Basic physiologic principles provide a systematic method by which hemodynamic data and the integration of the cardiovascular system can be understood.

**FLOW THROUGH A TUBE**

Flow through a rigid tube is governed by the pressure difference across the tube and the resistance.

\[ Q = \frac{(P_u - P_d)}{R} \]

where \( Q \) is flow, and \( P_u \) and \( P_d \) are upstream and downstream pressures, respectively. For laminar flow, resistance is inversely proportional to the fourth power of the tube radius \( r \). If a sample of blood is forced in one end of the rigid tube, an equal volume must exit the other end because blood is nearly incompressible. An abrupt increase in \( P_u \) results in an almost instantaneous increase in flow out of the tube. By comparison, flow from the downstream end of a tube that can distend, does not immediately increase as the tube is distended by a portion of the fluid entering it. It’s ability to distend is characterized by its compliance \( C \). Compliance is defined as the change in volume \( (V) \) for a given pressure change \( (C = \Delta V/\Delta P) \), or more accurately \( C = dV/dP \). The increase in volume of the tube also raises the pressure exerted by its walls by an amount equal to \( \Delta P/C \). Eventually, the diameter of the tube increases such that the pressure exerted by its walls equals that exerted by the fluid within. Subsequently, the flow of fluid out of the tube equals that entering it.

For most physiologic purposes, flow through a compliant tube can be simplified by modeling it as a cylindrical reservoir with a rigid tube affixed to its base (Fig. 37.1). If flow into the reservoir is increased, the height of the fluid gradually rises. The rate of rise is governed by the resistance of the rigid tube and the area of the reservoir. The higher the resistance or the smaller the area, the more rapid the rise. The compliance of a distensible tube is approximated by the area of the reservoir. The rise in \( P_r \), with an increase in flow into the reservoir increases flow through the affixed tube. The height of fluid in the reservoir continues to increase until flow out of the tube equals flow into the reservoir. The time required to reach this new equilibrium depends upon the time constant of the system, which is proportional to the product of the reservoir area, the force of gravity, and the pipe resistance. It takes approximately five time constants before flow through the pipe equals the new flow rate into the reservoir. The time constant can be quite large if the pipe has a high resistance or if the reservoir represents a large compliance (i.e., has a large area). Intuitively, this occurs because if the resistance is high the increase in flow will be small for a given increase in \( P_r \). Conversely, if the reservoir has a large area, the rate at which pressure (height) rises is small for a given increase in flow into the reservoir. In either case it will take longer for the outflow to increase to equal the inflow. A more complete model can be derived by regarding the circulation as many small reservoir-pipe in series, but the single reservoir-pipe model is adequate to explain the basic properties of the circulation.

**Figure 37.1** A model for venous return. Flow through a distensible tube can be approximated by a reservoir affixed to the end of a rigid pipe with fluid flowing freely into the reservoir. The upstream pressure \( P_u \) is determined by the height of the fluid in the reservoir \( (h) \) multiplied by the fluid density.
VENOUS RETURN

Implicit in the calculation of systemic vascular resistance is the premise that the circulation is modeled as a single vessel that includes both the arterial and venous systems. For this model, cardiac output is determined by the difference between the mean arterial \( P_a \) and right atrial \( P_{ra} \) pressures divided by the systemic vascular resistance \( R \). To understand the factors that determine venous return \( Q_v \), Guyton and colleagues recognized the advantages of dividing this single vessel circulation into two distinct (arterial and venous) vascular beds (Fig. 37.2). The pressure difference driving blood through the arterial portion of the circulation is the difference between \( P_a \) and a hypothetical pressure at the end of the arterial bed termed the mean systemic pressure \( P_{ms} \). \( P_{ms} \) should not be confused with mean arterial pressure. Therefore, cardiac output through the arterial bed \( (Q) \) is given by Equation 37.2.

\[
Q = (P_a - P_{ms})/R
\]

where \( R_a \) is the resistance of the arterial vessels. Because the flows through the arterial and venous beds are usually the same, cardiac output is also given by Equation 37.3.

\[
Q = (P_{ms} - P_{ra})/R_v
\]

where \( R_v \) is the resistance of the venous bed. Therefore, \( P_{ms} - P_{ra} \) is the driving pressure for \( Q_v \). If \( P_{ms} \) and \( R_v \) remain constant, a decrease in \( P_{ra} \) will increase \( Q_v \), by increasing the driving pressure across the venous bed.

MEAN SYSTEMIC AND MEAN PULMONIC PRESSURES

Guyton and colleagues used a right-heart bypass preparation to determine \( R_a \) and \( P_{ms} \) in dogs. Blood from the vena cavae was diverted from the right atrium (RA) into a reservoir and outflow from the reservoir was pumped into a cannula inserted into the main pulmonary artery (PA), thus bypassing the RA and right ventricle (RV). The height of the blood in the reservoir relative to the heart determined \( P_{ra} \) and pump flow replaced RV output. It is interesting to note that when pump flow was increased, the left ventricle (LV) output had to increase to match the output of the pump. This occurred because of increased LV end-diastolic volume (EDV), which causes LV end-diastolic pressure (EDP) and, thus, the PA pressure to rise. Under these conditions, the only way that the LV can affect cardiac output is by increasing its EDP, causing a passive increase in PA pressure to the point where the pump can no longer maintain its output. This would decrease the output of the pump by what might be termed its afterload.

Using this experimental preparation, the transient effects on \( Q_v \) resulting from changes in \( P_{ra} \) caused by adjusting the reservoir height can be measured. For example, elevating the reservoir increases \( P_{ra} \) thereby causing a transient decrease in \( Q_v \) because \( P_{ms} - P_{ra} \) decreases (assuming that \( P_{ms} \) does not change). Because pump flow would remain constant while \( Q_v \) decreased, blood would be translocated from the reservoir into the systemic blood vessels. After about five time constants (approximately 10 minutes), \( Q_v \) would reach a new equilibrium that would depend on the area of the reservoir, the amount of blood it initially contained, and its height. If the initial change in \( Q_v \) is determined for different reservoir heights, the relation between \( Q_v \) and reservoir height (i.e., \( P_{ra} \)) should be linear (Fig. 37.3), assuming \( P_{ms} \) and \( R_v \) are constant. As shown by rewriting Equation 37.3, \( Q_v \) can be calculated as follows.

\[
Q_v = (P_{ms} - P_{ra})/R_v
\]

This equation describes a straight line with a slope of \((-1/R_v)\). This line is termed the venous return line. Below approximately 1.5 mmHg, \( Q_v \) is independent of \( P_{ra} \) because \( P_{ra} \) is less than intrathoracic pressure, creating a waterfall so that the downstream pressure \( P_{ra} \) is essentially intrathoracic pressure, not \( P_{ra} \). By determining \( Q_v \) for a wide range of values for \( P_{ra} \) and finding the straight line that relates these two parameters, venous resistance and \( P_{ms} \) may be determined. The value of \( P_{ra} \) at which \( Q_v \) is zero is \( P_{ms} \) (i.e., \( P_{ms} = P_{ra} \) when \( Q_v = 0 \)). These same principles apply to the pulmonary circulation and the LV and left atrium (LA), except the relevant pressures are mean pulmonic and LA pressure.

Although this experiment allows \( P_{ms} \) and \( R_v \) to be determined, it provides no insight into the mechanical factors that determine \( P_{ra} \). Since \( P_{ra} = P_{ms} \) when \( Q_v = 0 \), \( P_{ms} \) could be determined by...
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Consequently, $P_{ms}$ is predominately determined by the volume of blood in the veins and their compliance. When a patient receives a volume infusion, initially most of the fluid resides in the systemic venous bed. This results in an increase in $P_{ms}$ that causes a parallel shift to the right of the venous return line (see Fig. 37.3). Consequently, if $P_{ms}$ remained the same, $Q_r$ would increase. However, as discussed below, $P_{ms}$ also increases by an amount that depends on RV contractility, PA pressure, and RV and RA compliances. The relative increases in $P_{ms}$ and $P_{ms}$ determine the overall change in $Q_r$ that ultimately results from changes in systemic volume.

VENOUS COUPLING WITH THE VENTRICLES

Changes in volume status

The coupling of $Q_r$ with the ventricles can be visualized using Starling curves. These curves take many forms, for example mean atrial or ventricular EDP versus stroke work, cardiac output, or stroke volume (SV); however, the relation between EDP and SV lends itself well to characterization of ventricular-venous coupling. Starling curves shift upward or downward with changes in contractility, arterial pressure, and compliance (Fig. 37.4) and tend to become flat as atrial pressure increases, which reflects the steepness of the compliance curve at high EDP.

In a steady state, SV must equal $Q_r$/beat. This corresponds to the intersection of the plot of $Q_r$ with the Starling curve. The effects of changing $P_{ms}$ are shown in Figure 37.5a for the systemic veins and the RV. At point A, curve 1 for $Q_r$ corresponding to a $P_{ms}$ of 12mmHg, intersects the Starling curve at a SV of 48mL/beat. As a result of an abrupt decrease in $P_{ms}$ from 12 to 8mMg (e.g. caused by acute bleeding), the line for $Q_r$ shifts downward and to the left (curve 2), and a new equilibrium point is reached at point C. In the first instant following the decrease in $P_{ms}$, $P_{ms}$ would not change. Consequently, $Q_r$ would decrease along the rightmost vertical dashed line in Figure 37.5a from point A to $A'$, which would cause $Q_r$ to decrease from 48 to 27mL/beat. Because the RV would still be ejecting 48mL/beat, RV volume would begin to decrease. The associated decrease in RV EDP would cause RV SV to move leftward on the Starling curve toward point B. The decrease in RV SV would also decrease $P_{ms}$, causing $Q_r$ to increase toward point B'. However, RV SV would still exceed $Q_r$ (in Fig. 37.5a being approximately 41 and 32mL/beat, respectively). Therefore, RV would continue to eject more than it received from the systemic veins and its EDV would continue to fall, resulting in a continuous decrease in RV EDP along the Starling curve. The resultant fall in $P_{ms}$ would cause $Q_r$ to continue to increase along curve 2. This process would continue until RV SV and $Q_r$ equalize (point C, 35mL/beat), the end result being a decrease in both $P_{ms}$ and SV.

A volume infusion produces a rise in SV by the same mechanisms that occur in acute hypovolemia, which decreases it (Fig. 37.5b). If enough volume were infused to raise $P_{ms}$ from 4 to 9mmHg (Fig 37.5b, line 2), SV would increase from 27 to 54mL/beat. If additional volume were infused, increasing $P_{ms}$ by the same increment to 14mmHg (line 1), the SV increases by only 10mL/beat to 64mL/beat. The larger rise in SV that occurs if the patient is hypovolemic is a consequence of the steepness of the Starling curve at lower $P_{ms}$.

Because identical changes in $P_{ms}$ can result both from changes in venous compliance and blood volume, it seems reasonable to refer to the functional volume status of a patient rather than trying to separate compliance from actual blood volume.
Figure 37.4 A family of Starling curves relating stroke volume (SV) to atrial pressure. An increase in arterial pressure or decrease in contractility shifts the curve downward and to the right (curves a–c). A decrease in ventricular compliance will have the same effect but it will also alter the shape of the curve. The dashed curve shows a decrease in SV as atrial pressure increases above 8mmHg. This descending limb does not actually exist but may appear to occur if an increase in atrial pressure shifts the Starling curve downward, for example by causing ischemia or atrioventricular valvular regurgitation. The equilibrium SV is determined by the points of intersection of a venous return line with the Starling curves (A, B, and C). As expected, increasing pressure, decreasing contractility, or decreasing compliance all cause a decrease in SV and an increase in atrial pressure.

For example, assuming normal values for $P_{na}$ and $P_{ns}$ (5 and 12mmHg, respectively), the pressure gradient causing $Q_s$ is 7mmHg. A patient with a $P_{na}$ of 15mmHg and a SV of 70mL (assuming normal SV of 60mL) would be functionally hypervolemic because to have a supranormal $Q_s$ with a $P_{na}$ of 15mmHg, $P_{ns}$ would still have to be 7mmHg greater (22mmHg) than $P_{nv}$. The requisite increase in $P_{ns}$ to 22mmHg could only be achieved by hypervolemia or by a decrease in venous compliance, which could only be inferred from knowledge of the disease state and previous therapy, not by measurement. Consequently, it seems reasonable to refer to such a patient as functionally hypervolemic and to a patient's functional volume status as being determined by actual blood volume and venous compliance. This implies that measurement of absolute blood volume might be less useful than expected because a patient could still be "functionally" euvolemic owing to changes in venous compliance despite abnormal absolute blood volume.

Figure 37.5 Effects of changes in mean systemic pressure on stroke volume (SV) and venous return. (a) Changes in the equilibrium point between venous return and right ventricular SV following an abrupt decrease in mean systemic pressure ($P_{ms}$), from 12–18mmHg. The end result is move from point A to a new equilibrium point at C (see text). (b) Changes in SV with volume status. Volume infusion increases SV and acute hypovolemia (curve 3) decreases it. Volume infusions at normovolemia are less effective in increasing SV (2 to 1) than at hypovolemia (3 to 2) because the Starling curve is relatively flat above the normal operating point.

Changes in the Starling curve
Changes in the Starling curve affect EDP and SV. In contrast to acute hypovolemia, a downward shift in the Starling curve leads to an increase in $P_{na}$ despite the decrease in SV. This can be
explained through the steps outlined in Figure 37.6. At the initial equilibrium (point A), \( Q_i \) and \( P_i \) equal 48mL/beat. In the instant following the shift in the Starling curve from curve a to b, SV falls to 33mL/beat (point A') because of the decrease in ejection fraction. However, atrial pressure, and therefore \( Q_i \), do not change, which causes ventricular volume to begin to increase. The resultant increase in EDP causes \( Q_i \) to decrease along line 1 from point A toward point B. Because \( Q_i \) would still exceed SV, EDV and, thus, EDP increase toward point B', resulting in an increase in SV. However, \( Q_i \) still exceeds SV (approximately 43 and 37mL, respectively). Therefore, the ventricle would continue to eject less than it received from the \( Q_i \). This process continues until the SV equals \( Q_i \) (point C, SV 37mL/beat). The end result would be a decrease in SV and an increase in \( P_{vc} \).

The Starling curve does not provide a unique measure of intrinsic ventricular function because arterial pressures and ventricular compliance also affect its appearance. Therefore, before developing an integrated model of the circulation by coupling both ventricles and the arterial circulation, a more basic approach to cardiac mechanics will be discussed.

**MECHANICAL PROPERTIES OF THE VENTRICES**

**Stroke volume versus cardiac output**

In describing cardiac function, cardiac output is usually the central focus. However, this can be misleading. Consider a patient about to undergo major vascular surgery who has a history of episodic pulmonary edema and mild, stable angina. An exercise history cannot be elicited because of claudication. A PA catheter is placed before induction of anesthesia; the data obtained are shown in Figure 37.7. Although the pressures appear relatively normal, the cardiac output of 3.5L/min seems quite low. However, as the heart rate is only 50 beats/min, the SV is 70mL (3500/50), which is normal. Although SV does tend to decrease somewhat with age, indexing for body surface area or weight does not decrease individual variation. Normal values for younger individuals are 60–70mL/beat, falling to 60mL/beat or less for elderly patients.

Following induction of anesthesia and tracheal intubation, the patient’s central pressures rise substantially and new SV segment depression is noted in the lateral precordial lead. Are the elevations in central pressures secondary to ischemic dysfunction? The cardiac output has increased from 3.5 to 5.6L/min accompanied by an increase in heart rate from 50 to 70 beats/min. Therefore, the SV has increased from 70 to 80mL. Using the arguments developed above, it is evident that the increase in central pressures could not have occurred solely because of LV dysfunction. Were this the case, without an increase in \( P_{ps} \) and \( P_{mp} \), the elevated atrial pressures would have caused a decrease in \( Q_i \) and, thus, SV. Consequently, regardless of the effects ischemia might have had on the ventricles, it could not account for an increase in SV without a concomitant increase in \( P_{ps} \) and \( P_{mp} \). Viewed somewhat differently, acute LV dysfunction would shift the LV Starling curve downward, causing an increase in \( P_{ps} \) and a decrease in SV (see Fig. 37.6). This would cause a passive increase in PA pressures, which would impair RV ejection. The resultant downward shift in the RV Starling curve would increase \( P_{ps} \) and decrease systemic \( Q_i \). The net effect would be an increase in central pressures and a decrease in SV.

From these considerations, the data in Figure 37.7 are not consistent with primary ventricular dysfunction but, in fact, are more consistent with acute hypervolemia. Increased myocardial oxygen consumption and decreased perfusion secondary to the increases in ventricular volumes and filling pressures could have caused the ischemia. It is possible that if the ischemia were not present, the SV would have increased above 80mL, but this cannot be determined from these data. Because the patient did not receive a rapid volume infusion, it seems likely that he had an acute decrease in the compliance of the systemic venous bed. This, in effect, would reduce the volume of the container for blood in the systemic circulation (e.g. the veins), resulting in functional hypervolemia (i.e. an increase in \( P_{ps} \)). Consequently, the changes in pressures observed likely resulted from inadequate anesthesia leading to sympathetic nervous activation and causing, among other things, a decrease in the compliance of the venous bed and functional hypervolemia. This interpretation suggests that therapy should be directed toward deepening anesthesia and thereby blunting the sympathetic response rather than toward treating the ischemia.

In contrast, consider the same data shown in Figure 37.7 but with a heart rate of 100 beats/min following intubation. In this case, the SV would have decreased from 70 to 56mL. These data, using the arguments presented above, are consistent with ischemic dysfunction. An important point is that if central pressures are acutely increased from ventricular dysfunction, the SV (but not necessarily the cardiac output) must decrease. This example illustrates that the interpretation of data obtained from a PA catheter is critically dependent upon the heart rate as this is the only number that differs in the two
different conditions just discussed. There are two other important points illustrated by this example. First, an increase in cardiac output does not necessarily indicate an improvement in ventricular function. Second, changes in filling pressures cannot be interpreted without measuring the associated changes in cardiac output and calculating SV.

Determinants of stroke volume
The terms preload, afterload, and contractility are often used in describing ventricular performance. Preload and afterload have precise definitions in papillary muscle experiments. However, because the geometry and dynamics of a functioning ventricle differ so radically from those of a papillary muscle, these terms are not directly applicable to an intact ventricle. Although a definition analogous to preload is possible in an intact ventricle, functionally it is more useful to define ventricular preload as the EDV. Because the force generated by a ventricle changes constantly during ejection, the term afterload, which is the constant force during shortening in papillary muscle experiments, cannot be applied directly to ejecting ventricles. Rather than struggling to find an analogous definition, it seems more important to define the factors that have a major influence upon SV. Because SV is, by definition, the difference between the EDV and the end-systolic volume (ESV), the problem becomes delineating the determinants of these two volumes.

End-diastolic volume
Although the pressure–volume relationship at end diastole (EDPVR), which relates EDV to EDP under different conditions, is usually referred to as the compliance curve, it is actually an elastance curve that is defined as the change in pressure per unit change in volume (dP/dV). Therefore, end-diastolic elastance is the slope of the EDPVR at any point. In contrast, compliance is the change in volume with pressure (dV/dP). The EDPVR is relatively flat over the normal operating range of ventricles (100–150mL) so that changes in EDP vary relatively small changes in EDV. However, as the EDV increases above normal, the EDPVR becomes steeper and a small rise in EDV causes a large increase in EDP (Fig. 37.8a). If the shape of the EDPVR were known, the EDV could be determined from the EDP. However, the shape of the EDPVR is not precisely known even in normal individuals, let alone for patients with heart disease. Consequently, accurate determination of the EDV from the EDP is not generally possible. Therefore, although the EDV increases with a rise in EDP, the magnitude of this increase is unpredictable. For this reason, the effects of changes in EDP on SV are more useful than the exact value of the EDP.

End-systolic volume
The isovolumic peak pressure–volume relation
If the outflow of the LV or RV were occluded to prevent ejection, it would develop the maximum peak pressure possible for that EDV and contractile state. This type of beat is termed isovolumic because there is no volume change during systole (i.e. EDV = ESV). These beats may be characterized by plotting ventricular pressure against volume (Fig. 37.8b). When beats at different EDV are plotted in this way, keeping contractile state constant, the relation between peak pressure and volume is nearly linear and is termed the end-systolic pressure–volume relation (ESPVR). For a given ESPVR, the peak pressure of an isovolumic beat could be determined from the EDV and vice versa. This is analogous to the relationship between pressure and volume at end-diastole except that the EDPVR is highly nonlinear. The ESPVR intersects the volume axis at Vp, which is the volume at which the ventricle does not develop any pressure. If the contractile state increases (e.g. by an inotrope), peak isovolumic pressure at any volume also increases. However, the increase would be proportionally greater at larger volumes, causing a counterclockwise rotation of the ESPVR around Vp. Therefore, for isovolumic beats, the slope of the ESPVR becomes steeper with an increase in contractility, and flatter with a decrease in contractility and its slope provides a volume-independent measure of contractility for isovolumic beats.

The end-systolic pressure–volume relation for ejection
The ESPVR model can be extended to describe end-systole for ejecting beats, providing a load-independent definition of contractility that relates ESV and end-systolic pressure (ESP) in a simple manner. The period of systole preceding ejection is isovolumic because both valves are closed, and it appears on a pressure–volume diagram as a vertical line positioned at the EDV (Fig. 37.9a). When the pressure within the ventricle exceeds that in the aorta or PA, the aortic or pulmonic valve opens and ejection begins. The onset of ejection is marked by an abrupt change
from a vertical line to a curve with decreasing volume and increasing pressure. The rise in pressure depends upon complex interactions between the arterial tree and ventricle. If ejection were interrupted by clamping the aorta or PA, the respective ventricle would continue to develop pressure until the pressure equaled the maximum possible for an isovolumic beat at the clamped volume (dashed line 1 in Fig. 37.9a). That is, the pressure would rise until it touched the ESPVR. This maximum decreases later in ejection because of the decreasing volume (dashed line 2). At end-ejection, the pressure falls on the ESPVR. That is, the ESP is determined by the volume at this point (ESV) and the contractile state of the ventricle defined as the slope of the ESPVR. The aortic or pulmonic valve then closes and isovolumic relaxation begins. When ventricular pressure decreases below atrial pressure, the mitral or tricuspid valve opens and the ventricle again fills until atrial ejection ends returning the ventricle to its EDV, and the cycle repeats itself.

If ejections are repeated with different vascular properties (Fig. 37.9b) or starting from different EDV (Fig. 37.9c), the end of ejection still always falls approximately on the ESPVR. This property allows SV to be described by two pressures and two elastances. Specifically, EDV is determined by EDP and EDPVR, and ESV by ESP and ESPVR (Fig. 37.9d). That is, end-diastolic and end-systolic elastances define the relationships between pressures and volumes at end-diastole and end-systole, respectively. Consequently, if these elastances are known, the pressures at end-diastole and end-systole will determine the respective volumes.

The time-varying elastance model
During an ejection, the pressure at any instant falls on the same line (Fig. 37.10). The slope of this time-varying elastance line increases with time during a beat until end-systole is reached. That is, it becomes progressively steeper until it reaches a maximum value, which corresponds to the time at which ESP is developed. The same time-varying elastance lines define the instantaneous relation between pressure and volume for both isovolumic beats and ejections if contractility is constant. For this reason, this description is called the time-varying elastance model of cardiac contraction. Unfortunately, relaxation cannot be defined accurately using a time-varying elastance model. This model has important implications in predicting myocardial oxygen consumption.

Limitations of end-systolic parameters
Although the ESPVR provides a very useful approach to understanding ventricular performance, it has limitations. First, end-ejection does not always occur precisely at the ESPVR, especially for large or rapid LV ejections or in the RV. As SV increases with increasing EDV, ESP begins to fall below that predicted by the ESPVR (shortening deactivation). This has been attributed to an internal resistance in the contracting ventricle. Second, the ESPVR appears to be nonlinear, especially if measured over a wide range of EDV and at different contractile states. As contractility increases, the ESPVR tends to become convex away from the volume axis. Conversely, the ESPVR for depressed ventricular activity tends to be concave away from the volume axis. Third, elastance at end-systole is not always the same as maximum elastance, probably because of resistive and inertial forces affecting end-ejection. The slope of the maximum elastance may be steeper than that for the end-systolic elastance. While maximum elastance is conceptually more appealing and more useful for modeling of cardiac mechanics, end-systolic elastance is clinically a more useful
Figure 37.9 Ventricular ejection and pressure-volume relationships (PVR). (a) Normal stages in an ejection cycle. Ejection occurs in a counterclockwise direction. Vertical dashed lines 1 and 2 indicate pressures that would developed if further ejection were prevented at the respective volumes. (b) Effects of changes in vascular properties on ventricular ejection. At unchanged end-diastolic volume (EDV) and pressure at ejection (diastolic pressure), an increase in arterial resistance would develop higher ejection pressures and a smaller stroke volume (SV). (c) Effects of changes in left ventricular EDV with constant vascular properties on ejection. Arterial pressures and SV change, although end-systole still occurs when the ejection intersects the ESPVR. (d) The SV is determined by two elastance curves, the ESPVR and EDPVR, and two pressures, end-systolic (ESP) and end-diastolic (EDP).
Time varying elastance model

Figure 37.10 Time-varying elastance model. During ejection, the ventricular pressure at a given time after the onset of systole is related linearly to ventricular volume. As systole progresses, the line defining this relationship rotates counterclockwise around Vo. That is, it becomes progressively steeper until it reaches a maximum value, the end-systolic pressure–volume relation, a change that can be viewed as progressive stiffening of the ventricle.

measure of contractility. The ESPVR will refer to this unless otherwise stated.

Afterload
Despite these limitations, the ESPVR provides a clinically useful framework for assessing ventricular performance and predicting how changes in blood pressure will affect cardiac performance. In contrast, making such assessments using the term afterload is difficult if not impossible. As discussed, the definition of afterload as defined for papillary muscles is difficult to apply to the intact ventricle. Systemic vascular resistance (SVR) is often used as a surrogate for afterload in the intact ventricle. However, this definition can be misleading. First, it is evident from the discussion of the ESPVR that SVR can only affect ejection by changing the ESP. Second, because ESP can also be affected by vascular compliance, even with a constant resistance, and because both compliance and resistance are greatly simplified vascular concepts, SVR is not a reasonable definition of afterload. Third, even if vascular compliance is constant, ESV is generally impossible to calculate from SVR alone, so that changes in SVR cannot be directly related to changes in ventricular performance. Consequently, vascular properties such as resistance and compliance are not measures of ventricular afterload but only modifiers of load as measured by pressure. Therefore, rather than attempting to apply the papillary muscle definition of afterload to the intact ventricle, it seems more sensible to relate the simpler concepts based on the ESPVR and EDPVR to performance. Moreover, because the time-varying elastance model shows that

ventricular volume at any instant during systole depends mostly upon the pressure at that instant, instantaneous pressure and, more importantly, ESP provide more information about ventricular ejection than do vascular properties.

THE STARLING CURVE

The Starling curve is useful for understanding the coupling of the heart and the vasculature. However, it is affected not only by the contractile state of the heart but also by the ESP and EDPVR. Some insight into the interaction of these factors on the Starling curve may be gained by considering the factors that can cause a decrease in SV for a given EDP. By definition, a decrease in SV can result from a decrease in EDV or an increase in ESV. For a fixed EDP, the EDV can decrease only if the EDPVR is shifted upward, that is, if the ventricle becomes stiffer. Such a change leads to a decrease in SV for any EDP simply because the corresponding EDV is smaller. This emphasizes the importance of the EDPVR in determining the shape of the Starling curve. In fact, as shown below, the shape of the Starling curve is largely a reflection of the EDPVR.

The Starling curve is often described as being shifted downward by increases in load or decreases in contractility, the definitions of load and contractility are usually vague and quantification of their interactions is lacking. Deriving Starling curves from pressure–volume diagrams can overcome these limitations by defining contractility as the slope of the ESPVR and load as ESP (Fig. 37.11). The shape of the Starling curve is essentially a reflection of the portion of the EDPVR for a volume greater than the ESV.

Derivation of Starling curves
Although the Starling curve is often described as being shifted downward by increases in load or decreases in contractility, the definitions of load and contractility are usually vague and quantification of their interactions is lacking. Deriving Starling curves from pressure–volume diagrams can overcome these limitations by defining contractility as the slope of the ESPVR and load as ESP (Fig. 37.11). The shape of the Starling curve is essentially a reflection of the portion of the EDPVR for a volume greater than the ESV.

The effects of load on the Starling curve can be derived using the same process with different ESP and, therefore, ESV. Curves 2 in Figure 37.11b was generated as described above except the ESP was decreased to 50mmHg (ESV = 20mL). The origin of this Starling curve is shifted slightly to the left
Derivation of Starling curves from pressure-volume relationships

Figure 37.11 Derivation of Starling curves from pressure-volume relationships. (a) Ejections with the same end-systolic volume (ESV, 40mL) but progressively increasing end-diastolic volumes (EDV, a through e). Contractility is defined as the slope of the end-systolic pressure-volume relation (ESPVR); here it is 5mmHg/mL. (b) The effect of load (ESP) on the Starling curve. Data for the end-diastolic pressure (EDP) derived from panel a are plotted against stroke volume (SV). Lowering the ESV by decreasing ESP to 50mmHg results in a larger SV for the EDV a–e. This shifts the Starling curve slightly upward and to the left. (c) Starling curves at depressed contractility (slope of the ESPVR 1.7mmHg/mL). (d) The effect of load at depressed contractility. Curve 1 was generated at an ESP of 150mmHg as in (a). Curve 2 was derived by fixing the ESV at 40mL [identical to curve 1 in (b)]. However, the ESP is only 50mmHg compared with 150mmHg in (a) because the ESPVR is much flatter. For any EDP the SV is much smaller than in (a) because ESP is increased.

The interaction of contractility and load on the shift of Starling curves can also be derived. Curve 2 in Figure 37.11d was generated with the same ESV as in Figure 37.11b, whereas curve 1 corresponds to the same ESP. Because the ESPVR is relatively flat, the ESV of 40mL reflects an ESP of only 50mmHg (line 2 in Figure 37.11c). The corresponding Starling curve is identical to curve 1 in Fig. 37.11b because the ESV at an ESP of 50mmHg is the same as that for the ventricle in Figure 37.11a with an ESP.
of 150 mmHg. However, the Starling curve for the ESP of 150 mmHg in the depressed ventricle (curve 1 in Figure 37.11d) is shifted markedly downward and to the right, because for an ESP of 150 mmHg, the ESV must increase to 100 mL. This shift is much greater for a depressed than for a normal ventricle because of the greater change in ESV for a given change in ESP. This is consistent with the observation that an increase in 'load' (ESP) has a much greater effect on a depressed than for a normal ventricle (dilated myopathy), the corresponding Starling curve appears almost normal for low loads. However, it would still shift markedly downward and to the right with an increase in blood pressure because of the large increase in ESV and, therefore, EDV and ESP. This illustrates why a patient with a dilated myopathy may have a normal EDP and SV if the blood pressure is well controlled but have a decrease in SV and large increases in filling pressures with hypertension.

INTEGRATING THE CARDIOVASCULAR SYSTEM

At equilibrium not only must Q, equal SV for each ventricle, but also SV of both ventricles must also be equal (they may not be exactly equal because of coronary and bronchial circulations or cardiac lesions producing intracardiac shunts). One way to analyze the effects of changes in any component of the circulation upon other components is to plot ventricular pressure-volume curves, Starling curves, and curves for Q, for each ventricle adjacent to each other. The interactions of the critical elements of these idealized hemodynamic analyses can then be visualized. Three important basic principles in such analyses are:

- EDV and ESV are controlled by two pressures, the ESP and ESPV, and two elastances, the EDPVR and the ESPVR.
- The value of Q, is determined by the difference between P, or P, and their respective atrial pressures divided by their respective venous resistances;
- P, and P, are largely determined by the volumes of blood and the compliances in the respective venous beds (normal values for both P, and P, are 10-15 mmHg).

These principles are used below to establish some basic behaviors of the circulation. The following discussion shows intuitively that transient changes in SV resulting from alterations in LV performance cannot be sustained unless there is a secondary effect on P,. Changes in P, are usually mediated by alterations in RV performance or by ventricular interactions.

Increasing LV contractility in a normal ventricle

Consider the effects of a hypothetical drug that can increase LV contractility and therefore its ejection fraction (EF), without directly affecting the RV or any vascular bed. Suppose that initially both ventricles have an EDV of 100 mL and EF of 60%, and that this drug increased the EF of the LV to 80%. Assume also that this drug was applied in such a way that the increase in LV EF occurred just before the onset of systole. The effects, which are derived from simulations in which the slope of the LV ESPVR was increased from 3 mmHg/mL to 5 mmHg/mL, are summarized in Figure 37.14. On the beat 1 after the drug was applied, the LV would eject 80 mL while the RV still ejected 60 mL. This, in effect, would translocate 20 mL of blood from the pulmonary circulation into the systemic circulation, which is equivalent to infusing 20 mL of blood directly into the systemic venous circulation. Clinically, such an infusion would not increase systemic venous return appreciably because P, would not change significantly, for several reasons. First, the compliance of the systemic venous bed is very large so that such a change in volume would produce relatively little change in the size of the veins and thus their distending pressure, P,. Second, regardless of venous compliance, because 20 mL is less than 0.5% of systemic venous blood volume it is too small a volume to increase P, appreciably. However, the situation in the pulmonary circulation is much different. Because the pulmonary circulation contains only 200 mL to 300 mL of blood, a 20 mL volume loss is 7-10% of the total pulmonary blood volume. Consequently, P, would decrease, thereby reducing venous return to the LV. Moreover, assuming that systemic pressure did not change much, the increase in contractility would cause a decrease in the LV ESV from 40 mL to 20 mL. Consequently, it would take an additional 20 mL to fill to the original EDV of 100 mL. Extracting this additional 20 mL from the pulmonary circulation would also decrease P,. The LV would then fill to an EDV of only 88 mL instead of 100 mL. Therefore, for beat 2, the LV will eject only 70 mL (0.8 × 88 mL) instead of 80 mL. Moreover, because the EDV of the LV has decreased from 100 mL to 88 mL, the LV ESP will also decrease slightly resulting in a slight drop in pulmonary artery pressures. Therefore, the RV ESP will also decrease slightly causing an ejection to a slightly lower RV ESV. Consequently, on beat 2 the RV EF will increase from 60% to 61% so that the RV will eject 62 mL (101 × 0.61) instead of 60 mL. Thus, on the second beat, an additional 9 mL (70.4-61.6 mL, to be exact) will be translocated from the pulmonary to the systemic circulation, bringing the total translocated to 29 mL. For the reasons stated above, this still will not appreciably increase systemic venous return. However, the total loss from the pulmonary circulation is now 29 mL, which is 10-15% of the initial total pulmonary blood volume. Accordingly, LV filling will decrease further so that in beat 3 the LV would fill to only 81 mL. As this process continues, the pulmonary circulation will become hypovolemic enough, i.e. P, will decrease enough to reduce the LV EDV substantially with a very little increase in systemic venous return. Because of this, and only a small change in RV EDP due to LV interaction from the slight decrease in LV ESP; the SV of the RV will not change appreciably. Thus, when a new equilibrium is finally reached, the SV will still be close to 60 mL/beat (actually 62 mL/beat) and the LV EDV will be reduced to approximately 77 mL. That is, the increase in LV EF will have resulted in a decrease in LV size without an appreciable increase in SV.

It is noteworthy that in this example, a pulmonary artery catheter (PAC) would show a decrease in pulmonary capillary wedge pressure (PCWP) and pulmonary artery pressures (PAP), but essentially no change in P or SV. Clearly, the patient's blood volume has not changed. This illustrates why the PCWP cannot be used to assess volume status. The decrease in PCWP without a change in SV indicates that the pulmonary circulation has become functionally hypovolemic. That is, P must have decreased otherwise the pressure gradient for pulmonary venous return, P - left atrial pressure (P), would have increased, thereby increasing venous return to the left atrium. The unchanged SV and P indicate that the functional volume of the systemic circulation, P, did not change. Therefore the CVP,
Simulation of the effects of an acute decrease in the contractility of the left ventricle

Figure 37.12 Simulation of the effects of an acute decrease in the contractility of the left ventricle (LV) on hemodynamics with initially normal ventricles and vascular volumes. The decrease in contractility is depicted by the shift in the end-systolic pressure–volume relationship (ESPVR) from curve a to the flatter curve b in (b), giving the downward shift in the Starling curve from curve a to curve b in (d). If blood were not translocated into the pulmonary circulation, venous return ($Q_v$) would still have to lie on line 1 in (d). The Starling curve would intersect this line at point B, corresponding to the dashed ejection curve in (b). However, because the LV stroke volume (SV) would then be only 40mL compared with the right ventricle (RV) SV of 60mL, blood would be translocated from the systemic into the pulmonary circulation until the SV of the two ventricles was again equal. This movement has little effect in the large systemic blood volume (change in mean systemic pressure is too small to show in (c)), but a relatively large effect in the smaller pulmonary blood volume (mean pulmonary pressure increases markedly in (d) from line 1 to line 2). When the new equilibrium is reached, SV is decreased because the increase in LV end-diastolic pressure (EDP) [point C in (d)] increased pulmonary artery pressure [dashed ejection in (a)], impairing RV ejection. As a result, the RV Starling curve shifts downward from a to b in (c). The new RV equilibrium occurs on essentially the same $Q_v$ line [point B in (c)] at a higher RV EDP. A lower RV contractile state would cause a greater depression in the RV Starling curve. In this case, the equilibrium SV and pulmonary capillary wedge pressure would be lower and right atrial pressure would be higher.
Figure 37.13 Effects of increased arterial pressure on cardiac performance with normal ventricles. Because of the steepness of the LV end-systolic pressure-volume relationship (ESPVR), the end-systolic volume (ESV) is normal (40mL) at an end-systolic pressure (ESP) of 130mmHg. To achieve a 60mL stroke volume (SV), the end-diastolic volumes (EDV) must be 100mL. If the LV ESP were increased abruptly to 180mmHg, for example, aortic cross-clamping, and there were no reflex changes in contractility, LV ESV would increase by only 15mL because the ESPVR is steep [dashed ejection in (b)]. This would cause a slight downward shift in the LV Starling curve [curve b in (d)]. The LV SV would decrease by 15mL on the first beat, translocating this volume from the systemic into the pulmonary circulation. This would have virtually no effect on mean systemic pressure or on systemic venous return (Qs) (c) but would increase the blood volume of the pulmonary circulation by approximately 10% and Pmv slightly [line 2 in (d)]. Consequently, Qs to the LV would increase slightly but the increase in LV EDP would be negligible; both the LV Starling curve and Qs curve would shift slightly and the intersection would occur at a slightly higher LV EDP [b in (d)]. Pulmonary artery pressure would not change. The RV would not detect the alteration in the LV; right atrial pressure would not change and, thus, Qs and the SV of the RV would remain 60mL. At the new equilibrium, the central pressures and SV would not have changed measurably. But the LV EDV would have increased by 15mL.
which approximates \( P_{m} \) and SV can be used to estimate the functional systemic blood volume, whereas the PCWP and SV can be used to estimate the functional pulmonary blood volume. However, because the RV is generally very compliant, a small increase in \( P_{m} \) reflecting a volume infusion can cause a relatively large change in RV EDV. Such a change would be detectable from an increase in SV. Because the LV is stiffer than the RV, such an increase in SV might be reflected by a larger increase in PCWP.

This is often the justification for using the PCWP as a surrogate for evaluating systemic volume status. However, such changes in PCWP may be variable and systemic volume status (SVS) is easily inferred from the SV and CVP. Therefore, physiologically, the PCWP can be, at most, a very indirect indicator of SVS but it can also be misleading when used for this purpose.

**Decreasing contractility in a normal left ventricle**

The methods described above can also be used to analyze the effects of an acute decrease in LV ejection fraction, for example from 60 to 30%, as might occur with severe global ischemia (Fig. 37.12). For simplicity, assume that systemic pressures and RV contractility do not change. Because the decrease in LV contractility causes the LV initially to eject less than the RV, blood is translocated from the systemic to the pulmonary circulation, increasing \( P_{m} \) and, thus, \( Q_{s} \) to the LV. Consequently, LV EDV and LV EDP increase, causing a passive increase in PA pressure. The subsequent increase in RV ESP impedes RV ejection, increasing RV EDV and EDP. The resultant increase in \( P_{m} \) decreases systemic \( Q_{s} \). As this process continues, RV SV falls because of the continuing passive increase in PA pressure, so that translocated volumes decrease with each beat. Concomitantly, LV SV increases because of the increase in \( Q_{s} \) to the LA. Eventually, the SV of the two ventricles would again be equal but less than the initial 60 mL and greater than the 30 mL ejected by the LV immediately following its depression.

These hemodynamic changes are governed by the following three mechanisms. First, translocation of blood into the pulmonary circulation increases \( P_{m} \) and \( Q_{s} \) to the LV thereby also increasing LV EDV and the LV SV. Second, increased LV EDV causes LV EDP to rise and passively elevates PA pressure, RV ESP, and RV ESV, thus decreasing RV SV. This increases \( P_{m} \) because systemic \( Q_{s} \) initially exceeds that required to fill the RV to its initial EDV from the now increased SV. The resultant increase in \( P_{m} \) without a change in \( P_{m} \) causes systemic \( Q_{s} \) to decrease. Third, the increase in PV EDF effectively decreases compliance of the RV and RA by shifting the septum rightward - further increasing \( P_{m} \) and decreasing systemic \( Q_{s} \). The equilibrium SV depends upon both RV contractility and compliance. A normal RV will not increase its ESV much with the increase in PA pressure. Therefore, the equilibrium SV will be closer to the initial 60 mL than the initial depressed LV SV of 30 mL, and \( P_{m} \) will not change much. In contrast, if the RV was initially depressed, the rise in PA pressure increases RV ESV more than for a normal RV, ultimately leading to a greater increase in \( P_{m} \) and a lower equilibrium SV.

This simulation illustrates that the LV can affect cardiovascular function mainly only by altering (in this case increasing) \( P_{m} \). The increase in \( P_{m} \) without a significant change in \( P_{m} \) causes systemic \( Q_{s} \) to decrease, which can occur both from ventricular interaction and from passive increases in PA pressure. As will be seen below, this concept allows for a much simpler analysis of circulatory dynamics. As in the preceding example, the patient's volume status did not change appreciably, yet both the pulmonary capillary wedge pressure (PCWP) and \( P_{m} \) increased. From these data alone, it would be difficult to determine whether systemic volume had changed because even assuming \( P_{m} \) had not changed, the exact decrease in \( Q_{s} \) expected from the increase in \( P_{m} \) would not be known precisely. Nonetheless, the increase in PCWP does not imply that systemic blood volume has increased. It is, in fact, a result of the increase in pulmonary blood volume.

**Ventricular loading**

Consider a 67-year-old man who has a history of limited exercise tolerance because of claudication who is about to undergo resection and grafting of an infrarenal aortic aneurysm. He also suffers from chronic stable angina but there is no evidence of cardiac dysfunction. Because of the uncertainty of the extent of his coronary artery disease, a PA catheter is inserted. The initial data are shown in Fig. 37.7. These data, although showing a slightly elevated PCWP, show a normal SV and are, therefore, consistent with ventricles that function relatively normally. The effects of increased arterial pressure with aortic cross-clamping are shown in Fig. 37.13. For simplicity, assume that the systolic pressure corresponds to ESP, although the actual ESP is always lower than systolic pressure. The LV EDV has to rise by 15 mL to match the SV of the RV, which does not produce a physiologically significant change in the LV EDP, or in interaction. Therefore, when the new equilibrium is reached, the LV EDP (and, therefore, PCWP) and SV do not change measurably. This illustrates why changes in systemic pressures usually do not produce measurable hemodynamic changes if both ventricles are normal.

In contrast, suppose that the contractile state of the LV was lower than suspected from the data in Figure 37.7. As shown in Fig. 37.14, at equilibrium there would be a marked elevation in LV EDP and thus PCWP, a small elevation in \( P_{m} \), and a decrease in SV. These changes are similar to those that would occur with depression of the LV without an increase in LV ESP. If the RV contractile state was depressed, at equilibrium \( P_{m} \) would be greater whereas the SV and left atrial pressure (\( P_{L} \)) would be lower.

**Ventricular unloading**

It is sometimes stated that an arterial vasodilator increases blood pressure if a patient has a dysfunctional LV because the resultant increase in cardiac output is greater than the decrease in arterial resistance. However, analysis using the ESPVR indicates that this is not possible unless the contractile state also increases. Consider a patient who has a LV myopathy of unknown etiology who presents with an acute decrease in contractility of his already compromised LV. The patient, who is complaining of increased dyspnea, an inability to lie flat, and severe fatigue, is found to have pulmonary edema and is transferred to an intensive-care unit where a PA catheter is placed (see Fig. 37.7 for typical data). Pressure–volume diagrams, Starling curves, and \( Q_{s} \) plots for this patient are depicted in Fig. 37.15. Because the \( P_{m} \) is as high as the normal \( P_{m} \) of 12 mmHg, \( P_{m} \) must have increased substantially. That is, the patient must be functionally hypervolemic in the systemic circulation to maintain a sufficient venous pressure between \( P_{m} \) and \( P_{m} \) to provide systemic \( Q_{s} \), even to match the low SV. Furthermore, the SV cannot be readily increased with additional volume because any further increment in LV EDV would markedly increase the LV EDP because of the steepness of the EDPVR in this region. This would further limit RV performance and exacerbate the
Figure 37.14 Effects of increased arterial pressure on cardiac performance with a normal right and depressed left ventricle (LV). The slope of the LV end-systolic pressure-volume relationship (ESPVR) is flatter than in Fig. 37.13, consequently the LV end-diastolic volume (EDV) corresponding to an LV end-systolic pressure (ESP) of 130mmHg is 90mL rather than 40mL. For a 60mL stroke volume (SV) LV EDV must then be 150mL. End-diastolic pressure-volume relationship (EDPVR) is slightly more compliant than in Fig. 37.13 so that LV end-diastolic pressure (EDP) is still 12mmHg despite the larger LV EDV. Because the LV is depressed, an increase in LV ESP to 180mmHg (e.g. by aortic cross-clamping) causes LV end-systolic volume (ESV) to increase by 40mL to 130mL instead of by only 15mL. Consequently, the Starling curve is shifted downward more [curve b in (d)] by aortic cross-clamping than in Fig. 37.13. Moreover, a much larger volume of blood is translocated into the pulmonary circulation, causing a greater increase in mean pulmonary pressure [line 2 in (d)] and, therefore, in LV EDV. The new equilibrium would occur at point B in (d). Mean systemic pressure and, therefore, the systemic venous return (Qs) line would not change appreciably (c). However, as LV EDV increases, LV EDP also begins to rise substantially because the LV EDPVR is relatively flat at higher volumes. The rise in LV EDP causes a passive rise in pulmonary artery pressure, thereby increasing RV ESP and ESV. Consequently, RV EDV increases [dashed ejection in (a)] because systemic Qs initially exceeds that required to fill the RV to its original 100mL EDV. However, with the increase in RV EDV and, therefore, RV ESP the RV Starling curve shifts downward [curve b in (c)]. Right atrial pressure (Pra) would increase, causing systemic Qs to fall [point B in (c)]. At equilibrium, there is a marked elevation in LV EDP and thus pulmonary capillary wedge pressure, a small elevation in Pra, and a decrease in SV. The effects of ventricular interaction are not shown but would stiffen the RV and RA, further increasing Pra and decreasing the equilibrium SV.
Effects of decreasing arterial pressure on cardiac performance with biventricular depression and systemic hypotension

Figure 37.15 Effects of decreasing arterial pressure (unloading) on cardiac performance with biventricular depression and systemic hypotension. The slope of the left ventricular (LV) end-systolic pressure-volume relationship (ESPVR) is very flat, giving an LV end-systolic volume (ESV) of 165 mL and pressure (ESP) of only 90 mmHg. The stroke volume (SV) is only 30 mL because with a LV end-diastolic volume (EDV) of 195 mL, the LV end-diastolic pressure (EDP) is 30 mmHg, resulting in elevated pulmonary artery pressure. The resultant elevation in right ventricular (RV) ESP causes RV ESV to increase to 115 mL. Compared with the RV in Fig. 37.12, the RV ESPVR is somewhat flattened, indicating some RV depression. In addition, because of ventricular interaction resulting from the acute increase in LV size and rightward septal displacement, the RV EDPVR is considerably stiffer than in Fig. 37.12. Therefore, even though the RV EDV is only 145 mL, the RV ESV and EDP also are increased. The high RV EDP (and, thus, right atrial pressure) impedes venous return. Arterial dilation would decrease LV ESP, causing a reduction in both LV EDV and EDP [dashed ejection, (b)]. This would cause a passive reduction in pulmonary artery pressure and, thus, RV ESP, which would cause a decrease in RV EDV and EDP [dashed ejection, (a)]. These changes cause a shift in the equilibrium point from A to B in (c) and (d).
pulmonary edema. Very large increases in RV EDP would be required to produce even small increases in RV EDV.

If this patient were given a pure arterial dilator, the resultant decrease in the LV ESP would increase LV SV, causing blood to be translocated from the pulmonary to the systemic circulation (Fig. 37.15). Even if Qs to the LV did not change, the LV EDV would decrease because of the reduction in the LV ESV. However, the actual LV EDV after application of the dilator would depend upon two factors. First, the decrease in Rv decreases from the translocation of blood from the systemic circulation would reduce LV filling. Second, because the LV EDV (actually ESV) is the downstream pressure for pulmonary Qs, the decrease in the LV EDV resulting from the decrease in the LV EDV would tend to increase LV filling. Regardless of which factor dominates, the LV EDV would decrease somewhat, resulting in a decrease in LV EDV and, thus, PA pressure and RV ESP. This, in turn, would reduce RV ESV thereby improving RV ejection, reducing RV F'DV and, therefore, Ppa. The final result would be an increase in Qs and a new equilibrium at which SV increases and both right- and left-sided EDP decrease. Importantly, this effect could not be achieved without the decrease in systemic blood pressure, which caused the initial decrease in LV ESV. This is consistent with published data on unloading. These results suggest that the only way that blood pressure could increase with 'unloading' would be if the contractile state of the LV improved (e.g. by resolution of global ischemia). This analysis leads to the conclusion that if systemic blood pressure increases with the use of an arterial or venous dilator, the LV most likely was ischemic.

The principle point in these examples is that changes in LV SV cannot be sustained unless systemic Qs is also affected. Because of the relatively large blood volume of the systemic circulation and the high venous compliance, the systemic vasculature is essentially unaffected by (uncoupled from) changes in forward flow of the LV. That is, the LV can only change Qs by altering Ppa because it cannot move sufficient blood out of the pulmonary vasculature to affect Psys. Conversely, changes in RV or LV performance can move enough blood from the systemic to the pulmonary circulation to increase Psys and, therefore, Qs to the LV. However, this still cannot be sustained unless Ppa also changes enough to cause systemic Qs, to match this change. The LV can cause the requisite changes in Ppa either directly by changing the effective compliance of the RV and RA by ventricular interaction or indirectly by affecting RV ejection via changes in the PA pressure. If the RV is normal, the former mechanism seems to be more important.

CONCLUSIONS

An understanding of basic physiologic principles simplifies analysis of data obtained with invasive cardiovascular monitoring. Moreover, it allows prediction of the effects of interventions on mechanical performance. Because the volumes of the ventricles are usually unknown, often one can only guess at the contractile state of the ventricles. However, as illustrated in the examples, perturbation of the system (e.g. by a drug or volume infusion) yields considerable information about the state of the ventricles. In this way, a hypothesis can be tested; if it proves to be wrong and things are not changing in a salutary direction, it may be revised and another therapy tried. By repeated perturbations, the principles described can usually be used to estimate the relative contractile state of both ventricles, the functional central and peripheral blood volumes, and the likely effect of various interventions upon ventricular mechanics.

Key References


Further Reading