Cardiorespiratory Interactions in Patients with an Artificial Heart

James L. Robotham, M.D.,* J. Brent Mays, P.A.-C., B.H.S., Melissa A. Williams, P.A.-C., B.H.S., William C. DeVries, M.D.

A retrospective analysis of the influence of respiration was carried out in three patients with artificial hearts. During spontaneous ventilation, large swings in intrathoracic pressure can produce a pattern reminiscent of pulsus paradoxus in the systemic arterial pressure. A decrease in intrathoracic pressure decreased biventricular filling and enhanced biventricular emptying. An increase in intrathoracic pressure increased biventricular filling, but acting as an increased afterload, impeded biventricular emptying. The influence of respiration on the artificial heart can be considered the result of the artificial ventricles' functioning effectively as extrathoracic pumps, such that changes in intrathoracic pressure produce gradients for biventricular filling and ejection relative to atmospheric pressure (which serves as the reference pressure for the artificial ventricles). Respiratory-induced variation in ventricular performance is clearly present with the artificial heart, but the mechanisms producing these changes appear to be markedly different from normal conditions, in which the ventricles are functionally within the thorax and have a compliant common septum allowing ventricular interaction. (Key words: Heart, artificial cardiorespiratory interactions. Lung: ventilation. Pulsus paradoxus.)

Evaluation of the cardiorespiratory interactions in patients with artificial hearts provides a unique opportunity to evaluate the effects of respiration on the circulatory system. Comparison of the phasic hemodynamic events during spontaneous ventilation in these patients to normal events in a native heart offers the potential for gaining clinically important insights into the effects of ventilation on the normal heart and should be helpful in managing patients with an artificial heart. With an intact circulatory system during spontaneous ventilation, the decrease in intrathoracic pressure increases systemic venous return, increasing right ventricular (RV) preload. Despite a small increase in RV afterload as lung volume increases, the increase in RV preload dominates and is responsible for the inspiratory increase in RV stroke volume. This increase in right-sided volume then competes for total intrapericardial volume with the left heart, reducing left ventricular (LV) diastolic compliance, a phenomenon known as ventricular interdependence.1-4

In the normal heart, this ventricular-interdependence-produced decrease in LV preload summates with the inspiratory increase in LV afterload produced by the decrease in intrathoracic pressure to reduce LV output during spontaneous inspiration.1,6-7 Brief comments have been made on the effects of ventilation on the artificial heart, but only one paper has specifically described an inspiratory decrease in ventricular filling during spontaneous ventilation with hypovolemia.9 The decrease in output was ascribed to compression of the recipient's intact right atrium by the expanding lungs. Compared to normal physiology, cardiorespiratory interactions occur with artificial ventricles, which have completely rigid external surfaces precluding ventricular interdependence and an inotropic state independent of neural input. Whereas both the normal ventricles and their respective arterial conduits are surrounded by intrathoracic pressure, the artificial ventricles are effectively exposed to atmospheric pressure during diastole. The peak systolic pressure generated by the artificial ventricles is regulated by the operator relative to atmospheric pressure, whereas the pressures in the pulmonary artery and aorta can be influenced by changes in intrathoracic pressure. A detailed consideration of the hemodynamic events with the artificial heart during spontaneous ventilation is unquestionably difficult because of the highly unusual traces that describe the performance of the artificial heart. However, when the significance of these unique pressure and flow measurements is appreciated, the principles elucidated may be applied to events during positive-pressure mechanical ventilation, and perhaps more importantly, may yield a broader appreciation of cardiorespiratory interactions with a native heart.

REVIEW OF THE JARVIK 7 HEART

The Jarvik 7 artificial heart (Symbion Inc., Salt Lake City, UT) is more accurately two artificial ventricles, since the recipient's atria remain. The pressure signals from the drive pumps and the measurements of air volume displaced as blood enters the ventricle can be used to in-
interpret the effects of ventilation on RV and LV filling and ejection.

Details of the mechanical characteristics of the Jarvik 7 artificial heart have been published. Two identical rigid chambers are implanted in the cardiac fossa, with artificial valves sewn to the respective atria, pulmonary artery and aorta. Each ventricle is divided by an elastic diaphragm, with blood on one side and air on the other. The rigid walls of the ventricular chamber determine the maximum amount of blood that can enter from the atrium into the ventricle during diastole, when the air side of the diaphragm is vented to atmospheric pressure. Pressurization of the air side of the diaphragm occurs during systole; the elastic limits of the diaphragm determine the maximum volume of blood that can be ejected from the ventricle. A pressure transducer on the air side of the diaphragm measures the drive pump pressures relative to atmosphere. Visual inspection of this signal allows determination of whether maximum filling or ejection has occurred (fig. 1A). The upper limit of the drive pump systolic air pressure is set relative to atmosphere by the operator and may be reached either as the pump continues to eject blood or when the diaphragm reaches its elastic limit, with complete ejection of the diastolic preload. When the diaphragm has been maximally displaced during the complete emptying of the ventricle in systole, further movement of the diaphragm abruptly ceases, and a characteristic sudden positive pressure transient followed by a plateau pressure equal to the set maximum pressure occurs in the drive pump pressure.

With complete (maximum) diastolic filling of the ventricle, the rigid chamber abruptly halts further diaphragm movement. The loss of momentum associated with the acute cessation of air egress from the chamber, produces a negative transient in the drive pump pressure during diastole (fig. 1A).

A pneumotachograph connected to the gas exit during diastolic filling is calibrated to measure the air volume leaving the chamber, which is equal to the blood volume entering the chamber. This signal is gated and integrated in the diastolic period only. The errors produced by gas compressibility are both consistent and small. The pneumotachograph is calibrated at the time of implantation and then after every three months. The tracing obtained during diastole is essentially equivalent to tricuspid or mitral flow (fig. 1B). When complete filling has occurred, a negative flow transient is observed as the valve closes, and is followed by absent air flow until systolic compression begins. Thus, both the drive pump pressure and the pneumotachograph traces can be used to define

![Diagrams](image-url)
the point of time at which complete filling of an artificial ventricle has occurred. The pneumotachograph traces provide an estimate of the variation in beat-to-beat ventricular filling.

**Methods**

Qualitative determination of changes in ventricular filling and emptying were made; due to the retrospective nature of the study, no quantitative estimates were made. If complete filling occurs during diastole and complete ejection occurs during systole, the stroke volume is quantitatively equal to the maximal pump displacement. Qualitative determination of acute changes in the ventricle's end diastolic and end systolic volumes, and hence stroke volume during a respiratory cycle, is limited to conditions in which either complete filling or complete ejection occurs. Under conditions of complete ventricular filling for each cardiac cycle of a respiratory cycle, the influence of ventilation on ventricular ejection can be evaluated; i.e., if complete diastolic filling indicated by negative diastolic transients in both the drive pressure and the pneumotachograph traces occurs with every beat, then the presence of a systolic plateau indicating complete emptying in one beat, followed by a lack of a systolic plateau in the next beat, means that for a constant preload the stroke volume of the second beat decreased. With complete ventricular emptying during each systole, the influence of ventilation on ventricular filling can be evaluated; i.e., if complete systolic emptying indicated by a systolic plateau occurs with every beat, then negative diastolic transients in the drive pressure and the pneumotachograph traces in one beat, not followed by negative transients in the next beat, means that for a constant end systolic volume, incomplete filling has occurred and hence diastolic inflow has decreased during the second beat.

Records from the postoperative period when right and left atrial, pulmonary artery, and radial artery pressures were measured during spontaneous and mechanical ventilation were reviewed from three patients. The transducers for these catheters were calibrated electronically every 8 h or after a change in the patient's position. Technical details of these procedures have been published.12

The effects of changes in intrathoracic pressure and lung volume over a full respiratory cycle, in addition to the effects of large transient decreases (hiccoughing) and increases (coughing) in intrathoracic pressure, were evaluated. Since neither an esophageal balloon nor a thoracostomy tube had been used to measure changes in intrathoracic pressure, simultaneous changes in atrial and pulmonary arterial pressures were used to define the qualitative direction and timing of changes in intrathoracic pressure. Concurrent dated and timed nursing notes allowed retrospective determination of the origin of the respiratory-induced changes in these vascular pressures, i.e., spontaneous or mechanical ventilation, hiccoughing, or coughing. In the two patients evaluated at the bedside, notation of respiratory events made directly on hardcopy records of the drive pump pressures obtained at that time were consistent with our retrospective data.

All implantations were performed according to clinical protocols approved by both the Food and Drug Administration and the Humana Hospital–Audubon Institutional Review Board.

**Results**

**Ventricular Filling**

Spontaneous inspirations were associated with diminished LV filling and stroke volume (fig. 2). Larger variations in LV filling and subsequent stroke volume were noted during spontaneous breathing with increasing degrees of airway obstruction caused by secretions. Suctioning of the patient's trachea markedly decreased these respiratory variations. Transient negative intrathoracic pressure produced by a hiccough markedly reduced LV inflow, occasionally with evidence of retrograde emptying of the LV or premature mitral valve closure (fig. 3). Large increases in biventricular diastolic filling were evident with a cough-induced rapid increase in intrathoracic pressure (fig. 4).

Respiratory variation usually was greater in LV filling than in RV filling. This reflects the strategy of obtaining complete filling without complete ejection on the right side and complete emptying without complete filling on the left side in order to minimize pulmonary arterial and venous pressures. Since complete RV ejection was avoided, the influence of respiration on RV filling was not demonstrable in each patient. When complete RV emptying did occur with each systole, respiratory variation in RV filling similar to that seen with the LV was observed.

**Ventricular Ejection**

A decrease in intrathoracic pressure increased RV output, whereas an increase in intrathoracic pressure decreased RV output (figs. 2–4).

The LV pump was set to accomplish complete ejection with each systole by setting the maximum systolic pressure sufficiently high such that small increases in intrathoracic pressure had little effect on LV ejection. Rapid decreases in intrathoracic pressure decreased the systemic arterial and pulmonary arterial pressures, and thereby reduced the respective ventricular afterloads and enhanced ejection; i.e., after complete filling, complete ejection occurs earlier during systole, or occurs although it had not been present before the fall in intrathoracic pressure. The op-
HEMODYNAMICS WITH AN ARTIFICIAL HEART

FIG. 2. Spontaneous ventilation with moderate airway obstruction. $P_{\text{LA}} = \text{left atrial pressure; } P_{\text{RA}} = \text{right atrial pressure; } P_{\text{PA}} = \text{pulmonary artery pressure; } P_{\text{AM}} = \text{arterial pressure; } LDP = \text{left ventricular drive pump pressure; } RDP = \text{right ventricular drive pump pressure; } LVDF = \text{left ventricular diastolic filling. The transition from inspiration (I) to expiration (E) is marked by the heavy dashed vertical line. A complete LV ejection occurs with every systole, indicated by a plateau of LDP at the end of each ejection period. Increased respiratory induced variation in the vascular pressures is evident with end inspiration occurring during beat 4 and the following diastole, while early expiration is noted during beats 1 and 2, 5, and 6. Systemic arterial pressure demonstrates a decrease in peak systolic pressure and a marked reduction in the area under the pressure curve in beat 5 (arrow). This results from minimum LV diastolic filling during the preceding diastolic period (first arrow in LVDF) and complete ejection of a small stroke volume from the LV during beat 5 as indicated by the extremely early ejection plateau in the LDP trace (first arrow). During the diastolic period, immediately after beat 5 as intrathoracic pressure rapidly increases, there is a transient negative pressure in the LV drive pressure (second arrow) indicating complete LV filling consistent with the LVDF pneumotachograph tracing showing elevated early and mid-diastolic flows with complete filling in late diastole (second arrow). Complete LV filling is also seen in the expiratory diastoles preceding beats 1, 2, and 7. The right-sided drive pressure demonstrates a diastolic filling transient during every beat but complete ejection only during beat 4 (arrow) when intrathoracic pressure is lowest. Left atrial pressure shows respiratory variation with the change in intrathoracic pressure and a prominent "y" descent at the beginning of each diastolic period (arrow), reflecting the low early diastolic LV pressure. Consistent with the therapeutic strategy of maintaining elevated peripheral vascular and RV diastolic pressures rather than elevated pulmonary vascular pressures, the "y" descent for the right atrial pressure ($P_{\text{RA}}$) is present but smaller. All pressures are in mmHg.

posite occurred with rapid increases in intrathoracic pressure (fig. 4).

The pattern of respiratory variation in systemic arterial pressure with an artificial heart was not the same as that with a native heart. With an artificial heart, the variation in the peak systolic pressure during quiet normal spontaneous respiration was absent. Instead, a relatively constant peak systolic pressure determined by the operator-set maximum pressure for ventricular ejection was present, with a variable degree of respiratory variation in the area under the curve reflecting the inspiratory fall in stroke volume. With large variations in intrathoracic pressure, both the peak systolic pressure and the area under the arterial pressure tracing were reduced, with inspiration producing a pattern similar to pulsum paradoxus normally seen with airways obstruction.

Discussion

A schematic representation of the forces that affect ventricular filling and emptying in an artificial heart will facilitate discussion of the results. Figure 5a illustrates two communicating compliant vascular beds in equilibrium with equal intravascular pressures initially surrounded by atmospheric pressure. If the surrounding pressure of one (A) is increased or the pressure of the other surrounding compartment (B) is decreased, the result will be a translocation of volume from the higher to lower pressure chamber (A to B). The same principles can be applied if one vascular bed is considered to be actively ejecting into the other. If A is ejecting into B, a negative pressure around the downstream vascular compartment B will increase the volume ejected. Conversely, if B is ejecting into A, an increase in pressure around the vascular compartment A will tend to diminish ejection. If three vascular beds are connected in series, each with potentially different surrounding pressures, the complexity increases but the basic mechanisms remain (fig. 5b). If the pressure in the middle compartment (e.g., the right atrium) decreases due to an isolated decrease in the surrounding intrathoracic pressure, blood flow into it from the systemic veins will increase, while blood flow out of the compartment (i.e., ventricular filling) will decrease. With a positive intrathoracic pressure, the opposite events will occur; i.e., venous blood flow into the middle chamber will decrease and flow out (i.e. ventricular filling) will increase. If the pressure around the first compartment (e.g., abdominal pressure around the systemic veins) increases as the pressure around the middle compartment decreases (fig. 5c), filling of both the middle and third compartments (e.g., right atrium and ventricle) will be enhanced. Contrast this situation to that in figure 5b, in which the pressure around the first compartment is constant; the dashed-line volume in the third compartment in figure 5c is larger than that in figure 5b.

Differences Between an Artificial and a Native Heart

Table 1 summarizes the observations of preload, afterload, and stroke volume in patients with an artificial heart during spontaneous ventilation, and compares them to the expected events with a native heart and normal circulatory system.

Preload

Atrial filling in both the artificial and native heart is largely determined by the pressure gradient between the upstream venous compartment and the respective atrial compartment. Thus, right atrial filling is influenced not only by respiratory variation in the surrounding intrathoracic pressure, but also by any increase in the abdominal pressure produced by diaphragmatic descent, which in the volume-replete patient raises the abdominal venous pressure and thereby increases systemic venous return.
In the native heart, these same factors affect RV filling. Since incomplete RV ejection usually was required with the artificial heart to minimize pulmonary vascular pressures, complete RV filling was observed throughout the respiratory cycle; i.e., respiratory variation was absent. When complete RV ejection did occur, the artificial RV exhibited a pattern of respiratory variation similar to that of the artificial LV listed in table 1.

Although increases in alveolar pressure surrounding the pulmonary capillary bed relative to intrathoracic pressure surrounding the native heart (i.e., increasing transpulmonary pressure during inspiration) tend to increase pulmonary venous return, a relative increase in alveolar pressure does not appear to be a dominant factor affecting LV filling in either patients with artificial hearts or native hearts since LV inflow decreases during spontaneous inspiration under both conditions. In the native heart, the decrease in LV filling is due to ventricular interdependence as the increased RV volume decreases LV compliance. With artificial ventricles, the influence of ventricular interdependence on LV filling is removed as a factor influencing ventricular compliance, since rigid walls separate the two chambers. Atrial interdependence may still exist, although after a pericardectomy, its influence on atrial compliance is small. If atrial interdependence were a dominant influence on ventricular filling, RV filling would have been diminished as LV filling increased.

Afterload

In the native heart under normal conditions, the increase in lung volume with inspiration increases pulmonary vascular resistance and hence RV afterload. Since the RV and pulmonary artery are both surrounded by intrathoracic pressure, a change in intrathoracic pressure alone cannot change the gradient between them to affect RV afterload. The inspiratory increase in RV afterload produced by a normal tidal increase in lung volume is small compared to the increase in RV preload, and hence the RV stroke volume increases. LV afterload increases in the native heart during spontaneous inspiration because of the decrease in intrathoracic pressure surrounding the LV, assuming that the pressure surrounding the extrathoracic compartments either is constant or increases. It is important to note that the increase in LV afterload arises not because of the pressure gradient between the LV and the intrathoracic aorta, but because of the gradient imposed as blood leaves the intrathoracic aorta to enter the extrathoracic arterial beds. The inspiratory increase in LV afterload in the native heart summates with the ventricular-interdependence-produced decrease in LV preload to reduce LV stroke volume during spontaneous respiration. In contrast, an inspiratory decrease in intrathoracic pressure with an artificial LV, reducing the afterload, tends to increase stroke volume, but a marked reduction in preload dominates to produce the net decrease in LV stroke volume with inspiration.

Stroke volume

Despite major differences in physiology between the native and the artificial heart, spontaneous inspiration increases RV stroke volume and decreases LV stroke volume in both types of hearts, but for different reasons. Normally, a spontaneous inspiration increases both the RV and LV afterload. With an artificial heart, both afterloads decrease during a spontaneous inspiration with the decrease in intrathoracic pressure. On the right side this produces the increased stroke volume with a constant RV end-diastolic volume, which occurs when there is complete filling during each diastole. If RV end-diastolic volume is allowed to vary with respiration, the artificial RV behaves like the artificial LV, decreasing its stroke volume during inspiration because of decreased filling, despite a reduced afterload. With a native heart, an increased RV preload is responsible for the inspiratory increase in RV stroke volume despite a small increase in afterload, because of the increase in lung volume.

With a native heart, LV stroke volume decreases during the inspiratory decrease in intrathoracic pressure because LV preload decreases and LV afterload increases. With a large decrease in intrathoracic pressure, the inspiratory decrease in LV stroke volume combined with retention.
of blood in the intrathoracic aorta produces the diminished peripheral systolic arterial pressure referred to as pulsus paradoxus. The decrease in LV stroke volume in the artificial heart must be caused by a decrease in preload, since the afterload to LV ejection is decreased by the inspiratory decrease in intrathoracic pressure. An inspiratory decrease in LV stroke volume combined with retention of blood in the intrathoracic aorta produces a diminished peripheral pulse volume (i.e., area under the curve) and if substantial, produces a decrease in the peak
systolic pressure measured in a peripheral artery, as seen in figure 2. Thus, even with the artificial LV pump peak systolic pressure in the air side of the ventricular chamber set at a constant value, if there is a marked reduction in the stroke volume ejected into the peripheral arterial system, the peripheral systolic arterial pressure will decrease. In this sense, a phenomenon similar to pulsox paradoxus also is exhibited during airway obstruction with an artificial heart. Consistent with our observations, Rouby et al. have recently published a tracing demonstrating an inspiratory decrease in systolic arterial pressure (pulsox paradoxus) during spontaneous ventilation in a patient with an artificial heart.16

Since the drive pump pressure was set to obtain complete ejection, respiratory variation in LV stroke volume resulted predominantly from alterations in LV diastolic filling. This explains the large y descent seen in the left atrial pressure wave in early diastole as the left atrium empties into a low-pressure empty ventricle.

**ANALYSIS OF THE ARTIFICIAL HEART AS AN EXTRATHORACIC VENTRICLE**

The influence of ventilation on RV and LV preload, afterload, and stroke volume in the artificial heart is markedly different in many respects to that in the native heart. The respiratory changes in the right and left mechanical ventricles reflect the effects of changes in intrathoracic pressure on the filling and ejection of functionally extrathoracic ventricles. Ventricular filling under all circumstances is determined by the pressure gradient between the atrium and ventricle. The artificial ventricle is functionally an extrathoracic chamber surrounded by atmospheric pressure during diastole and connected to an atrium and artery that are surrounded by intrathoracic pressure. The upstream intrathoracic atrium therefore empties into a ventricle initially set to atmospheric pressure. This continues until the limits of the pump chamber are approached and intraventricular pressure increases, causing filling to stop. As the intrathoracic pressure surrounding an atrium is decreased, atrial pressure relative to atmosphere will decrease, and less blood will enter the ventricle. When atrial pressure relative to atmosphere decreases to zero or becomes equal to the intraventricular pressure, ventricular filling will stop, since at that point no atrioventricular gradient exists. If atrial pressure relative to atmosphere is then decreased further, blood will move retrograde to the lower-pressure atrium from the ventricle until the respective atrioventricular valve closes. In our study, this situation was reflected by transient retrograde airflow out of the left ventricle and presumptive premature closure of the mitral valve with a hiccup during diastole.

The opposite events occur during an acute increase in intrathoracic pressure around an atrial capacitance chamber. As the pressure around the atrium increases, blood is "squeezed" from the atrium into the ventricle. Blood continues to be transferred from the atrium to the ventricle until 1) the atrium is empty, indicating that venous return to the atrium has stopped; 2) the limits of the ventricular chamber are reached, causing ventricular diastolic pressure to rapidly rise; or 3) the next systole begins prior to complete filling. The filling of the artificial LV demonstrated a consistent immediate increase when intrathoracic pressure increased during spontaneous expiration or a cough.

If a local mechanical constraint by the lungs were the dominant force influencing the gradient for atrial filling and emptying, then little change would have been noted with a hiccup when lung volume changed minimally.

**TABLE 1. Expected Events in Early Inspiration or Expiration in Spontaneous Ventilation**

<table>
<thead>
<tr>
<th>Artificial Heart</th>
<th>Native Heart</th>
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</thead>
<tbody>
<tr>
<td>Inspiration (ITP)</td>
<td>Expiration (ITP)</td>
</tr>
<tr>
<td>RV</td>
<td>LV</td>
</tr>
<tr>
<td>RV</td>
<td>LV</td>
</tr>
</tbody>
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† = increase, ‡ = decrease. — = no net change.

Operator-controlled pump setting is adjusted for complete RV filling with variable ejection and complete LV ejection with variable filling for the artificial heart. Changes in intrathoracic pressure may influence RV preload in the artificial heart if systemic venous pressures are low. Under these circumstances with operator-set complete RV ejection, respiratory variation in RV filling will be the same as in the LV.

Where the influences of the respiratory-induced changes in preload and afterload would have opposite effects on stroke volume, the net result most commonly seen is given.

Data for events with a native heart are from references 1, 3, 4, 6, 7, 18.
Lung inflation during either spontaneous or positive pressure ventilation under zone III conditions (pulmonary artery pressure > left atrial pressure > alveolar pressure) according to West et al.\textsuperscript{17} tends to express alveolar blood out of the lungs toward a downstream constant-pressure left atrium,\textsuperscript{14} (in a manner analogous to an increase in pressure around the veins, as shown in fig. 5c). If this mechanism were a dominant factor, then LV filling would have increased during spontaneous inspiration. Our study showed that it was not a dominant factor.

Under normal circumstances, ventricular stroke volume is determined by changes in preload, afterload, and contractility. If for the same preload, stroke volume varies, either contractility or afterload has changed. Unless the drive system fails, contractility can be considered constant for the artificial heart, set by the operator to deliver a given pressure as long as an expressible volume remains in the chamber. Thus, under conditions of constant preload, which for the artificial heart is most easily ascertained by observation of complete filling, variation in stroke volume reflects variation in afterload. As illustrated in figure 6, the right ventricle, pulmonary artery, left ventricle, and intrathoracic aorta normally are surrounded by similar surface pressures; this is not the case with the artificial heart. The right and left artificial ventricles eject into intrathoracic vascular compartments. With the pump’s maximal pressure maintained constant relative to atmospheric pressure, an increase in pressure around the pulmonary artery or aorta will increase the intravascular pressure against which the ventricle must eject, and hence will impede ventricular ejection. For example, if intrathoracic pressure during a cough increases above the maximum set level of ventricular pressure, no ejection will occur. In this sense, positive intrathoracic pressure increases the afterload imposed on each ventricle and thereby lengthens the time to complete ejection, causes incomplete ejection, or in the extreme case prevents ejection. A given increase in intrathoracic pressure would obviously affect the lower pressure RV more than the LV. Conversely, a negative intrathoracic pressure lowers the respective arterial pressures relative to atmosphere. This reduces the afterload against which the artificial ventricles must pump, and hence enhances ventricular ejection. With complete filling, this enhancement may be reflected by complete ejection not previously present or by complete ejection earlier in systole. Thus, the observation of a decrease in LV stroke volume during inspiration must result from a decrease in preload, since the simultaneous decrease in afterload in the intrathoracic aorta should increase the stroke volume.

Ironically, in past work, pulsus paradoxus was hypothesized to result when either a thick-walled ventricle or tamponade fluid surrounding the heart “protected” the LV from “seeing” the decrease in intrathoracic pressure and hence caused blood to pool in the left atrium or pulmonary vascular bed. Although this reasoning is incorrect for the normal native heart,\textsuperscript{7} it is precisely the dominant mechanism of the inspiratory decrease in LV stroke volume and arterial pressure with an artificial LV. Similarly, the incorrect reasoning that in the normal system a decrease in arterial pressure relative to atmospheric secondary to a decrease in intrathoracic pressure would reflect a decrease in LV afterload, when actually it reflects an increase in afterload,\textsuperscript{5} is correct with an artificial LV, because the LV and aorta have different surrounding compartment pressures.

Our observations suggest that careful evaluation for potential clinical problems should be considered if inspiratory variation in ventricular filling or arterial pressure is pronounced. Understanding the physiology of this respiratory variation requires consideration of numerous factors that can affect ventricular filling and ejection, including the degree of intrathoracic pressure fluctuation, the underlying hemodynamic status of the patient, and the operator-controlled settings of the artificial heart.

Respiratory-induced variation in both ventricular filling and ejection occurs in an artificial heart. This can produce a pattern similar to that of pulsus paradoxus in patients with airway obstruction. Neither changes in ventricular interdependence nor changes in contractility can explain the respiratory variation that results from a decreased LV preload. A conceptual framework based on consideration of the artificial ventricles as extrathoracic pumps can account for respiratory variation during either spontaneous or mechanical ventilation. While the respiratory variation in biventricular output is similar to but not the same as that normally observed, the mechanisms produc-

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**Fig. 6.** The difference between the normal circulation, in which the native RV and LV exist within the intrathoracic compartment, and the circulatory system with an artificial heart, in which the ventricles exist functionally as extrathoracic pumps. The influence of ventricular interdependence in which the right and left hearts compete for the same total space within the pericardium is not indicated, but is another major difference between the normal circulatory system and one with artificial ventricles. See text for discussion.
ing these variations differ markedly from the normal physiologic condition. Patterns of respiratory-induced variations may have clinical diagnostic use.

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