Radiologic Appearance of the Jarvik Artificial Heart Implant and Its Thoracic Complications

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The principles of total artificial heart devices, their radiologic appearance, and the complications associated with their use have become increasingly relevant as these devices are used more often in patients with end-stage cardiac disease. The Jarvik artificial heart implant is the one used most frequently. Intrathoracic complications related to its use were evaluated radiographically in seven patients undergoing implantation at our institution. Because of the relatively large size of the device, complications from compression of the left lung are common. Four of the seven patients had prolonged atelectasis of the left lower lobe and two had major vascular compression (pulmonary veins in the left lung in one and the inferior vena cava in another). Other chest complications were infection in the surgical site (three patients), mediastinal bleeding (one patient), and extensive fibrous adhesions around the device, making explantation difficult (one patient). All seven patients had pulmonary edema before surgery, which regressed over several days after surgery.

Radiographs readily display the major components of the Jarvik heart and are valuable for detecting pulmonary complications associated with its use.

Currently, cardiac transplantation is the only feasible long-term treatment for patients with end-stage heart disease. Unlike patients with end-stage renal disease who can be maintained on chronic dialysis, these cardiac patients have no alternative once drug therapy has failed.

However, cardiac transplantation has major limitations. The scarcity of donor hearts requires it to be limited to otherwise healthy individuals, preferably those under 50 years old. The possibility of rejection and the need for long-term immunosuppression are additional limiting factors. For these reasons, great efforts have been made to develop permanently implantable artificial hearts, particularly for older patients with irreparable heart damage [1, 2]. Of several models available, the Jarvik artificial heart has been used most often. Of 84 implanted devices, 71 have been Jarvik models [3]. To study the radiographic appearance of the device and its thoracic complications, we reviewed the chest radiographs of seven patients who received a Jarvik heart at the University of Arizona.

Materials and Methods

A Jarvik total artificial heart device (Symbion Inc., Salt Lake City, UT) consists of two independent ventricles, each with a pneumatically driven diaphragm. After the ventricles are filled with blood from the native right and left atria, the diaphragms are driven upward like pistons by extracorporeal pneumatic pumps, ejecting blood into the systemic and pulmonary circulations (Fig. 1). Figure 2 is a radiograph of the Jarvik heart showing its structural components. The rigid ventricles are constructed of Biomer, a segmented polyurethane. The diaphragms that separate the blood chamber from the air chamber consist of alternating layers of Biomer and graphite lubricant for maximum flexibility and durability. The native atria are joined to the artificial ventricles by Dacron felt cuffs. The ventricles are joined to the native great vessels by Dacron vascular grafts. Four pyrolytic, carbon-disk, artificial valves maintain

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Fig. 1.—Schematic drawing of a single ventricle from the Jarvik artificial heart shows function during diastole and systole.



Fig. 2.—In vitro left anterior oblique radiograph of Jarvik-7/100 total artificial heart device shows the four artificial valves, the two ventricular diaphragms (curved arrows), and the polyurethane tubings (straight arrows) that connect the device to an extracorporeal pneumatic pump. The two ventricular units are structurally identical and connected to each other with a Velcro patch. Anterior tilt to right ventricle allows optimal anatomic relationship between outflow valves and native great vessels.

unidirectional blood flow. The ventricles are connected at their apices to the extracorporeal pneumatic pumps by reinforced polyurethane tubes that enter through the anterior chest wall. They are inflated and deflated by controlled pulses of air such that the stroke volumes of the ventricles can be varied to meet the circulatory needs without changing the heart rate, thus responding to changes in cardiac preload as a normally functioning human heart responds.

The ventricular pumping chambers of the Jarvik heart are positioned one behind the other and have a distinctive radiographic appearance. During diastole, the blood chambers are filled and only a crescent of air is visible in the air chambers (Fig. 3). On a radiograph obtained during systole, the air chambers are completely distended (Fig. 4). At our institutions, pneumatic heart devices have been used primarily to support the circulation of potential transplant candidates until a suitable donor heart can be procured. Since August 1985, seven acutely ill patients, two women and five men 25–40 years old (mean age, 36 years), received eight artificial hearts (one patient had implants on two separate occasions). Four patients had acute or endstage viral cardiomyopathy, two had idiopathic cardiomyopathy, and one had Ebstein anomaly. The immediate indications for implantation included donor heart failure (one patient), acute rejection of donor heart (two patients), acute cardiogenic shock (two patients), and progressive deterioration without a donor available (two patients). The Jarvik-7/100 model (with a maximum stroke volume of 100 ml) was used in two cases, and the Jarvik-7/70 model (with a 70-ml maximum stroke volume) was used in five. The duration of implantation ranged from 12 hr to 243 days.

The radiographs of these patients were reviewed and the findings are the basis of this report.

Results

Patients who have received artificial heart devices are subject to a number of thoracic complications, some related directly to the device and others to the surgical procedure. In our patients, the complications included left lower lobe atelectasis (four patients), a major vascular compression (two patients), mechanical malfunction of the device (one patient), infections (three patients), mediastinal bleeding with pericardial tamponade (one patient), and development of fibrous adhesions around the device (one patient). All patients had pulmonary edema before surgery; failure of the edema to resolve after implantation could be considered a complication.

Atelectasis

As with heart and heart-lung transplantation patients, recipients of artificial heart devices exhibit left lower lobe atelectasis after surgery. This is often difficult to appreciate on anteroposterior chest radiographs alone, because of the overlying density of the device (Fig. 5), necessitating lateral or oblique films for evaluation. All seven of our patients exhibited some degree of left lower lobe atelectasis after surgery. In four patients, the atelectatic changes did not resolve despite aggressive respiratory therapy. Persistent atelectasis in these patients was believed to be caused by mechanical impact of the device against the left lower lobe due to imperfect fit of the device within the chest. If the device is well positioned and appropriately sized, atelectasis due to compression from mechanical action of the artificial heart should be minimal.

Pulmonary Edema

Preimplant pulmonary edema in artificial heart recipients is usually severe and after implantation may persist until extravascular fluid is mobilized. Even in uncomplicated postoperative courses, pulmonary edema persisted for 3–6 days after implantation. Pulmonary edema was clinically severe in three patients, with mechanical ventilation being required for 7–31 days after surgery. Radiographically, diffuse alveolar infiltrates were present. In the other four patients, postoperative pul-



Fig. 3.—A and B, In vivo posteroanterior radiographs of Jarvik heart from two different patients during systole (A) and diastole (B). Note exchange of air and blood in ventricles (arrowheads).



Fig. 4.—A and B, Lateral radiographs of Jarvik heart during systole (A) and diastole (B). A = aortic valve; M = mitral valve; P = pulmonic valve; T = tricuspid valve; RV = right ventricle; LV = left ventricle.

monary edema was clinically less severe and radiographically manifested as hilar and basilar infiltrates. These patients were weaned from the ventilator in 1-3 days.

In order to enhance mobilization of pulmonary fluid, a diastolic vacuum of up to 7 mm Hg is produced by the Jarvik

extracorporeal pump system to lower mean right and left atrial pressures. The use of diastolic suction with Jarvik heart devices has the advantage of increasing ventricular filling without volume loading or raising atrial pressures, thereby assisting with diuresis of retained fluid.



Fig. 5.—A and B, Radiographs show compression atelectasis of left lower lobe on lateral view (arrows in B), which is obscured on posteroanterior view (A) by artificial heart device. In this patient, device is larger than normal ventricles.

Major Vascular Compression

Persistent pulmonary edema more severe in the left lung despite the use of ventricular diastolic vacuum indicated obstruction of the left pulmonary veins in one patient. The cause of obstruction was the disproportionate size of the implant relative to the size of the patient's thoracic cage (Fig. 6). Placement of struts between the sternal edges to enlarge the thoracic cavity in order to alleviate compression of the pulmonary veins was not successful and the patient died.

In a second patient, progressive lower extremity edema, hepatosplenomegaly, and ascites prompted transfemoral venous catheterization, which revealed a 10-mm gradient across the diaphragmatic portion of the inferior vena cava. Relief of compression was accomplished by repositioning the device farther to the left.

Malfunction of the Mechanical Heart

In one of the patients, a left atrial catheter resulted in left inflow ("mitral") valve regurgitation that necessitated removal of the catheter. This was evident on the chest radiograph as worsening pulmonary edema.

Infection

Patients with artificial hearts have an increased risk of developing septic complications from the presence of a large foreign body and the percutaneous drive tubings. Infection along the tracts of the pneumatic tubes can lead to mediastinitis, pneumonitis, and septicemia. Risk for infection is greater the longer the device is in place. In our patient with



Fig. 6.—Severe left-sided pulmonary edema from compression of left pulmonary veins due to discrepancy in size of Jarvik device and thoracic cage. Metallic struts (*arrows*), inserted to increase chest size, were unsuccessful in alleviating venous compression. The patient died 8 hr later.

the longest-duration implant (248 days), a total of seven infections were documented, some of which had apparently entered the chest around the drive lines entering the chest wall. Causative organisms included *Staphylococcus* and *Candida paropsilosis* periprosthetic and drive-line infections; en-



Fig. 7.—Chest CT scans at mediastinal (A) and lung (B) windows show thick fibrous adhesions (arrows) surrounding Jarvik heart, compression atelectasis of left lower lobe, pleural effusion, and chronic loculated pneumothorax.



terobacterial, influenza A, *Legionella*, and *Candida albicans* pneumonias; and disseminated herpes simplex. At the time of a subsequent heart transplant, infection around the device was well evaluated with CT (Fig. 7).

Mediastinal Bleeding

Because patients are anticoagulated to prevent thrombotic and embolic events, sequential chest radiographs are important to detect bleeding complications. Progressive postoperative mediastinal widening was seen in one patient who developed atrial tamponade from hemorrhage into the pericardium around the device, necessitating surgical drainage.

Fibrous Adhesions

Development of fibrous adhesions between the device and pleural surfaces can cause air leaks, leading to pneumothorax or pneumopericardium (Fig. 7). Mechanical motion of the artificial ventricles and adherent fibrous tissue may cause shearing of small distal airways, resulting in air leaks. An airfilled ventricular chamber must not be mistaken for a loculated pneumothorax. In the patient with the longest-term implant (243 days), removal of the artificial heart before transplantation of a donor heart was difficult because of formation of dense fibrous adhesions around the device and between the device and the left lung.

Discussion

In the last 20 years, more than \$200 million has been invested in artificial hearts, of which the Jarvik heart is now the most widely used. Its development has been well chronicled by previous investigators [1–3]. With few exceptions, the primary use of artificial hearts is as a temporary measure.

Currently designed artificial heart devices do not conform well to the shape or size of the pericardial space. Their position is influenced by the shape and dimensions of the thorax. Ideally, the device is positioned in the pericardium so that the left ventricle is inferior, lateral, and posterior to the right ventricle. This helps prevent obstruction of the pulmonary veins and venae cavae. The distance between the sternum and the vertebral bodies is the major limiting factor in obtaining fit without venous compression. Careful preoperative evaluation of the recipient's thoracic volume is necessary. Currently, the bulkier 100-ml Jarvik-7 device is used only in patients weighing more than 75 kg and in those with a sternalvertebral distance of at least 13 cm [4, 5].

Artificial hearts are now being used successfully as a temporary measure until a donor heart is available for transplantation. Permanent implantable artificial hearts may someday be available for older patients with irreparable heart damage who may not qualify for transplantation and for patients eligible for transplantation who may die before a suitable donor heart is found. In such cases, the mechanical heart will serve as a "bridge to life." The Jarvik heart serves that purpose today when successful transplantation can follow.

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