improved durability and performance relative to previous VADs. Our major rationale for choosing the HeartWare HVAD was its relatively small size and ability to be contained within the pericardium, thus eliminating the potential need to create a pocket or rotate the pump configuration. In addition, the device's integrated inflow cannula could decrease the chance of inflow obstruction related to the abnormal position of the ventricles in CC-TGA. TEE guidance ensured successful selection of the optimal insertion site for the VAD inflow cannula.

In summary, we report a case of successful HeartWare HVAD implantation in a patient with CC-TGA to support the function of the RV (systemic ventricle).

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The role of the total artificial heart in the treatment of post–myocardial infarction ventricular septal defect

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Post–myocardial infarction ventricular septal defect (VSD) can be a fatal complication. Mechanical support to stabilize the patient until repair or transplant may be necessary, because emergency operative repair carries a mortality as high as 60%.[1-3] The CardioWest Total Artificial Heart (TAH-t; SynCardia Systems, Inc, Tucson, Ariz) has been used successfully to replace the failing heart as a bridge to transplant.[4] We report the cases of the first 2 patients treated with TAH-t implantation for unstable cardiogenic shock resulting from post-MI VSD rupture.

CLINICAL SUMMARIES

Patient 1

A 68-year-old man was transferred to our institution in extremis after an anterior MI and left anterior descending stent 2 placement weeks previously. Evaluation by echocardiography showed a large pericardial effusion, a 22-mm apical VSD, and biventricular failure. The patient had deterioration to cardiogenic shock (systolic blood pressures less than 75 mm Hg, pulmonary arterial pressure 50/20 mm Hg, central venous pressure of 16-21 mm Hg, and pulmonary wedge pressure of 31 mm Hg) and was intubated and transferred to our institution on maximal pressor support. At arrival, emergency venoarterial extracorporeal membrane oxygenation (ECMO) was instituted through the femoral vessels, and the patient's condition was allowed to stabilize for 24 hours. The patient underwent surgery for possible repair and device support; however, because of extensive myocardial damage and poor function, the decision was made intraoperatively for TAH-t placement. The patient was extubated within 24 hours and supported for 76 days until heart transplant. He had an uncomplicated hospital course and was discharged 2 weeks after transplant. At 3 years of follow-up, the patient remains in excellent condition.

Patient 2

A 65-year-old woman with a medical history significant for diabetes mellitus type 2, hypertension, hyperlipidemia, and scleroderma was admitted to outside hospital for an acute inferior MI. She was taken for emergency cardiac catheterization and had 2 stents placed in the proximal and mid right dominant coronary artery. On the next day, she became dyspneic and hypotensive. Physical evaluation noted a new heart murmur, and echocardiography revealed a large inferior base VSD of 14 mm with biventricular failure. The patient was intubated, an intra-aortic balloon pump was placed, and pressor support was maximized. The patient's condition continued to deteriorate, and she was transferred to our

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institution and stabilized with femoral venoarterial extracorporeal membrane oxygenation. The patient was deemed a transplant candidate and taken to the operating room 4 days later for mechanical circulatory support. Intraoperative evaluation noted extensive bilateral myocardial necrosis and friability, including the left apex, septum, and a large portion of the right ventricle. The decision was made for TAH-t placement. On postoperative days 0, 1, and 3, the patient required reexploration and evacuation of hematoma for bleeding and mediastinal hematoma. She continued to progress to multiorgan failure, coagulopathy, and vasodilatory shock despite multipressor support. Evidence of sepsis, including bilateral pneumonia and lactic acidosis, led to discontinuance of support 9 days after TAH-t implantation.

DISCUSSION

VSDs are rare, life-threatening complications that occur after MI in 1% to 2% of patients.[1] As many as 90% of patients would die without intervention; however, mortality with emergency surgical or percutaneous repair is still greater than 50%.[1] Patients who have cardiogenic shock and biventricular failure develop require mechanical support for stabilization until definitive repair or transplant can be performed. Implantable left ventricular assist devices have been successfully used as a bridge to transplant after post-MI VSD.[3] Securing the inflow cannula to infarcted apical myocardium can be technically difficult, and post-MI ventricular arrhythmias may complicate left ventricular assist device management. Severe valvular insufficiency and post-MI extensive left ventricular myocardial loss with poor function may favor replacement of both ventricles.

The TAH-t is a pneumatic, pulsatile pump that replaces the patient's ventricles and native valves. Implantation of the TAH-t has been shown to improve outcomes in unstable patients in end-stage cardiogenic shock by providing immediate hemodynamic stabilization and end-organ perfusion until cardiac transplant can occur. Survivals to transplant with the TAH-t as great as 79% have been reported,[4] comparable to biventricular support survival of 56% at 6 months.[5] Use of the TAH-t currently obligates the patient to undergo a transplant, and every effort should be made to preserve the patient's own myocardium. All mechanical support devices possess inherent risk factors, including infection, thromboembolism, and hemorrhage.

We present the first 2 cases of patients with post-MI VSD rupture with end-stage cardiogenic shock to undergo TAH-t placement. TAH-t support is an alternative option to biventricular support devices for critically unstable patients with post-MI VSD and extensive ventricular myocardial loss. Mortality was 50% in this small series, which underlines the complexity of predicting outcomes in this patient population.

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Left ventricular vegetations: A rare manifestation of Libman-Sacks endocarditis

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Libman-Sacks endocarditis, first described by Libman and Sacks[1] in 1924, is the pathognomonic valvular lesion of systemic lupus erythematosus or antiphospholipid antibody syndrome that may develop on the endocardial surface of the heart. These lesions have a propensity toward formation on the left valves, particularly the ventricular surface of the mitral valve. We report the case of a 26-year-old woman without a history of cardiac disease who was seen for an atypical form of Libman-Sacks endocarditis. The disease progressed in an