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 are of 1.7 m^2 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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FEATURE ARTICLES

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 bump (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 contra-indicated 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 μg/kg/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 μmol/L), and liver injury (aspartate aminotransferase 668 IU/L; alanine aminotransferase 695 IU/L), severe metabolic acidosis ($[H^+]$ 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 μg/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 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.

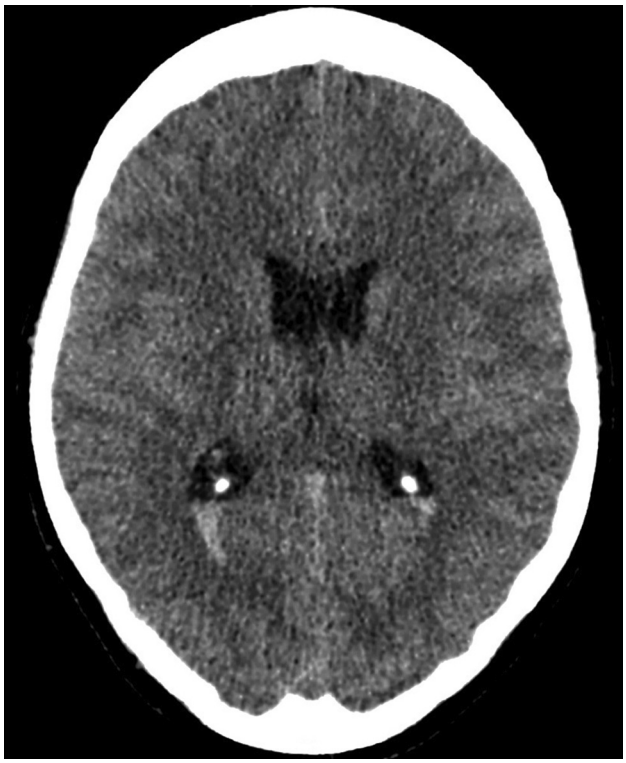

Fig 1. Non–contrast cranial computed tomographic scan showing acute subarachnoid hemorrhage.

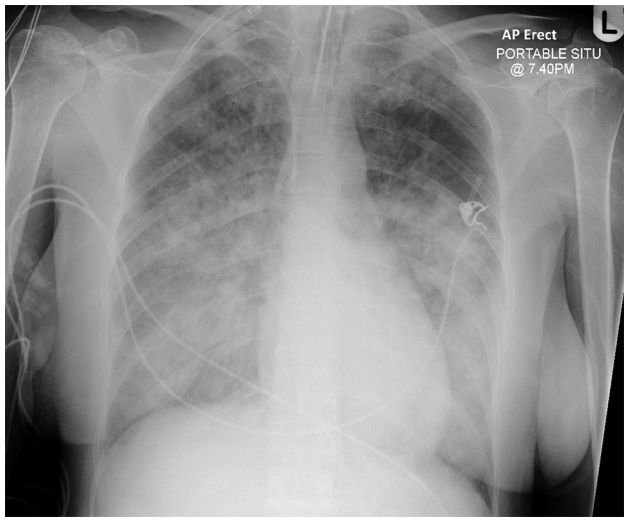

Fig 2. Chest roentgenogram on admission showing gross pulmonary edema.

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