End-Stage Cardiomyopathy

Ventricular Assist Devices

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1. INTRODUCTION
Heart disease is the number one cause of morbidity and mortality in Western society. This problem has become well recognized in the literature over the past few decades, and researchers have mounted intense efforts to remedy the situation. A heart transplant is clearly the most effective therapy for patients with advanced heart failure; it is able to restore them to a near-normal lifestyle, and long-term survival is excellent (1). However, the number of donor hearts available for transplantation is limited to only about 2000 each year, compared to over 10,000 people each year who could benefit from such a therapy.

Successful development of a total artificial heart or advanced ventricular assist devices could help resolve this imbalance. Development efforts to do so were organized under the National Institutes of Health leadership in 1964. The ideal pump was originally thought to be a totally implantable device, with an electrical motor that could completely replace the heart function physiologically as well as anatomically. Yet, despite many decades of research, we still do not have such a device. In the mid-1970s, parallel research on ventricular assist devices took place; the result was a variety of ventricular assist devices described in this chapter.

2. MECHANICS

2.1. Volume Displacement Pumps
The human heart is a volume displacement pump. To create a unidirectional flow with a single pumping chamber, inflow and outflow valves are required. For example, in the left ventricle of the native heart, the mitral valve functions as the inflow valve; the aortic valve functions as the outflow valve. The result is unidirectional blood flow. Similarly, ventricular assist devices designed as volume displacement pumps would require these two types of valves. Some ventricular assist devices have incorporated mechanical prosthetic valves; others have utilized bioprosthetic valves (Fig. 1). Importantly, the choice of valve mandates different types of anticoagulation therapy and thus leads to a different natural history of valve failure.

A major obstacle in designing clinically acceptable ventricular assist devices has been the array of problems associated with the blood contact surfaces. Specifically, any type of stagnant blood flow within the pocket of the pumping chamber can result in thrombus formation. Also, the artificial blood contact surface is quick to promote a clotting cascade, resulting in significant thromboembolism. Yet, initial attempts at making the blood contact surface as smooth as possible have not yielded satisfactory outcomes. One proposed solution for this was the construction of textured blood contact surface to promote early platelet and fibrin deposition, which resulted in formation of stable pseudointima; such a design was applied in the HeartMate® system (Thoratec Corporation; Pleasanton, CA) (Fig. 2) (2).

The mechanism involved in ejecting blood varies from model to model. Some ventricular assist devices either have a compressible blood-filled sac or a flexible diaphragm within a hard shell. In others, compressed air serves as a medium to collapse either the sac or the diaphragm and thereby eject...
blood. Compressed air seems to provide a simple and reliable way of either activating the diaphragm or compressing the sac, with a pressure more comparable to a physiologically acceptable waveform. However, this design requires a bulky driving console, compromising the patient's mobility.

In other ventricular assist devices, the diaphragm is activated by a slow-torque electrical motor that is small enough to be incorporated into the implantable unit. Thus, patient mobility is significantly improved with this type of ventricular assist device compared to the ones driven by a pneumatic console. Because the diaphragm is propelled by a noncompressible metal, the pressure waveform during ejection is less physiological, with a rather rapid rise in pressure over time (dp/dt); the peak dp/dt could be many times higher than that generated by a normal heart. This could translate into a significant mechanical load on the inflow valve during the ejection phase and subsequent premature inflow valve failure. Furthermore, such an abnormal pulse wave and sudden increase in patient’s cardiac output could adversely affect the central nervous system by causing cerebral edema.