functions of its different vessels. Thus, the large arteries and veins are primarily compliance vessels in the sense that only small pressure differences are needed to drive the cardiac output through these vessels, while their changes in volume are highly significant. The main site of resistance is in the tissues themselves (primarily at the level of the smallest arteries, the *arterioles*), where volume changes are less important but where large pressure drops are observed.

Second, the approximation of a linear relation between flow and pressure differences (1.3.1) is a good approximation when we allow for changes in resistance. This may appear to be a circular argument, since we can always make (1.3.1) true by introducing the definition $R = (P_1 - P_2)/Q$. In fact, tissues exhibit reasonably constant values of $R$ under conditions where the diameters of their blood vessels remain constant. When $R$ changes, a physiological explanation must be sought. This explanation usually involves a stimulus that leads to contraction or relaxation of the smooth muscles in the walls of the arterioles. Such a stimulus may be generated by the nervous system, by circulating hormones (or other substances circulating in the blood), or by the action of locally produced products of metabolism. Some of these effects will be studied in Section 1.9, on autoregulation.

Finally, there is no such justification for the linear compliance relations that we have assumed. It is an excellent approximation to say that the volume of a blood vessel is determined by the internal pressure, but the relationship between volume and pressure becomes progressively less compliant as it is distended. The linear model is introduced for simplicity.

## 1.4 The Heart as a Pair of Pumps

A pump is a device that can accept fluid at low pressure ($P_1$) and transfer it to a region where the pressure is high ($P_2 > P_1$). Thus, the pump performs work on the fluid; the rate at which work is performed by the pump is the product of the volume rate of flow $Q$ and the pressure difference $P_2 - P_1$. To characterize the pump we need to say how $Q$ depends on $P_1$ and $P_2$.

Consider, for example, the left side of the heart (Figure 1.3). The left ventricle is equipped with an inflow (mitral) valve and an outflow (aortic) valve. When the ventricle is relaxed (diastole) the inflow valve is open and the outflow valve is closed. During this period of time, the left ventricle receives blood from the left atrium at a pressure that is essentially that of the pulmonary veins. Thus, for the left ventricle, $P_1 = P_{sv} = 5$ mmHg. When the ventricle contracts (systole), the inflow valve closes and the outflow valve opens. Then the left ventricle actively pumps blood into the systemic arterial tree. Thus, for the left ventricle, $P_2 = P_{sa} = 100$ mmHg

What determines the output of the left ventricle under these conditions? To answer this question we follow an approach introduced by Sagawa and

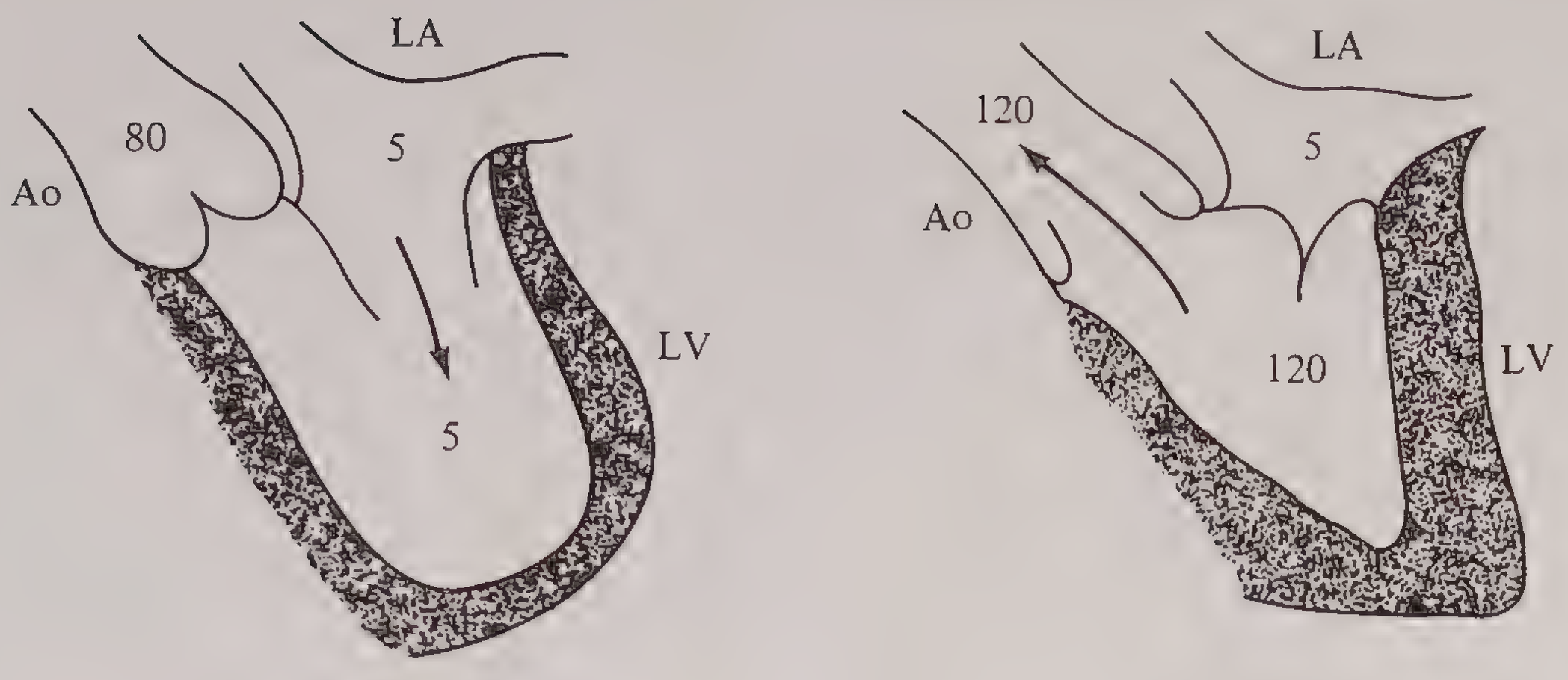


Figure 1.3. Cross section of the left side of the heart with the left ventricle in DIASTOLE (relaxed) and SYSTOLE (contracted). LA = left atrium, LV = left ventricle, Ao = Aorta. The numbers in the chambers are pressures in millimeters of mercury (mmHg). Note that the inflow (mitral) valve is open in diastole and closed in systole, whereas the outflow (aortic) valve is closed in diastole and open in systole. Pressures are approximately equal across an open valve, but can be very unequal, with the downstream pressure higher, across a closed valve. The valves ensure the unidirectional flow of blood from left atrium to left ventricle to aorta.

his collaborators: We regard the ventricle as a compliance vessel whose compliance changes with time. Thus, the ventricle is described by

$$V(t) = V_{\mathrm{d}} + C(t)P(t), \tag{1.4.1}$$

where $C(t)$ is a given function with the qualitative character shown in Figure 1.4. The important point is that $C(t)$ takes on a small value $C_{\mathrm{systole}}$ when the ventricle is contracting, and a much larger value $C_{\mathrm{diastole}}$ when the ventricle is relaxed. For simplicity, we have taken $V_{\mathrm{d}}$ to be independent of time.

Using (1.4.1), we can construct a pressure–volume diagram of the cardiac cycle (Figure 1.5). For a detailed explanation of this type of diagram, see the paper by Sagawa et al., cited in Section 1.14. For our purposes, it is sufficient to note that the maximum volume achieved by the ventricle (at end-diastole) is given by

$$V_{\mathrm{ED}} = V_{\mathrm{d}} + C_{\mathrm{diastole}}P_{\mathrm{v}}, \tag{1.4.2}$$

while the minimum volume (achieved at end-systole) is given by

$$V_{\mathrm{ES}} = V_{\mathrm{d}} + C_{\mathrm{systole}}P_{\mathrm{a}}, \tag{1.4.3}$$

where $P_{\mathrm{a}}$ is the pressure in the arteries supplied by the ventricle and $P_{\mathrm{v}}$ is the pressure in the veins that fill it. Thus, the stroke volume is given by

$$V_{\mathrm{stroke}} = V_{\mathrm{ED}} - V_{\mathrm{ES}} = C_{\mathrm{diastole}}P_{\mathrm{v}} - C_{\mathrm{systole}}P_{\mathrm{a}}. \tag{1.4.4}$$

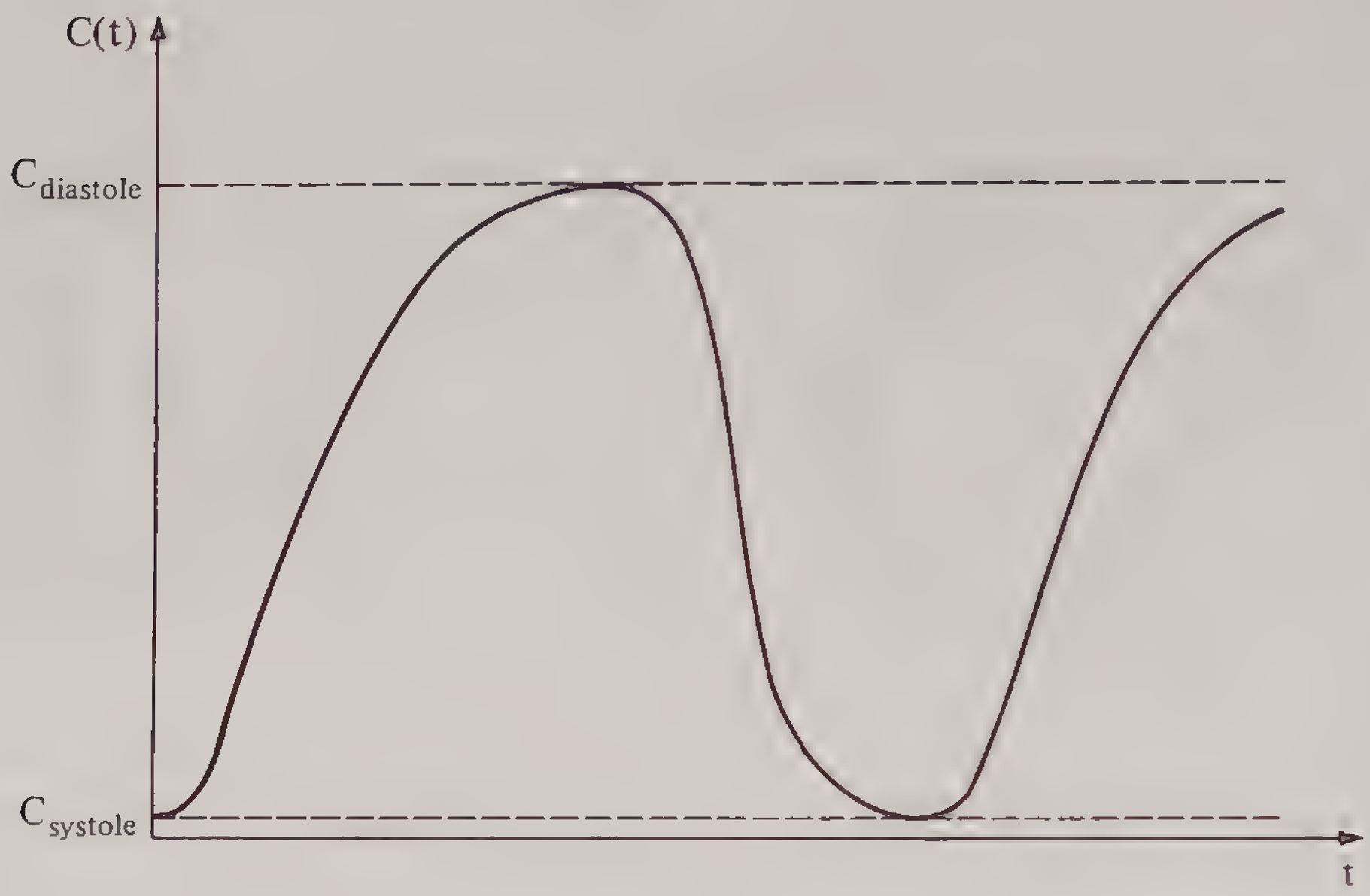


Figure 1.4. Compliance of either ventricle as a function of time. During diastole, the compliance rises from a low value $C_{\text{systole}}$ to a high value $C_{\text{diastole}}$ as the ventricle relaxes. During systole, it does the reverse as the ventricle contracts.

A particularly simple special case is when $C_{\text{systole}} = 0$, so that the systolic line is vertical in Figure 1.5. This gives a stroke volume

$$V_{\text{stroke}} = C_{\text{diastole}} P_{\text{v}}, \tag{1.4.5}$$

which is the model of the ventricle that we shall use. Finally, if $F$ (frequency) is the heart rate (beats per minute), we get cardiac output

$$Q = F V_{\text{stroke}} = F C_{\text{diastole}} P_{\text{v}}. \tag{1.4.6}$$

For the present, $F$ is taken to be constant, although we shall consider changes in heart rate later in the chapter. We define

$$K = F C_{\text{diastole}}, \tag{1.4.7}$$

so that $Q = K P_{\text{v}}$. We call $K$ the *pump coefficient* of the ventricle. Although $F$ is the same for the two sides of the heart (which are driven by the same pacemaker), the constant $C_{\text{diastole}}$ is greater in the thin-walled right ventricle than in the thick-walled left ventricle, so $K$ is greater on the right than on the left. Also, the two sides of the heart are connected to different venous systems. Thus, we have the following expressions for the right and

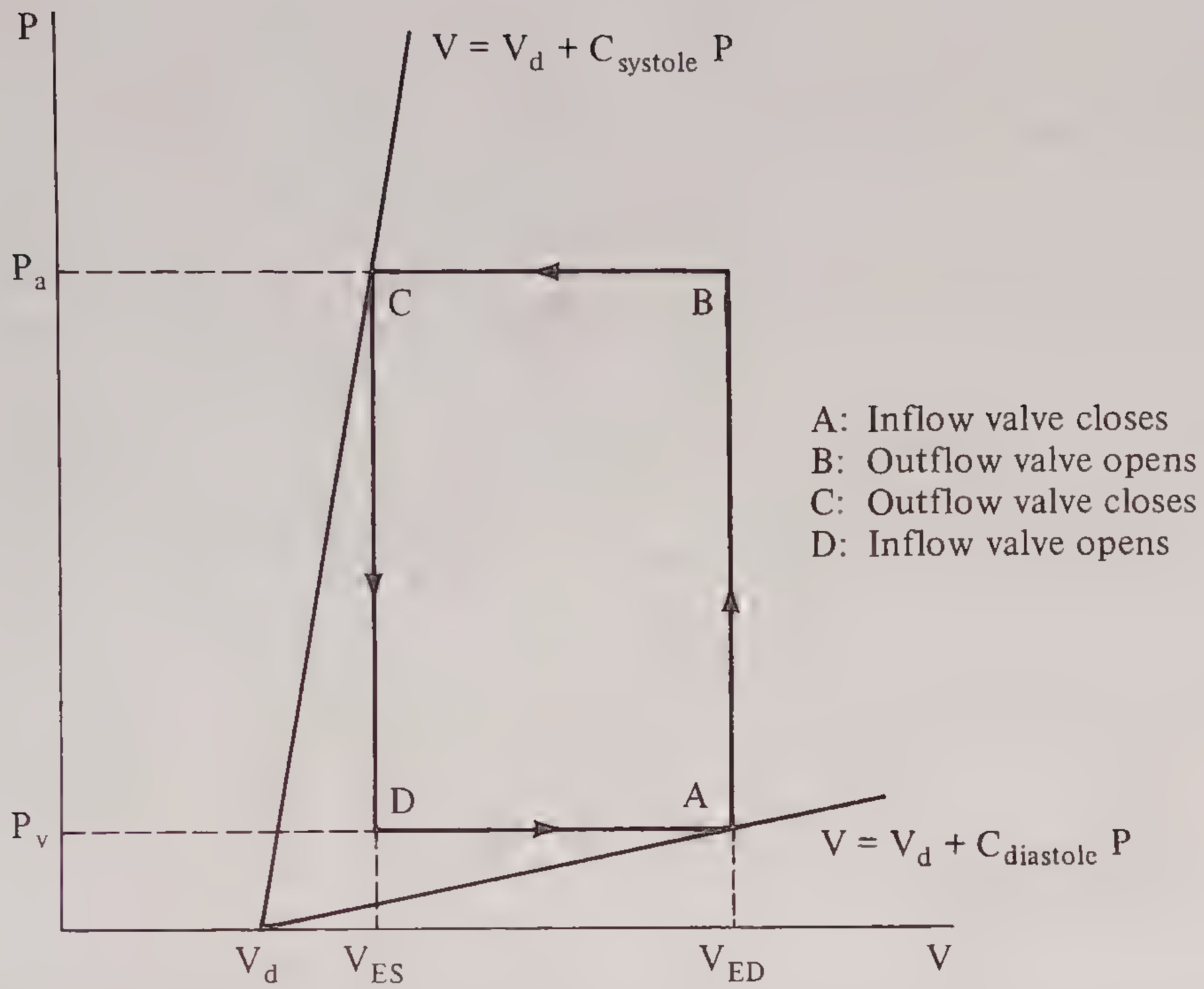


Figure 1.5. Pressure–volume diagram for either ventricle. Landmarks on the volume axis ($V$) are $V_\mathrm{d}$ = "dead volume," i.e., the volume at zero transmural pressure, $V_\mathrm{ES}$ = end-systolic volume, $V_\mathrm{ED}$ = end-diastolic volume. Landmarks on the pressure ($P$) axis are the venous (inflow) pressure $P_\mathrm{v}$, and the arterial (outflow) pressure $P_\mathrm{a}$. Since the venous pressure is essentially the same as the atrial pressure, it may be considered the inflow pressure for the ventricle. Slanting lines radiating from $V_\mathrm{d}$ describe the pressure–volume relationships of the ventricle at the end of diastole and at the end of systole; these lines are labeled with their equations. At other times, the pressure–volume relationship is indicated by a similar slanting line (not shown) that lies between the two lines shown. Rectangular loop ABCDA shows the path followed by the ventricle during the cardiac cycle. On the vertical branches (AB and CD), both valves are closed, so the volume of the ventricle is constant. On each of the horizontal branches, one valve is open: the arterial (outflow) valve along BC and the atrioventricular (inflow) valve along DA. The pressure difference across the open valve is here idealized as zero, and the arterial and venous pressures are idealized as constants. The names of the four phases of the cardiac cycle are AB = isovolumetric contraction, BC = ejection, CD = isovolumetric relaxation, DA = filling.

left cardiac outputs:

$$Q_{\mathrm{R}} = K_{\mathrm{R}} P_{\mathrm{sv}} \tag{1.4.8}$$

$$Q_{\mathrm{L}} = K_{\mathrm{L}} P_{\mathrm{pv}}. \tag{1.4.9}$$

Throughout this section we have tacitly assumed that the pressure outside the heart is zero (atmospheric). If not, then the distending pressures during diastole are not simply $P_{\mathrm{sv}}$ and $P_{\mathrm{pv}}$ but $P_{\mathrm{sv}} - P_{\mathrm{thorax}}$ and $P_{\mathrm{pv}} - P_{\mathrm{thorax}}$, where $P_{\mathrm{thorax}}$ is the pressure in the chest. In fact, $P_{\mathrm{thorax}}$ is slightly negative (with respect to the atmosphere), and this contributes to increased cardiac output by increasing the end-diastolic volume $V_{\mathrm{ED}}$. This effect was first noticed because it disappears when the chest is opened during surgery. In the model developed below, we assume for simplicity that $P_{\mathrm{thorax}} = 0$ so that we can use (1.4.8) and (1.4.9) without modification. Then, effects of $P_{\mathrm{thorax}} < 0$ are considered briefly in Exercises 1.8 through 1.9.

## 1.5 Mathematical Model of the Uncontrolled Circulation

In this section we put together the ideas that have been developed above to construct a mathematical model of the circulation. In the form that we first present it, the model lacks the control mechanisms that regulate the circulation and make it serve the needs of the body. In subsequent sections we will use this model in several ways:

1. to study the *self*-regulating properties of the circulation, independent of external control mechanisms;

2. to explain the need for external control mechanisms;

3. to serve as a foundation on which we can construct a simple model of the control of circulation.

Our model is defined by the following equations (see Figure 1.6): First, we have the equations of the right and left hearts:

$$Q_{\mathrm{R}} = K_{\mathrm{R}} P_{\mathrm{sv}}, \tag{1.5.1}$$

$$Q_{\mathrm{L}} = K_{\mathrm{L}} P_{\mathrm{pv}}. \tag{1.5.2}$$

Second, we make the assumption that the systemic and pulmonary arteries and veins are compliance vessels. For simplicity, we use (1.3.2) instead of

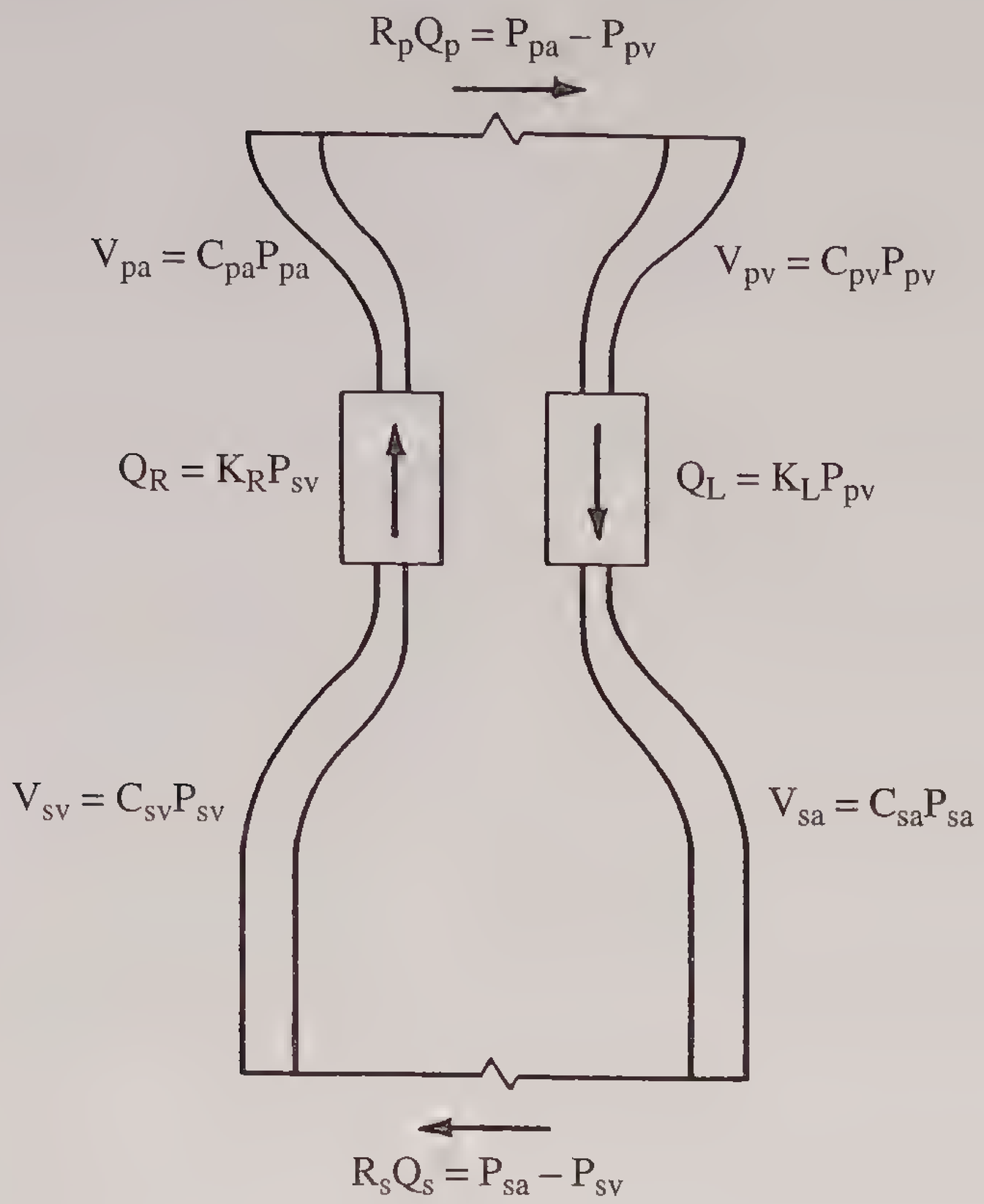


Figure 1.6. Equations of the circulation. One equation is shown for each of the eight principal elements of the circulation. For additional equations that relate these elements to each other, see the text. Each element is characterized by one parameter ($K$ = pump coefficient, $C$ = compliance, $R$ = resistance) and two or three unknowns ($Q$ = flow, $P$ = pressure, $V$ = volume). Subscript notation is s = systemic, p = pulmonary, a = arterial, v = venous, L = left, R = right.

(1.3.3). That is, we neglect $V_{\rm d}$ in these vessels. This gives the equations

$$V_{\rm sa} = C_{\rm sa}P_{\rm sa}, \tag{1.5.3}$$
$$V_{\rm sv} = C_{\rm sv}P_{\rm sv}, \tag{1.5.4}$$
$$V_{\rm pa} = C_{\rm pa}P_{\rm pa}, \tag{1.5.5}$$
$$V_{\rm pv} = C_{\rm pv}P_{\rm pv}. \tag{1.5.6}$$

Third, we assume that the systemic and pulmonary tissues act like resistance vessels, so that

$$Q_{\rm s} = \frac{1}{R_{\rm s}}(P_{\rm sa} - P_{\rm sv}), \tag{1.5.7}$$
$$Q_{\rm p} = \frac{1}{R_{\rm p}}(P_{\rm pa} - P_{\rm pv}). \tag{1.5.8}$$

At this point, we have an equation for each element of the circulation. Each equation contains a *parameter* that characterizes that element. These

parameters are the pump coefficients $K_{\mathrm{R}}$, $K_{\mathrm{L}}$, the resistances $R_{\mathrm{s}}$, $R_{\mathrm{p}}$, and the compliances $C_{\mathrm{sa}}$, $C_{\mathrm{sv}}$, $C_{\mathrm{pa}}$, and $C_{\mathrm{pv}}$. Suppose we are given the values of these parameters. Can we use equations (1.5.1) through (1.5.8) to determine the flows, pressures, and volumes of the model circulation? The answer to this question is negative; we do not yet have enough equations to determine the 12 unknowns

$$Q_{\mathrm{R}}, Q_{\mathrm{L}}, Q_{\mathrm{s}}, Q_{\mathrm{p}}; P_{\mathrm{sa}}, P_{\mathrm{sv}}, P_{\mathrm{pa}}, P_{\mathrm{pv}}; V_{\mathrm{sa}}, V_{\mathrm{sv}}, V_{\mathrm{pa}}, V_{\mathrm{pv}}.$$

The missing equations refer not to any particular element but to the circulation as a whole and to the way that its elements are connected. (Try to discover the missing equations for yourself before reading further.)

First, it is reasonable to assume that the total blood volume $V_0$ is given. This gives the equation

$$V_{\mathrm{sa}} + V_{\mathrm{sv}} + V_{\mathrm{pa}} + V_{\mathrm{pv}} = V_0, \tag{1.5.9}$$

in which $V_0$ is an additional parameter.

Next, we assume that the circulation is in a *steady state*, so that the flow into each of the compliance vessels must equal the flow out (why?). It follows that $Q_{\mathrm{R}} = Q_{\mathrm{L}} = Q_{\mathrm{s}} = Q_{\mathrm{p}}$, so we can drop the subscripts and just refer to all of the flows as $Q$, the cardiac output.

With these additional assumptions, we have nine equations for the nine unknowns $Q, P_{\mathrm{sa}}, P_{\mathrm{sv}}, P_{\mathrm{pa}}, P_{\mathrm{pv}}, V_{\mathrm{sa}}, V_{\mathrm{sv}}, V_{\mathrm{pa}}, V_{\mathrm{pv}}$. The model is complete.

Our next task is to solve the equations of the model. That is, we want to express each of the unknowns in terms of the parameters. (Try this for yourself before reading further.) An efficient plan of attack is as follows: First, express all of the pressures in terms of the flow $Q$. Then use the compliance equations to get the volumes in terms of $Q$. Finally, substitute in the equation for the total blood volume and solve for $Q$. With $Q$ known (in terms of parameters only) we can go back and express the pressures and then the volumes in terms of parameters.

Here are the details. From the pump equations, we get the venous pressures in terms of $Q$:

$$P_{\mathrm{sv}} = \frac{Q}{K_{\mathrm{R}}}, \tag{1.5.10}$$

$$P_{\mathrm{pv}} = \frac{Q}{K_{\mathrm{L}}}. \tag{1.5.11}$$

Substituting this result in the resistance equations, we get the arterial pressures in terms of $Q$:

$$P_{\mathrm{sa}} = \frac{Q}{K_{\mathrm{R}}} + R_{\mathrm{s}}Q, \tag{1.5.12}$$

$$P_{\mathrm{pa}} = \frac{Q}{K_{\mathrm{L}}} + R_{\mathrm{p}}Q. \tag{1.5.13}$$

Substituting all four pressures into the compliance equations, we obtain

$$V_{\mathrm{sv}} = \frac{C_{\mathrm{sv}}}{K_{\mathrm{R}}} Q, \tag{1.5.14}$$

$$V_{\mathrm{pv}} = \frac{C_{\mathrm{pv}}}{K_{\mathrm{L}}} Q, \tag{1.5.15}$$

$$V_{\mathrm{sa}} = \left[\frac{C_{\mathrm{sa}}}{K_{\mathrm{R}}} + C_{\mathrm{sa}} R_{\mathrm{s}}\right] Q, \tag{1.5.16}$$

$$V_{\mathrm{pa}} = \left[\frac{C_{\mathrm{pa}}}{K_{\mathrm{L}}} + C_{\mathrm{pa}} R_{\mathrm{p}}\right] Q. \tag{1.5.17}$$

To save writing, we introduce the following combinations of parameters

$$T_{\mathrm{sv}} = C_{\mathrm{sv}}/K_{\mathrm{R}}, \tag{1.5.18}$$

$$T_{\mathrm{pv}} = C_{\mathrm{pv}}/K_{\mathrm{L}}, \tag{1.5.19}$$

$$T_{\mathrm{sa}} = (C_{\mathrm{sa}}/K_{\mathrm{R}}) + C_{\mathrm{sa}} R_{\mathrm{s}}, \tag{1.5.20}$$

$$T_{\mathrm{pa}} = (C_{\mathrm{pa}}/K_{\mathrm{L}}) + C_{\mathrm{pa}} R_{\mathrm{p}}. \tag{1.5.21}$$

Then (1.5.14) through (1.5.17) can be summarized by the equations

$$V_i = T_i Q, \; i = \mathrm{sv},\, \mathrm{pv},\, \mathrm{sa},\, \mathrm{pa}. \tag{1.5.22}$$

We are now ready to substitute these expressions in the equations for the total blood volume and solve for $Q$. We get

$$(T_{\mathrm{sa}} + T_{\mathrm{sv}} + T_{\mathrm{pa}} + T_{\mathrm{pv}}) Q = V_0, \tag{1.5.23}$$

so

$$Q = \frac{V_0}{(T_{\mathrm{sa}} + T_{\mathrm{sv}} + T_{\mathrm{pa}} + T_{\mathrm{pv}})}. \tag{1.5.24}$$

The solution is completed using the equations $V_i = T_i Q$ and $P_i = V_i/C_i$. We get

$$V_i = \frac{T_i V_0}{(T_{\mathrm{sa}} + T_{\mathrm{sv}} + T_{\mathrm{pa}} + T_{\mathrm{pv}})}, \tag{1.5.25}$$

$$P_i = \frac{T_i V_0}{C_i (T_{\mathrm{sa}} + T_{\mathrm{sv}} + T_{\mathrm{pa}} + T_{\mathrm{pv}})}, \tag{1.5.26}$$

where $i =$ sa, sv, pa, and pv. Thus, we have a formula for each unknown in terms of parameters only.

The quantitative use of these formulae depends on having numerical values for the parameters. In particular, we need *normal resting values* for the parameters so that we can use the model to predict the effects of parameter changes away from the normal resting state of the circulation. It is easy to determine the parameters from data such as are given in Table 1.1 and Section 1.2 because each equation of our model (1.5.5–1.5.9) contains exactly one of the parameters, so it can be written as a formula

Table 1.2. Normal Resting Parameters of the Model Circulation

| | **Systemic** | **Pulmonary** |
|---|---|---|
| $R$: | $R_s = 17.5$ | $R_p = 1.79$ mmHg/(liter/min) |
| $C$: | $C_{sa} = 0.01$ | $C_{pa} = 0.00667$ liters/mmHg |
| | $C_{sv} = 1.75$ | $C_{pv} = 0.08$ liters/mmHg |
| | **Right** | **Left** |
| $K$: | $K_R = 2.8$ | $K_L = 1.12$ (liters/min)/mmHg |
| $V$: | $V_0 = 5.0$ liters | |

for that parameter in terms of the observed pressures, volumes, and flows. The results of this procedure are summarized in Table 1.2.

The procedure that we have just used for *identification* of parameters is based on the assumption that the model is correct. If we improve the model, then the best choice of parameters may change. An example of this is studied in Exercises 1.7 and 1.17.

## 1.6 Balancing the Two Sides of the Heart and the Two Circulations

The reader has probably noticed that most of the equations of the previous section came in pairs. The reason for this is the symmetry of form between the right and left heart and the systemic and pulmonary circulations. In fact, we can obtain one member of a pair from the other by making the subscript interchanges s $\leftrightarrow$ p and R $\leftrightarrow$ L (try it and see!). In the few equations that stand alone (because they refer to the circulation as a whole) these interchanges give us back the same equation as before.

If the corresponding parameters were quantitatively equal (that is, if we had $K_R = K_L, R_s = R_p$, etc.), then the two circulations would be quantitatively symmetrical with $P_{sa} = P_{pa}$, and so on. A glance at Tables 1.1 and 1.2 shows that this is far from being the case.

This raises the question of how the two sides of the heart and the two circulations are coordinated. What keeps the outputs of the right and left hearts equal? What mechanisms control the partition of blood volume between the systemic and pulmonary circulations? These are vital (and closely related) questions. If the left output exceeded the right output by only 10% for 1 minute, this would be enough to empty the vessels of the pulmonary circulation.

In our steady–state model of the circulation, the right and left cardiac outputs are equal by definition. In a time-dependent version of the model, we could see how this equality of output is maintained. Suppose, for ex-

ample, that $K_{\mathrm{R}}$ is suddenly reduced. Temporarily, $Q_{\mathrm{R}}$ will be less than $Q_{\mathrm{L}}$, so there will be a net transfer of blood volume away from the pulmonary circulation and into the systemic circulation. This will raise the systemic venous pressure and lower the pulmonary venous pressure. The effect of these pressure changes will be to drive the cardiac outputs back toward equality. A net rate of transfer of volume will persist until equality of output of the two sides has been restored. Then a new equilibrium is established with a different partition of the blood volume than before.

In the steady–state model, we compute only the end result of this process. Using (1.5.25) and (1.5.18) through (1.5.21), we see that

$$\begin{aligned}
\frac{V_p}{V_s} &= \frac{V_{\mathrm{pa}} + V_{\mathrm{pv}}}{V_{\mathrm{sa}} + V_{\mathrm{sv}}} \\
&= \frac{T_{\mathrm{pa}} + T_{\mathrm{pv}}}{T_{\mathrm{sa}} + T_{\mathrm{sv}}} \\
&= \left( \frac{C_{\mathrm{pa}} + C_{\mathrm{pv}}}{K_{\mathrm{L}}} + C_{\mathrm{pa}} R_{\mathrm{p}} \right) / \left( \frac{C_{\mathrm{sa}} + C_{\mathrm{sv}}}{K_{\mathrm{R}}} + C_{\mathrm{sa}} R_{\mathrm{s}} \right), \quad (1.6.1)
\end{aligned}$$

where $V_{\mathrm{p}}$ is the total pulmonary volume and $V_{\mathrm{s}}$ is the total systemic volume. Thus, the partition of the blood volume between the two circulations is determined by the parameters, and a change in parameters that temporarily produces a disparity in output between the two sides of the heart eventually results in a volume shift that compensates for the parameter change and restores the equality of output.

The key to the success of this intrinsic control mechanism is the dependence of *cardiac output* on *venous pressure*. Suppose instead that the cardiac outputs of the two sides of the heart were given and equal. In that case $Q$ would be a parameter and we would have to drop equations (1.5.1) and (1.5.2). We would have lost two equations but only one unknown, so we would be free to specify one more relationship. In fact, we could then assume that the pulmonary and systemic volumes $(V_{\mathrm{p}}, V_{\mathrm{s}})$ were separately given. This would lead to the equations

$$V_{\mathrm{sa}} + V_{\mathrm{sv}} = V_{\mathrm{s}}, \quad (1.6.2)$$

$$V_{\mathrm{pa}} + V_{\mathrm{pv}} = V_{\mathrm{p}}, \quad (1.6.3)$$

which would replace (1.5.9), increasing the number of equations by one. Thus, we would have the eight equations (1.5.3) through (1.5.8) and (1.6.2) and (1.6.3) for the eight unknowns $(V_i, P_i)$ with $Q$ as a new parameter. With these assumptions the pulmonary and systemic volumes would be arbitrary; there would be no mechanism available to hold them in a reasonable relationship to each other. These considerations show the importance of the dependence of cardiac output on venous pressure, not only for maintaining a balance between the two sides of the heart, but also for establishing a controlled partition of the blood volume between the pulmonary and systemic circulations.

## 1.7 The Need for External Circulatory Control Mechanisms

The arterioles in an exercising muscle dilate, and the systemic resistance $R_s$ falls. The cardiac output rises, and the systemic arterial pressure is maintained. The increase in cardiac output comes primarily from an increase in heart rate while stroke volume remains fairly constant.

In this section we study the consequences of a change in $R_s$ in our model of the *uncontrolled* circulation. We shall find a predicted response that is very different from the observed response described above. In the uncontrolled circulation a decrease in $R_s$ results in only a modest increase in cardiac output. The most noticeable effect is a substantial fall in systemic arterial pressure. This shows the need for the external circulatory control mechanisms that are outlined in the next section.

We begin with an obvious but important remark. The effects of a change in $R_s$ cannot be predicted solely from the equation of the systemic resistance, even though that is the only equation where $R_s$ appears. If we neglect $P_{sv}$ in equation (1.5.7) (an excellent approximation because $P_{sv}$ is about 2 mmHg, whereas $P_{sa}$ is about 100 mmHg), we get $P_{sa} = QR_s$. From this we might conclude that $P_{sa}$ is proportional to $R_s$ with $Q$ constant or that $Q$ is inversely proportional to $R_s$ with $P_{sa}$ constant. Neither conclusion is correct, since both $P_{sa}$ and $Q$ vary when $R_s$ changes. The actual effects on $P_{sa}$ and $Q$ cannot be predicted without taking all of the other equations into account. That is the essence of a system of *simultaneous* equations.

In fact, we have already taken these equations into account when we solved for the unknowns in terms of the parameters. The formulae that we need are

$$Q = \frac{V_0}{T_{sa} + T_{sv} + T_{pa} + T_{pv}} \tag{1.7.1}$$

and

$$P_{sa} = \frac{V_0}{C_{sa}} \frac{T_{sa}}{T_{sa} + T_{sv} + T_{pa} + T_{pv}}, \tag{1.7.2}$$

where $T_{sa}$, etc., are given by (1.5.18) through (1.5.21).

Using these formulae and the parameter values given in Table 1.2, we can find the effects on $Q$ and $P_{sa}$ of reducing $R_s$ to 50% of its normal value (while leaving the other parameters unchanged). The results are summarized in Table 1.3.

Note that the increase in cardiac output was only about 10% whereas the drop in arterial pressure was about 40%. This mechanism of adjusting the cardiac output is definitely inadequate to sustain reasonable levels of exercise, where cardiac output must be doubled or even tripled and where blood supply to nonmuscular tissue must be maintained.