma Score 3) and ventilated patient with hypoxemia, severe hypotension (blood pressure 65/35 mm Hg) despite maximal inotropic support (epinephrine at 1 $\mu\text{g}/\text{kg}/\text{min}$), tachycardia (125 beats/min), elevated central venous pressure (22 mm Hg), and peripheral vasoconstriction. Laboratory investigations demonstrated acute kidney injury (urea 11.3 mmol/L; creatinine 233 $\mu\text{mol}/\text{L}$), and liver injury (aspartate aminotransferase 668 IU/L; alanine aminotransferase 695 IU/L), severe metabolic acidosis ($[\text{H}^+]$ 90 mmol/L; lactate 7.0 mmol/L; base deficit of 16.9 mmol/L) and coagulopathy (international normalized ratio 2.21), confirming acute cardiogenic shock (INTERMACS 1: "crash-and-burn" profile). Chest radiographs showed gross pulmonary edema (Fig 2), and troponin-I levels were only moderately elevated (8.30 $\mu\text{g}/\text{mL}$), suggesting a diagnosis of neurogenic stressed myocardium [1]. Repeat TTE revealed global LV akinesis (ejection fraction [EF] 5%) with moderate right ventricular (RV) contraction, prompting immediate short-term VAD implantation.

To prevent propagation of intracranial hemorrhage, cardiopulmonary bypass and anticoagulant therapy were



Fig 1. Non-contrast cranial computed tomographic scan showing acute subarachnoid hemorrhage.

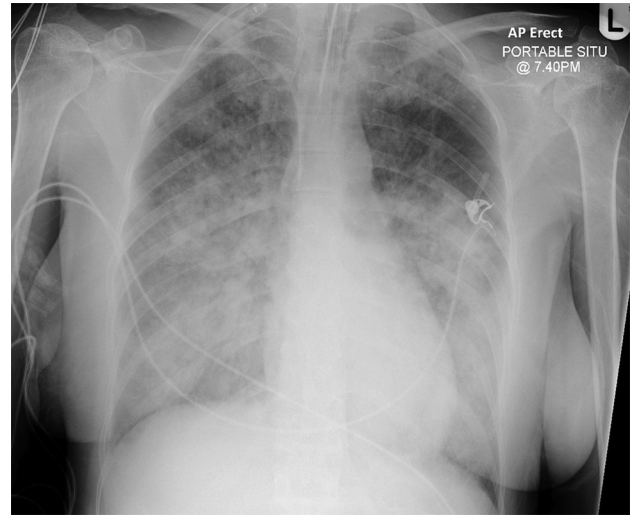
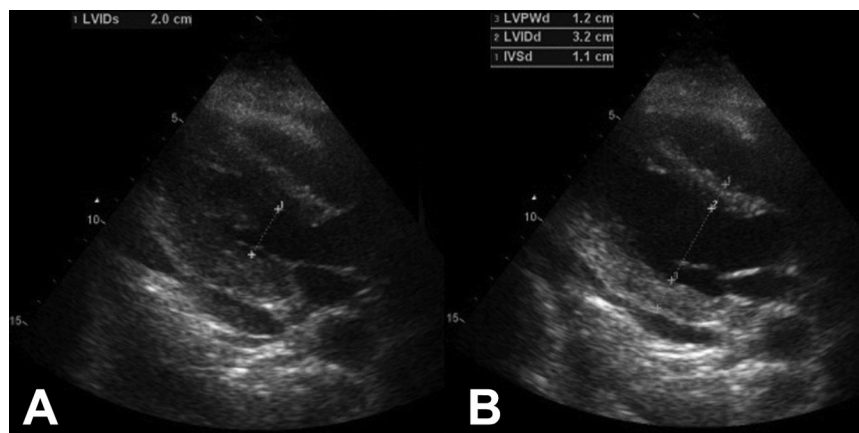


Fig 2. Chest roentgenogram on admission showing gross pulmonary edema.

avoided, and heparin-free VAD priming was performed. To avoid blood stasis within the VAD pumps, the pumps and associated lines were prepared before device implantation to allow immediate operation once the cannulas were in position. After median sternotomy, the left-sided VAD (CentriMag LVAD) was implanted first with the inflow cannula inserted into the left atrium through the right superior pulmonary vein, and the outflow cannula was placed in the ascending aorta. To prevent fluctuations in flow and line shuddering, the LVAD was set to operate at a relatively low pump speed (2000 rotations per minute [RPM]) until the right-sided VAD (CentriMag RVAD) was implanted. The RVAD inflow cannula was placed in the right atrium and the outflow cannula in the main pulmonary artery. Subsequent LVAD and RVAD flow rates were 5 L/min (3700 RPM) and 4.5 L/min (3000 RPM), respectively. Biopsy of the RV was performed, and pathologic examination demonstrated prominent myocardial adipose tissue with mild myocyte hypertrophy and myocytolysis.

Neuroprotective measures; avoidance of hypercapnea; and maintenance of adequate oxygenation, normothermia, and normoglycemia were initiated, and nimodipine and phenytoin therapy were commenced. Hypertensive episodes were avoided, and mean arterial pressure was maintained at 65 to 75 mm Hg. Urine output recovered immediately, and renal and liver biochemistry results normalized after 1 week. The patient was extubated within 72 hours and was alert and oriented by the fourth postoperative day. Given the thrombotic risk from the initial lack of anticoagulant therapy, epinephrine infusions were continued postoperatively to optimize residual ventricular ejection. CT angiography at 2 days showed no cerebral aneurysms. Heparin was therefore cautiously introduced 96 hours postoperatively, and the ventricles were completely unloaded. Target activated partial thromboplastin times were 35 to 40 seconds initially and 40 to 50 seconds after the first week. The

Fig 3. Transthoracic echocardiographic view showing myocardial recovery before explantation of ventricular assist devices. (A) Systole. (B) Diastole.



patient was completely weaned from inotropic support on postoperative day 8, when full anticoagulation was achieved and spontaneous ventricular ejection was demonstrable.

After 21 days of Bi-VAD support, TTE on low VAD flow rates and no inotropic support revealed a small LV (diastolic and systolic dimensions of 3.2 cm and 2.0 cm, respectively) with an EF of 55% to 60% (Fig 3). The Bi-VAD was explanted on postoperative day 24, and magnetic resonance imaging at 10 days revealed an EF of 74%, LV end-diastolic volume of 85.9 mL, LV end-systolic volume of 22.3 mL, and a cardiac output of 8.08 L/min.

Apart from mild right lower limb weakness, neurologic recovery was complete. Repeat CT imaging suggested a small localized infarction in the posterolateral parietal lobe. The patient was transferred on postoperative day 34 to the neurosurgical institute, where further imaging revealed a posterior communicating artery aneurysm, not visible on initial CT angiography, which was successfully embolized. At her 4-month outpatient review she reported no cardiac or neurologic limitations (Glasgow Outcome Score 5), and TTE findings were completely normal.

Comment

SAH is associated with high morbidity and mortality, particularly if associated with myocardial dysfunction and out-of-hospital cardiac arrest [2, 4]. Myocardial dysfunction precludes aggressive triple-H therapy for cerebral vasospasm and impairs cerebral blood flow and subsequent neurologic recovery. Apostolides and colleagues [3] were the first to use IABP counterpulsation in this setting and documented improvements in cardiac performance and tolerance to triple-H therapy. The use of VADs in this setting has been contraindicated, owing to the requirement for further surgical procedures and anticoagulation.

The risk of aneurysmal rebleeding is maximal (at least 3% to 4%) on the first day after SAH, reducing to a constant rate of 1% to 2% per day over the subsequent 4 weeks [5]. Rebleeding is associated with a case fatality rate of around 70% [5]. Factors that particularly increase

the rebleeding risk in this case included the patient's poor neurologic status on admission and the longer time to treatment of the aneurysm. Anticoagulant therapy was therefore withheld for the initial 96 hours as a precautionary measure to reduce the risk of hematoma progression in the event of rebleeding.

On balance, initiating early anticoagulant cover that leads to hematoma expansion is likely more problematic than the relatively benign procedure of exchanging a pump with thrombus. Moreover, when managing a long-term LVAD supported child with new-onset progressive subdural hemorrhage, Haque and colleagues [6] described a successful strategy of reversing anticoagulation to facilitate hematoma evacuation.

Although RV contraction was only moderately impaired and hence LVAD support may have sufficed hemodynamically, the rationale for RVAD support was primarily to reduce intracranial pressure and improve the cerebral perfusion pressure by alleviating cerebral venous congestion.

By securing optimal systemic flows and stable, low-normal, mean arterial pressures, the Bi-VAD expedited reversal of multiorgan dysfunction and potentially reduced the risk of rebleeding from untreated aneurysmal disease, respectively. The Bi-VAD also reduced the risk of LV thrombosis by optimizing left heart filling and subsequent LV ejection.

In conclusion, this report demonstrates a successful salvage strategy using a heparin-free Bi-VAD implantation technique in SAH-induced cardiogenic shock. By stabilizing hemodynamics, reducing cerebral venous congestion, and avoiding early anticoagulation, this strategy facilitated myocardial recovery, reversal of multiorgan dysfunction, and neurologic recovery. This strategy could potentially be implemented in patients with cardiogenic shock and other concurrent hemorrhagic pathologic conditions.

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Successful Bicuspid Aortic Valve Repair Using External Aortic Annuloplasty

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A 33-year-old man presented with severe aortic insufficiency due to a prolapsed bicuspid aortic valve. The ventriculoaortic junction was dilated to 29 mm without root dilatation, and external ring annuloplasty was performed using a Gelweave (Terumo, Tokyo, Japan) graft to reduce the size to 22 mm. The leaflets were repaired by dividing and suturing a raphe between the right and left cusps. This combination provided adequate coaptation depth (8 mm) and showed excellent results, with trivial aortic insufficiency. This approach is suitable for repair of a bicuspid aortic valve with a dilated ventriculoaortic junction without root dilatation.

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Bicuspid aortic valve (BAV) is one of the most common congenital cardiac anomalies, and approximately 20% of patients are young or middle-aged adults with aortic insufficiency (AI) [1]. Aortic valve repair in such patients has become more common during the last decade because it avoids long-term prosthesis-related complications [1-4]. AI in BAV patients is commonly caused by

dilated, elliptically shaped, ventriculoaortic junctions (El Khoury type I) or by cusp diseases (El Khoury type II) [2]. Cusp repair alone may not be adequate, and aortic root replacement or annuloplasty should be considered [2, 3].

External aortic annuloplasty was initially reported in valve-sparing aortic root remodeling [5] and has been widely accepted [6]. We applied this concept to our BAV patient with AI because of the isolated dilatation of the ventriculoaortic junction and cusp prolapse (El Khoury types Ic and II). External ring annuloplasty combined with cusp repair provided excellent results.

The patient was a 33-year-old man diagnosed with severe AI due to BAV. Transthoracic echocardiography showed left ventricular (LV) dilatation with an end-diastolic diameter of 64 mm, mildly depressed LV function with an ejection fraction of 0.48, normal aortic root with a sinotubular junction of 29 mm, and a ventriculoaortic junction of 29 mm. His aortic valve was bicuspid, with a median raphe on the conjoint cusp showing severe eccentric AI. The conjoint cusp was made of the right and left cusps (type 1, L/R, I) by the classification system of Sievers and Schmidtke [7]. Although he remained asymptomatic, he was considered a surgical candidate for elective aortic valve repair because of severe AI with LV dilatation and dysfunction.

After induction of anesthesia, intraoperative transesophageal echocardiography (TEE) confirmed the same BAV morphology as shown by transthoracic echocardiography, with a ventriculoaortic junction of 29 mm, an aortic valve area of 5.8 cm², coaptation depth of less than 2 mm with a gap, and severe eccentric AI extending out of the gap (Fig 1A). An echocardiographic variable suggested by Pettersson and colleagues [1], termed the tissue normality index (TNI): $[TNI = (\text{diastolic cusp area} - \text{systolic cusp area}) / \text{diastolic cusp area}]$, was calculated to estimate cusp pliability. The TNI was 0.63 for his non-coronary cusp.

A standard median sternotomy was performed, and aortic and bicaval cannulations were used to perform cardiopulmonary bypass. The ascending aorta was clamped and transected 1 cm above the sinotubular junction. Selective antegrade cardioplegia was administered, and complete diastolic arrest was obtained. The aortic valve showed 2 cusps with different morphologies. The non-coronary cusp was looked normal (reference cusp), with a geometric height of 20 mm. The conjoint cusp was made of the right and left cusp fusion with a raphe, and the geometric heights of the cusps were 18 mm and 20 mm, respectively. The free margin of the conjoint cusp was elongated, which caused prolapse. The ventriculoaortic junction was dilated to 29 mm.

We decided to perform external aortic annuloplasty combined with cusp repair. Both coronary arteries were freed and taped (Fig 2A). Six 2-0 Ti-Cron (Covidien, Mansfield, MA) pledgetted mattress sutures were placed circumferentially from inside out in the subvalvular plane (Fig 2B) below the nadirs of each cusp and at the base of each interleaflet triangle. Special care was taken not to place the stitch into the membranous septum between the

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