PHYSIOLOGY OF THE ARTERIAL PULSE

Although the arterial pulse, which is considered a fundamental clinical sign of life, has been the subject of study by many physiologists as well as clinicians in the past (1–28), it received less attention by clinicians for many years after the discovery of the sphygmomanometer (29). There has been a renewed interest in this field in recent years since new techniques such as applanation tonometry are now being applied for its study (30–33). The physiology of the arterial pulse is, however, quite complicated, and the subject is often given only cursory description even in the most popular textbooks in cardiology. Also, the retained terminology and nomenclature do not help to clarify the issues (21,34). The most detailed review of the complicated physiology of both the normal and the abnormal arterial pulse can be found in some of the excellent papers of O’Rourke and his co-workers (21,35–38). The subject has, however, remained somewhat elusive even to the most interested clinicians. Therefore, in this chapter an attempt will be made to simplify some of the concepts for the sake of better understanding.

The purpose of the arterial system is to deliver oxygenated blood to the tissues but, more importantly, to convert intermittent cardiac output into a continuous capillary flow. This is primarily achieved by its structural organization (6). The central vessels, namely the aorta up to the iliac bifurcation and its main branches—the carotid and the innominate arteries—are very elastic and act in part as a reservoir in addition to being conduits. The vessels at the level of the radial and femoral arteries are more muscular, whereas the iliac, subclavian, and axillary vessels are intermediate or transitional in structure. When an artery is put into stretch, the readily extensible fibers of the vessel wall govern its behavior. The more elastic the vessel, the greater is the volume accommodated for a small rise in pressure.

It is well known that the recording obtained with a pulse transducer placed externally over the carotid artery has a contour and shape very similar to a pressure curve obtained through a catheter placed internally in the carotid artery and recorded with a strain gauge manometer system (Fig. 1). While the former records displacement of the vessel transmitted to the skin through overlying soft tissues, the latter is a true recording of the internal pressure changes. The displacement in the externally recorded tracing is due to
changes in the wall tension of the vessel similar to the recording of an apical impulse reflecting the change in left ventricular wall tension. The wall tension is governed by the principles of Laplace relationship. The tension is directly proportional to the pressure and the radius and inversely related to the thickness of the vessel wall. Since ejection of the major portion of the stroke volume takes place in the early and mid-systole, the cause of major change in tension in early and mid-systole is a result of changes in both volume and pressure. During the later part of systole and during diastole, however, the predominant effect must be primarily due to changes in pressure, although volume may also play a part. The dominance of the pressure pulse effect on the tension of the vessel wall for the greater part of the cardiac cycle is the main reason for the similarity of the externally recorded carotid pulse tracing and the internally recorded pressure curve.

Fig. 1. (A) Simultaneous recordings of electrocardiogram (ECG), phonocardiogram, and the carotid pulse. (B) Intra-aortic pressure recording in the same patient. Note the similarity of the carotid pulse tracing and the aortic pressure recording.
The contraction of the left ventricle imparts its contractile energy on the blood mass it contains, developing and raising the pressure to overcome the diastolic pressure in the aorta in order to open the aortic valve and eject the blood into the aorta. As the ventricle ejects the blood mass into the aorta with each systole, it creates a pulsatile pressure as well as a pulsatile flow. By appropriate recording techniques applied in and/or over an artery, one can show the pulsatile nature of the pressure wave, the pulsatile nature of the flow wave, as well as the dimensional changes in the artery as the pressure wave travels (36).

What is actually felt when an artery is palpated by the finger is not only the force exerted by the amplitude of the pressure wave, but also the change in the diameter. For instance, the pressure pulse of both arteriosclerosis and hypertension in the elderly and that caused by significant aortic regurgitation will look similar when recorded. It will show a rapid rise in systole and a steep fall in diastole with an increased pulse pressure (the difference between the systolic and the diastolic pressure). However, the arterial pulse in these two different situations will feel different to the palpating fingers. The difference is essentially in the diameter change. The pulse of aortic regurgitation is associated with a significant change in diameter, whereas this is usually not the case in arteriosclerosis. The diameter change due to the high volume of the pulse in aortic regurgitation can be further exaggerated by elevating the arm, which helps to reduce the diastolic pressure in the brachial and the radial artery.

Since pressure and radius are two important factors that affect wall tension, as shown by Laplace relationship, it is probably reasonable to consider both of them together. What is actually felt when the arterial pulse is palpated can therefore be restated as the effect caused by a change in the wall tension of the artery.

Laplace’s law is expressed as follows:

\[
\text{Tension} = P \times r
\]

for a thin-walled cylindrical shell. If the wall has a thickness, then the circumferential wall stress is given by Lamé’s equation, as follows:

\[
\text{Tension} \propto P \times r / 2h
\]

Amplitude of the pulse will depend not only on the amplitude of the pressure wave, but also on the change in dimensions between diastole and systole (or simply the amount of change in wall tension).

**The Volume Effect**

According to Laplace’s law, the volume has a direct effect on the wall tension in that it relates to the radius. The actual volume of blood received by each segment of the artery and its effect on the change in wall tension of that segment depends also on the vessel involved. The proximal elastic vessels (aorta and its main branches) receive almost all of the stroke volume of the left ventricle. The elastic nature of these vessels allows greater displacement and change in their radius. However, as one goes more peripherally, total cross-sectional area increases. Therefore, each vessel receives only a fraction of the stroke volume. In addition, the vessels are more muscular and less distensible. For similar rise in pressure, the change in vessel diameter is less. The corollary of this is that to achieve similar diameter change in the peripheral vessels, the pressure developed must be higher.
Pressure in the Vessel

The pressure pulse generated by the contraction of the left ventricle is transmitted to the most peripheral artery almost immediately, and yet the blood that leaves the left ventricle takes several cardiac cycles to reach the same distance. Thus, it must be emphasized that pressure pulse wave transmission is different and not to be confused with actual blood flow transmission in the artery. The analogy that can be given is the transmission of the jolt produced by an engine of the train to a series of coaches while shunting the coaches on the track as opposed to the actual movement of the respective coaches produced by the push given by the engine. This is the classic analogy given by Bramwell (6).

The mechanics of flow dictate that it is the pressure gradient, not the pressure, that causes the flow in the arteries. There is very little drop in the mean pressure in the large arteries. Almost all of the resistance to flow is found in the precapillary arterioles. This is where most of the drop in mean pressure also occurs in the arterial system (11,12,35). The shape of the pressure pulse changes as it propagates through to the periphery. Although the mean pressure decreases slightly, the pulse pressure (systolic pressure minus the diastolic pressure) increases distally so that the peak pressure actually increases as the wave propagates (11,37). The higher peak systolic pressure achieved in the less distensible and more muscular peripheral vessels helps to accommodate the volume received by the distal vessels.

Reflection

Experimental studies have clearly shown that pressure pulse wave generated artificially by a pump connected to a system of fluid-filled closed tubes or branching tubes with changing calibre gets reflected. The reflective sites appear to be branching points (11,12). This implies that the incident pressure pulse (not flow) produced by the contracting left ventricle gets reflected back. It is reflection of the pressure pulse that gives the pulse wave its characteristic contour (Fig. 2). The pressure and the velocity waveforms vary markedly at different sites in the arteries. The peak velocity generally occurs before the peak in pressure at all sites (17). As one moves to the periphery, the pulsatile pressure fluctuations increase while the oscillations of flow diminish as a result of damping. The peripheral pressure fluctuations often become amplified to the extent of exceeding the central aortic systolic pressure. This is further evidence that the pressure waves get reflected peripherally (17,37).
Since the pressure pulse normally travels very fast (m/s), the recorded arterial pressure wave at any site in the arterial system is usually the result of the combination of the incident pressure wave produced by the contracting left ventricle and the reflected wave from the periphery (37,38).

### Pulse Wave Contour

When one records the arterial pulse wave with a transducer, one may be able to identify three distinct components in its contour:

- The **percussion wave**, which is the initial systolic portion of the pressure pulse
- The **tidal wave**, which is the later systolic portion of the pressure pulse
- The **dicrotic wave**, which is the wave following the dicrotic notch (roughly corresponding to the timing of the second heart sound) and therefore diastolic.

### Factors That Affect the Magnitude of the Initial Systolic Wave

Although this portion of the arterial pulse may also be influenced and modified by reflected waves from the periphery, the rate of rise and the amplitude of the incident pressure wave of the arterial pulse is still dependent on the ejection of blood into the aorta by the contracting left ventricle. Thus, the characteristics of the proximal arterial system and the effect of the left ventricular pump become pertinent (Table 1; Fig. 3).
Ejection of blood into the aorta by the contracting left ventricle during systole leads to a rise in aortic pressure from the diastolic level at the time of the aortic valve opening to the peak in systole. The rise in aortic pressure from its diastolic to the systolic peak is determined by the compliance of the aorta as well as the stroke volume. The aorta is very compliant because of its greater content of elastin compared to the smooth muscle and the collagen in its walls. Because the walls of the aorta are compliant, it expands to accom-
modulate the blood volume. The increase in pressure for any given stroke volume will be
determined by the compliance of the aorta. Increasing age leads to changes in the struc-
tural components of the walls of the aorta and reduced compliance (39). When the aorta
is rigid and stiff, the pressure will rise steeply to an increased peak systolic pressure,
giving rise to an increased pulse pressure.

In some elderly patients the decreased compliance of the proximal vessels could be
severe enough to hide the slowly rising percussion wave of the aortic stenosis due to
marked increase in pressure despite small increase in volume (36). In addition, the
pressure rise will be steeper and faster if the stroke volume is delivered to the aorta with
a faster rate of ejection, as would be expected with increased contractility.

**The Left Ventricular Pump**

The left ventricular output is dependent on the filling pressures (preload), the intrinsic
myocardial function, and the afterload against which it pumps (determined by the vas-
cular properties of the arterial system, which affect its compliance, the peripheral resis-
tance, and the peak systolic pressure). When the ventricle begins to contract at the end
of diastole, the intraventricular pressure rises as more and more myocardial fibers begin
to shorten. When the left ventricular pressure exceeds the left atrial pressure, closing the
mitral valve, the isovolumic phase of contraction begins. During this phase the rate of
pressure development is rapid. The rate of change of pressure \(\frac{dP}{dt}\) during this phase
usually is reflective of the contractile state of the left ventricle. It is increased when the
left ventricle is hypercontractile and is usually depressed when the ventricular function
is diminished. When the left ventricular pressure exceeds the aortic diastolic pressure,
the aortic valve opens and the ejection phase of systole begins. Recordings of pressures
in the left ventricle and the aorta obtained by special microtip sensors show that the left
ventricular pressure exceeds that of the aorta in the early part of systole (40–42). This
pressure gradient is termed the *impulse gradient* because it is generated by the ventricu-
lar contraction. The aortic flow velocity reaches a peak very soon after the onset of
systole. During the latter half of systole, the rate of myocardial fiber shortening slows
and the left ventricular pressure begins to fall. When the left ventricular pressure falls
below that in the aorta, the gradient reverses, and this is associated with the deceleration
of aortic outflow. The initial rapid rise in the aortic outflow velocity to its peak is caused
by early and abrupt acceleration of blood flow out of the left ventricle. This acceleration
is achieved as a result of the force generated by the ventricular contraction. The peak
aortic flow velocity achieved is dependent on the magnitude of the force \(F\) multiplied
by the time \((t)\) during which it acts. The physical term “Impulse” describes \(F \times t\).

The kinetic energy imparted by the ventricular contraction to the blood mass it ejects
can be viewed as the total momentum gained by the blood mass. The force of contraction
and the peak rate of pressure development \(\frac{dP}{dt}\), by determining the rate of accelerati-
on of flow, should influence the rate of rise of the incident pressure. Both the mass \((m)\)
of blood ejected, namely the stroke volume, and the velocity \((v)\) of ejection would be
expected to affect the amplitude of the incident pressure wave. The stroke volume and
the velocity of ejection together represent the *momentum of ejection* \((mv)\). The effect of
the maximum momentum achieved during ejection as well as the rate of change in
momentum on the rate of rise and the amplitude of the pressure pulse can be explained
by an analogy. This is best understood by observing a strength-testing game in carnivals
where a person hits a platform on the ground with a large wooden hammer, displacing
Momentum of Ejection

**Mass (Stroke Volume)**

In conditions with large stroke output, momentum of ejection will be augmented, causing increased amplitude of the pressure wave. The stroke volume will be increased in hyperdynamic and hypervolemic states such as anemia, hyperthyroidism, Paget’s disease, and pregnancy. Large stroke volumes can also occur in the absence of the above in certain cardiovascular conditions such as aortic regurgitation and persistent ductus arteriosus (Fig. 4). In addition, bradycardia as seen with chronic complete atrioventricular (A-V) block by virtue of increased diastolic filling of the ventricle can also produce increased stroke volume.

Diminished stroke volume will therefore be expected to cause decreased momentum of ejection and decreased amplitude of the pressure pulse. This will occur in conditions with significant obstruction to either left ventricular outflow or inflow. Severe aortic stenosis, often with some left ventricular dysfunction, and severe mitral stenosis are examples of such states. Cardiac failure with poor pump function will also result in severe reduction in stroke volume. Severe reduction in filling of the left ventricle as seen in patients with cardiac tamponade will have a similar effect on the stroke volume and therefore on the momentum of ejection.

**Velocity of Ejection**

The velocity of ejection will be determined by the strength with which the left ventricle contracts as well as the by the impedance to ventricular ejection.

**Contractility**

Increased velocity of ejection, in the absence of outflow obstruction, will likely lead to larger amplitude of the pressure pulse in the aorta since more volume will be delivered over a shorter duration. The rapid velocity also implies a stronger contractile force.
and increased $dP/dt$ and therefore will be expected to cause a faster rate of rise of the pressure pulse or rapid upstroke of the pulse.

In the absence of obstruction to the left ventricular outflow, the velocity of ejection is mainly determined by the pump function of the left ventricle as well as the preload and the afterload. In conditions with large stroke volume due to Starling effect, the ejection velocity would also be increased unless significant left ventricular dysfunction coexists.

In mitral regurgitation there is increased contractility as a result of the Starling effect of the increased filling of the left ventricle (from both the normal pulmonary venous return and the volume of blood that went backwards into the left atrium due to the regurgitation), resulting in faster ejection. However, there is no increased stroke volume received by the aorta, because the left ventricle has two outlets during systole, namely the aorta and the left atrium. The amplitude of the pressure pulse is expected to be normal, but the rate of rise may be rapid (43).

In hypertrophic cardiomyopathy, the ventricle is hypercontractile and ejects the blood very fast. This leads to a very rapid rise of the arterial pulse. In addition, in this condition the pattern of ejection is such that the flow into the aorta often may be biphasic with a smaller secondary peak in late systole, which may partly contribute to a secondary late systolic pressure rise in the aorta (44). This is more pronounced in patients who have a dynamic obstruction to the outflow when during the middle of systole the anterior leaflet of the mitral valve is pulled from its closed position and moves anteriorly towards the interventricular septum (systolic anterior motion [SAM]). While there is still controversy as to the cause of the SAM (45), it has been attributed to a Venturi effect caused by the rapid outflow velocity. This septal mitral contact obstructs ejection, and the aortic flow ceases. In late systole when the intraventricular pressure begins to fall, the anterior mitral leaflet moves away from the septum, the obstruction subsides, and the ejection resumes. This will then give rise to a second peak in the pulse in late systole, which will be smaller than the first peak.

In aortic stenosis, although the ejection velocity is significantly increased due to the obstruction, the accompanying volume increment is low. Therefore the increase in radius and the tension in the aortic wall will be expected to be slow. In addition, the increased velocity of the aortic jet through the stenotic valve, by a Venturi effect, will cause a decrease in the lateral pressure (26,46), thereby contributing to a slower rate of pressure rise in the aorta, giving rise to a delayed upstroke and a shoulder in the ascending limb of the pressure wave called the anacrotic shoulder (Fig. 5).

**Impedance to Ejection**

In addition to the factors affecting the afterload, impedance to ejection includes the vascular properties of the arterial system and the peripheral resistance. The load that the ventricle needs to handle during contraction is usually called the afterload, sometimes considered to be synonymous with intraventricular systolic pressure. In fact, afterload is more closely related to left ventricular wall tension or stress. According to Lamé’s modification of the Laplace relationship, the tension is directly proportional to the pressure and the radius and is inversely related to the thickness. Thus, when the left ventricle faces a chronic pressure load (e.g., hypertension or aortic stenosis) or volume load (e.g., mitral or aortic regurgitation), eventually its walls will undergo hypertrophy. This will then tend to normalize or reduce the wall stress or tension. Since wall tension is an important determinant of myocardial oxygen consumption, increased afterload would be disadvantageous to ventricular myocardial fiber shortening.
The vascular properties of the arterial system, especially the aorta, are also important because they determine the yield or the compliance. The peripheral resistance is a result of the combined resistance to flow of all the vessels in the arterial system. This is predominantly determined by the precapillary arteriolar tone (commonly referred to as the resistance vessels). The level of sympathetic activation generally determines this vasomotor tone. In addition, the arteries in general will offer variable resistance to flow depending on the bulk of the smooth muscle, the relative content of collagen and elastin in their walls, as well as the tone of the smooth muscle in the media. The tone of the smooth muscle is in turn locally mediated by the endothelial function. It is well known that the normal endothelium produces nitric oxide, which causes smooth muscle relaxation. The basal diastolic tension in the arteries is related to the tone of the vessels and the peripheral run-off. The change in wall tension caused by the pressure wave amplitude as felt by palpation is to a certain extent dependent on the basal diastolic tension in the vessel before ejection.

**Low Peripheral Resistance**

Low peripheral resistance may result from:

1. Peripheral vasodilatation resulting from withdrawal of the sympathetic tone as seen in patients with aortic regurgitation (large stroke volume turning off the renin–angiotensin system leading to precapillary vasodilatation), secondary to certain drugs and septic or anaphylactic shocks.

2. Development of arteriovenous communications, congenital (arteriovenous malformations), iatrogenic (arteriovenous fistulae), or resulting from pathological processes in various systemic organs (cirrhosis of the liver, chronic renal disease, chronic pulmonary disease, Paget’s disease, and beriberi).

Conditions with low peripheral resistance and vasodilatation will lead to increased amplitude of the pulse because the diastolic pressure and the diastolic tension in the
vessels in these states is low and therefore the effect of change in tension is better appreciated. Conditions that are associated with increased stroke output (mass augmenting momentum of ejection) with low peripheral resistance (e.g., persistent ductus or arteriovenous communications, aortic regurgitation) cause increased velocity of ejection. Both of these factors lead to a large amount of change in tension leading to greater amplitude of the pulses (bounding pulses).

**High Peripheral Resistance**

High peripheral resistance is usually caused by high sympathetic tone causing constriction of the precapillary arterioles. The effect of the vasoconstriction is also present on the other vessels. This will lead to decreased compliance of the proximal vessels, making them less distensible as well as causing increased basal diastolic tension in the arteries. Therefore, the change in tension during systole is less. The increased resistance to ejection will decrease ejection velocity and, to some degree, the volume.

In severe hypertension, the basal tension being high in diastole, the change in tension during systole may be poorly felt since much of the change in tension is due to pressure rise alone. It is not uncommon to find constricted and poorly felt peripheral arterial pulses in the context of significantly elevated intra-arterial pressures in some patients receiving inotropic and vasoactive agents. However, in some very elderly patients with stiffened arteries resulting from arteriosclerosis (medial degeneration and sclerosis of the media), the arterial pressure rise might be quite steep and large with large-amplitude pulses due to very marked increase in the pulse pressure (15,35,37,38). The diastolic pressure and tension in these elderly patients’ vessels are not usually increased.

**Transmission or Velocity of Propagation of the Pulse Wave**

As mentioned earlier, the velocity of pulse transmission is generally very rapid. The velocity of pulse transmission is not to be confused with the velocity of ejection or blood flow. The latter can be easily measured by Doppler and is only about 0.3 m/s at the radial, whereas at the level of the femoral, the pulse wave transmission velocity is almost close to 10 m/s. It gets faster as one moves more peripherally, because the peripheral vessels are more muscular and therefore stiffer. The pulse wave velocity is normally faster in the elderly due to the stiffness of the vessels (4). Vasoconstriction and the increased tone of the vessel walls also make the arterial system stiffer and allow faster propagation. The elasticity of the arterial segments is also influenced by the distending pressure (39). As distending pressure increases, the vessel becomes more tense. This is due to greater recruitment of the inelastic collagen fibers and consequently a reduction in elasticity and conversely an increase in stiffness (27,48). The mean arterial pressure determines the background level of distending pressure in the arterial system. Thus, when the mean arterial pressure rises, the arteries become more tense and allow the pulse wave to travel faster (39). The pressure pulse wave travels slowly when the mean arterial pressure is lower (36,37,39).

The concept can also be demonstrated by plucking the middle of a string that is held steady and firm at either end. The oscillations produced by the transmission of the wave and its reflection will be grossly visible when the string is held loose. When it is held tight, the transmission is fast and the oscillations become a blur, and when the frequencies reach audible range, one can hear a tone as well. The tightness with which the string is held is analogous to the stiffness of the vessels in the arterial system.
In severe left ventricular failure with very low cardiac output and stroke volume, associated with decreased rate of pressure development, the momentum of ejection and the rate of change in momentum may be poor and lead to a low amplitude arterial pulse, and if the mean arterial pressure is low, this will tend to slow pulse wave velocity. In aortic stenosis, the velocity of ejection is increased, but the rate of change in momentum is very slow because of less mass or volume being ejected per unit time. In addition, the increased velocity of ejection caused by the pressure gradient across the aortic valve gives rise to a Venturi effect, causing a decrease in lateral pressure in the aorta (26,46). This results in a slowly rising low-volume pulse with lower mean arterial pressure, which may also be transmitted slowly to the periphery. In severe aortic stenosis, one may actually feel an appreciable delay between the upstroke of the brachial arterial pulse and that of the radial arterial pulse (brachioradial delay) in the same arm. This is a rare sign (49), which has also been observed by us in some patients with severe degree of aortic stenosis with low output. It is conceivable that the delay may also result from other factors, including more compliant arteries, which will also make the wave travel slower (50,51).

Reflection

Wave of propagation would eventually die out in a completely open system. The arterial system is far from being a completely open system. Although the total collective cross-sectional area increases peripherally, because of change in caliber and branching, reflection of the incident pressure wave occurs from these sites. Complete occlusion of a vessel or a branch will result in complete and fast reflection. Under normal conditions close to 80% of the incident wave is reflected from the periphery. The main reflection site for the proximal segment of the arterial system may be the aortoiliac bifurcation (22). For the more peripheral muscular portion of the arterial system, reflecting sites are at the level of the arterioles. Increase in peripheral resistance or vasoconstriction will increase the intensity of wave reflection (reflection coefficient) (37,38). On the other hand, the effect of lowering the peripheral resistance and vasodilatation will cause a decrease in wave reflection. This can be demonstrated pharmacologically by the intra-arterial injection of nitroglycerin or acetylcholine (25,52) as observed by the changes in the arterial pressure pulse waveform.

The arterial pulse waveform has been studied by breaking it down into its component harmonics much like a musical wave. The majority of the energy of the pulse is contained in its first five harmonics. Vascular impedance studies relating corresponding harmonic component of pressure and flow waves have allowed quantitative analysis and have given better insights into arterial pressure and flow mechanics (11,14,15).

The peripheral circulation has been considered to provide two discrete components of the reflecting sites: one representing the resultant of all reflecting sites from the upper body and the other representing the resultant of all reflecting sites from the trunk and the lower extremities. This has been described as an asymmetrical T tube in shape model (36,38,53).

The Effects of Wave Reflection on Pressure and Flow Waveforms

The effect of wave reflection is related to the time of arrival of the reflected wave during the cardiac cycle. The latter will depend not only on the pulse wave velocity, but also on the distance from the individual reflecting sites. Reflected waves from the upper
limbs arrive earlier at the ascending aorta compared to those that arrive from the lower body. Reflected waves from the most peripheral reflecting sites will arrive earlier at the larger peripheral arteries before they will arrive at the central aorta.

When the reflected wave is in the same direction, as the incident wave it will facilitate flow, whereas when it has an opposite direction to the incident wave it will diminish flow. Therefore the upper and lower body reflections will have different effects on the ascending and the descending aortic flow. Reflected waves from the lower body arriving in the upper arm vessels show the effect of facilitation of forward flow, altering its contour from that seen in the ascending aorta.

Reflected pressure wave always adds to the pressure waveform.

The recorded arterial pressure waveform will depend on the incident wave, the intensity of wave reflection from the peripheral sites, and the timing of reflection during the cardiac cycle where the two meet and merge (36,37). Reflection added to the incident central aortic pressure wave contributes to the shape of the central aortic pressure. In general it tends to augment the central aortic pressure. In the peripheral more muscular arteries, such as at the level of the femoral or the radial artery, the reflection from the distal sites arrive in such a way as to fuse with the peak of the incident pressure wave, giving rise to an elevated systolic pressure and a larger pulse pressure compared to the central aortic pressure wave. Such amplification of the pressure pulse peripherally is more marked in the lower extremities than in the upper extremities. The peripheral amplification in the arterial pressure appears to be somewhat related to the frequency component. In fact, transfer of pressure wave to the periphery has been studied by relating the degree of amplification of the peripheral pressure compared to the aortic pressure to the individual harmonic frequency component of the pressure pulse. The amplification appears to be least in the lower frequencies, peaking at relatively higher frequencies (36,37). This must be considered in relation to the fact that most of the energy of the arterial pressure pulse is actually in the first five harmonics.

When the arteries are relatively compliant and the pulse wave velocity is relatively slow (as in young adults), reflected waves return to the central aorta in diastole augmenting diastolic pressure and therefore coronary blood flow, which occurs predominantly during diastole. In these normal young individuals, reflected wave from the periphery causes a secondary wave or hump in diastole (dicrotic wave) to the central aortic pressure (Fig. 6A). When arteries are stiffer and the pulse wave velocity is higher, as with increasing age, reflected waves arrive earlier and augment the central aortic systolic pressure, causing a second late systolic peak to the waveform (tidal wave) that is higher than the first peak (22) (Fig. 6B). It also adds to the duration of the pressure pulse. This would in effect increase the left ventricular workload and compromise the coronary perfusion (54,55).

The duration of ejection (ejection time) also plays an important role in how the central aortic pressure contour is modified by the reflected wave from the periphery (17,19, 35). When the ejection duration is increased, the reflected wave from the periphery will arrive at the central aorta during late systole and thus cause a secondary wave in late systole (Fig. 6B). When the ejection time is shortened, the reflected wave will arrive after the incisura on the aortic pressure curve (dicrotic notch on the carotid pulse), which corresponds to the aortic valve closure. This will obviously give rise to a diastolic wave (dicrotic wave) (Fig. 7A). The importance of the effect of ejection time or duration of ejection has been demonstrated in humans during the Valsalva maneuver. During this
maneuver, one tries to exhale forcefully against a closed glottis. During the strain phase, there is increased intrathoracic and intra-abdominal pressure. This will lead to a decrease in venous return, accompanied by decreased stroke volume and falling blood pressure, which causes reflex tachycardia secondary to sympathetic stimulation. All of these lead to a shortened duration of ejection. When the straining phase ends, there is a sudden surge of all the damped venous return from the splanchnic and the peripheral veins. This will in turn increase the stroke output, which will be ejected into an arterial system that is constricted by the marked sympathetic stimulation that occurred during the strain phase. Ejection of a larger stroke volume into constricted arterial system leads to a sudden rise in the arterial pressure (blood pressure overshoot). This in turn will cause reflex bradycardia resulting from baroreceptor stimulation. The ejection time will therefore be increased during the poststrain phase. The pattern with late systolic peak has been observed in the beats with longer duration of ejection and the pattern with the diastolic (dicrotic) wave when the duration of ejection is short and the arterial pressure is low (8,17,23,36,37) (Table 1).

### Intensity of Reflection

Intensity of reflection is related to the degree of arteriolar tone. Vasoconstriction will intensify reflection, and vasodilatation will abolish the same. Vasodilatation associated with exercise will be expected to cause less amplification of pressure in the active limb, whereas in the inactive limb the amplification may in fact be greater. The amplification of brachial pressures caused by leg exercise can be abolished by reactive hyperemia of the arm (56). Intra-arterial injection of nitroglycerin or acetylcholine in a single vascular bed can be shown to abolish secondary diastolic waves (usually caused by reflection) in the arterial pressure wave (25,37).
Fig. 7. (A) Carotid pulse in a patient with cardiac tamponade. Note the prominent dicrotic wave (D). If palpable, it could give rise to a bifid pulse. DN, dicrotic notch. Systolic time intervals QS2 (total duration of electromechanical systole), LVET (left ventricular ejection time), and PEP (pre-ejection period) are given in milliseconds. (B) Same patient as in Fig. 7A after pericardiocentesis. Note the change in the D. It is much smaller. The improvement in the stroke output following the relief of tamponade is evidenced by the increase in LVET.

In summary, multiple factors determine pressure pulse wave reflection and its effects, including vasomotor tone, vascular properties, mean arterial pressure, left ventricular ejection time, and the pattern of left ventricular ejection (37) (Table 1, Fig. 3).
Clinical Implications of Pressure Pulse Wave Reflection

The multiple factors that determine wave reflection and its effects are of clinical relevance in the assessment of the arterial pulse.

Wave reflection is the primary reason for the alteration of the incident pressure wave centrally in the aorta contributing to the change in its contour and duration, as stated earlier. If the reflected waves arrive in diastole and cause diastolic pressure rise, it helps in the coronary perfusion, whereas if it arrives in systole and augments the late systolic pressure, then it will add to the increased left ventricular workload, thereby increasing the systolic left ventricular wall tension, which is one of the major determinants of myocardial oxygen consumption (37,38).

If the left ventricular function is relatively good, then the contracting left ventricular pump, despite the increased demand of myocardial oxygen, may sustain the increased pressure load in late systole. However, in situations where the left ventricular systolic function is severely compromised, as in late stages of myocardial dysfunction of any etiology, the left ventricle will be unable to sustain the late systolic augmentation of the pressure. In fact, the reflected pressure wave will impede forward flow from the left ventricle, causing it to diminish its output. This will in turn abbreviate the left ventricular ejection time or duration. Such patients are usually in cardiac failure with poor left ventricular function and decreased stroke output. This will not be an issue in patients with cardiac failure purely on the basis of diastolic dysfunction where the congestive symptoms are related to decreased compliance and stiffness of the ventricular myocardium resulting in elevated ventricular diastolic filling pressures. They will have normal left ventricular ejection time (37).

When the initial (percussion) and the late systolic (tidal) portion of the arterial pressure pulse are well separated, one may feel a bifid or bisferiens pulse (e.g., bisferiens pulse of combined aortic stenosis and aortic regurgitation) (21,35,36). In this situation, aortic stenosis causes slower rate of change in momentum of ejection leading to slower rate of pressure rise, as discussed previously. However, when the bisferiens pulse is felt, usually the co-existing aortic regurgitation is usually significant. The high stroke volume accompanying the aortic regurgitation will cause a large-amplitude pressure pulse wave. The increased velocity of turbulent flow at peak systole due to the obstruction will cause a decrease in lateral pressure in the aorta due to the Venturi effect similar to that seen in isolated aortic stenosis (Fig. 8) leading to a drop in pressure rise during the middle of systole, thereby separating the initial from the late systolic peak (38,46). In this instance, however, the late systolic peak will be greater than the initial one. In aortic valve disease, the ejection duration is prolonged, and this may have some effect on the harmonic components of the arterial pressure pulse wave since the left ventricular pump ejects its stroke volume over a longer period of systole. The pressure pulse has been noted to have predominance of lower frequencies (19).

When the dicrotic (diastolic) wave becomes large and palpable, it may mimic the bisferiens pulse. This is usually produced under circumstances of low momentum of ejection (usually due to low stroke volume), e.g., severe left ventricular failure, cardiac tamponade, and cardiomyopathy (Fig. 7A and B). In these states there may be increased sympathetic stimulation and activation of the renin–angiotensin system. Sympathetic stimulation will cause vasoconstriction. This will be accompanied by increased intensity of reflection. The pulse wave velocity could be normal or low. The activation of the renin–angiotensin system, by making the vessels stiffer, will favor increased pulse wave velocity.
However, the poor left ventricular function and output will cause a poor momentum of ejection, resulting in a decreased rise in the mean arterial pressure. The low mean arterial pressure will make the pulse wave travel slower. The ejection time may be shortened due to the low stroke volume. Intense vasoconstriction will be associated with good intensity of reflection. Thus the reflected wave will arrive in the central aorta after the aortic valve closure, i.e., after the incisura in diastole.

The three conditions that govern reflection in severe cardiac failure are also present in low-output states such as shock and cardiac tamponade. They are:

1. The low arterial pressure, which favors slow wave travel
2. Intense vasoconstriction, which intensifies wave reflection
3. Shortened left ventricular ejection time, which makes the reflection arrive in diastole (dicrotic wave) after aortic valve closure

The importance of low stroke volume and shortened ejection duration has also been demonstrated by the fact that the dicrotic wave is exaggerated in beats following shorter diastoles, during the strain phase of the Valsalva maneuver (breathing against the closed glottis) and during amyl nitrite inhalation. Amyl nitrite is a rapidly acting arterial dilator. It is fairly quickly inactivated in the lungs, and the effect is primarily on the arterial system, which causes rapid onset of arterial dilatation with fall in blood pressure. The brisk sympathetic stimulation secondary to the hypotension produces reflex sinus tachycardia and decreased stroke output initially and decreased ejection time. The reflex sympathetic stimulation leads to vasoconstriction, which favors reflection. All three conditions are thus met to cause prominent dicrotic waves. The sympathetic stimulation also leads to venoconstriction, causing increased venous return. The increased venous return results in increased cardiac output (stroke volume × the heart rate /min) (Fig. 9).

Compliant arterial system (as in the younger patients, post-aortic valve replacement in patients with aortic regurgitation) with associated slow pulse wave velocity can cause the reflected wave to travel slowly enough to augment the diastolic portion of the arterial pressure wave, thereby causing a prominent dicrotic wave.
ARTERIAL PULSE CONTOUR IN HYPERTROPHIC CARDIOMYOPATHY

In hypertrophic cardiomyopathy, obstructive or nonobstructive, the left ventricular contractility is markedly increased. The pattern of ejection is such that the aortic outflow tends to be biphasic in systole unlike the normal monophasic outflow (44). This is particularly marked in patients with obstruction to the outflow caused by the sudden SAM of the anterior mitral leaflet towards the interventricular septum during the middle of systole presumably caused by the Venturi effect of the rapid outflow velocity. The latter mechanism has been questioned by some, and the SAM has been attributed to pushing or pulling of the mitral valve by the anatomical distortion aggravated by vigorous contraction in these patients with marked hypertrophy of the left ventricular walls associated with small cavity (45). In any event the flow ceases in mid-systole and resumes in late systole. The biphasic outflow therefore generates a bifid contour of the pressure wave in the aorta. The late systolic pressure may also be augmented by reflected wave from the periphery. The initial part of ejection being rapid and strong is associated with a faster and greater momentum of ejection resulting in a sharply rising and peaked initial systolic (percussion) wave, which has a larger amplitude than the late systolic (tidal) wave. (Figs. 10 and 11).

Fig. 9. Prominent dicrotic wave (D) following amyl nitrite inhalation in a patient with hypertrophic cardiomyopathy. Amyl nitrite causes rapid onset of arterial dilatation with fall in blood pressure. The brisk secondary sympathetic stimulation produces increased vasoconstriction and tachycardia with shortening of ejection time. Venoconstriction with increased venous return will eventually increase the cardiac output. While the cardiac output is increased, the stroke volume may actually fall due to the tachycardia. The exaggerated diastolic inflow in the presence of the hypertrophic cardiomyopathy brings out the third (S3) and the fourth (S4) heart sounds. Apexcardiogram (Apex) reflects these events with exaggerated rapid filling wave and atrial kick, respectively.
Fig. 10. Simultaneous recordings of the ECG, the carotid pulse, the phonocardiogram, and the apexcardiogram from a patient with hypertrophic obstructive cardiomyopathy. Carotid pulse shows a rapid rate of rise of the percussion wave.

Fig. 11. Bisferiens carotid pulse in a patient with hypertrophic obstructive cardiomyopathy. The initial systolic or percussion (P) and the late systolic tidal (T) waves can be felt separately. The initial systolic wave is more prominent than the late systolic tidal wave.
ASSESSMENT OF THE ARTERIAL PULSE

The essential elements of examination of the arterial pulse should be geared towards the assessment of the volume, the upstroke, and the pulse contour abnormalities. In addition to these essential points, the observer would also be able to assess heart rate, rhythm, and arterial wall characteristics. The assessment of the arterial pulse will be discussed for the following points:

1. Rates, rhythm, and pulse deficit
2. Symmetry and radiofemoral delay
3. Vessel wall characteristics
4. Amplitude
5. Upstroke
6. Contour abnormalities
7. Bruits
8. Pulsus alternans
9. Peripheral signs of aortic regurgitation

Rate, Rhythm, and Pulse Deficit

The heart rate per minute can be quickly ascertained in most instances by counting the peripheral arterial pulse from any site for at least 15 s. When the pulse rhythm is irregular, the presence of an arrhythmia is suggested. In such instances the pulse rate may not reflect correctly the heart rate. Simultaneous cardiac auscultation and palpation of the peripheral arterial pulse will reveal a faster rate at the apex of the heart than suggested by the peripheral pulse. This difference is termed the pulse deficit. Simultaneous auscultation may also assist in determining the possible cause of the irregularity. A pause in the peripheral pulse due to an extrasystole or premature beat can be correctly distinguished from that caused by a dropped beat, such as seen in second-degree sinoatrial or A-V block. An exaggerated pulse deficit is most often present in atrial fibrillation, especially when the ventricular rate is not well controlled. The R-R intervals in atrial fibrillation are usually variable. This leads to varying lengths of diastole. The ventricular filling following short diastole is poor, leading to low stroke volume and ejection velocity, resulting in poor ejection momentum and poor pulse, which cannot be felt. Similar effects can also be caused by premature beats.

Symmetry and Radio-Femoral Delay

All peripheral pulses should be palpated, including the temporal arteries in the head, the carotids in the neck, the brachial, the radial, and the ulnar in the upper extremities, the abdominal aorta, the femoral, the popliteal, the posterior tibial, and the dorsalis pedis in the lower extremities. The presence of the pulse at all these sites must be ascertained. Comparison of the similar pulse at opposite sides of the body should be made. An absence of the pulse at any site and the presence of significant discrepancy between the two sides usually indicates a proximal blockage in the vessel with the absent or weaker pulse.

The temporal artery is usually felt for the presence of tenderness commonly seen in temporal arteritis. The carotid pulse, being most central and closer to the aorta, should be preferentially used for the assessment of the pulse volume, upstroke, as well as detection of contour abnormalities. It may be occasionally anatomically difficult to palpate in some patients with short and thick necks. It is usually located somewhat medially to the sternomastoid muscles.
The brachial pulse is located medially in the antecubital fossa. The brachial arterial pulse is commonly used for measurement of blood pressure in the arms. It should also be felt for the proper placement of the stethoscope for blood pressure measurement. The radial and the ulnar pulses are felt laterally and medially, respectively, on the anterior aspect of the wrist. One of these could be congenitally absent. Both of these arteries are usually connected in the hand through the anastomotic arches. In some patients these connections may be inadequate. Radial artery often is used for intra-arterial blood pressure monitoring in critically ill patients, and such instrumentation may lead to occlusion of the vessel. This is usually tolerated by most because of the anastomotic connections in the hand, which continue to be perfused through the ulnar artery. In patients with poor anastomotic connections such iatrogenic blockage may lead to gangrene of the hand. The adequacy of the anastomosis in the hand must be determined prior to any such instrumentation. This is usually determined by the radial compression test (Allen’s test). Both the radial and the ulnar arteries should be blocked by direct compression of the vessels against the underlying wrist bones. The patient is asked to make a tight fist, thus emptying the hand of the venous blood. This should leave the hand pale until the radial artery is released from the compression. This should result in immediate hyperemia of the hand, resulting in the disappearance of the pallor and return of the normal pink appearance. The test should be repeated a second time with release of the ulnar artery. Similar result should be obtained to indicate intact anastomosis.

The abdominal aorta may or may not be palpable in the adult depending on the degree of obesity. In obese individuals mere palpability may be an indication of the presence of an aneurysm. Palpability of a wide area of pulsation over the region of the abdominal aorta, particularly when the pulsation is expansile, would indicate the presence of an abdominal aortic aneurysm. Expansile pulsation can be easily checked by palpating with two index fingers of each hand placed on either side of the pulsating aorta approximately 1–2 in apart. If the fingers are further separated by each pulsation, as opposed to being lifted up without separation, the pulsation can be considered expansile.

The femoral arteries should be palpated in each groin below the inguinal ligament. Diminished or absent femoral pulses indicating proximal blockage is often seen in peripheral vascular disease. Normally the femoral and the radial pulses occur simultaneously. When the femoral pulse lags behind the radial (radio-femoral delay), occlusion of the aorta either due to coarctation or atherosclerosis is indicated.

The differential effects of the anatomical variations in coarctation of aorta may be diagnosable at the bedside by the careful comparison of the brachial pulses between the two arms. If both the brachial pulses and the carotids are strong with delayed or diminished femoral pulses, it will indicate the coarctation to be distal to the left subclavian artery. However, when the left brachial arterial pulse is weak or diminished compared to the right, it will indicate the coarctation to be proximal to the left subclavian artery. If the right subclavian has an anomalous origin from the aorta distal to the coarctation, then the right brachial pulse will be diminished or poor.

Popliteal pulses are felt by applying pressure over the popliteal fossa with both hands encircling the knee with the thumbs on the patella and the fingertips held over the popliteal fossa with the knees very slightly bent to relax the muscles. The normal popliteal pulse is often difficult to feel, especially in heavy patients. Popliteal pulses may be more easily felt in patients with significant aortic regurgitation and in other causes of wide pulse pressure.
The dorsalis pedis and the posterior tibial can have marked anatomical variations, resulting in the absence occasionally of one or the other without proximal occlusive disease. Other signs of occlusive disease causing absence of these pulses in the feet should be looked for (temperature of the foot, color, skin perfusion assessment by blanching, presence or absence of hair on the dorsum of the toes).

**Vessel Wall Characteristics**

The readily accessible arteries such as the brachials and the radials can be rolled between the finger and the bone to get a feel for the thickness and the stiffness of the walls. A calcified vessel will feel hard and not easily compressible. Vessels affected by medial sclerosis would feel thicker and stiffer and less pliable.

**Amplitude**

The amplitude of the pulse is assessed by determining the displacement felt by the palpating fingers. The displacement is dependent on the change in tension between diastole and systole developed in the arterial wall palpated. The tension is increased according to Laplace’s law as the radius and the pressure increase. The radius is increased with large stroke volumes. Patients with low output and low stroke volume will have a “thready” or weak pulse as a result of poor displacement and reduced level of tension developed. Such a low-amplitude pulse (low-volume pulse) is known as pulsus parvus. The peak systolic arterial pressure also contributes to the tension developed on the vessel wall. Thus the presence of a marked increase in pulse pressure could also result in increased amplitude of the pulse.

The amplitude of the arterial pulse should be assessed in general using the carotid pulse. Carotid pulse amplitude may be low in the presence of significant obstructive lesions such as severe aortic and mitral stenoses. Large stroke volumes, as seen in aortic regurgitation, would result in exaggerated pulse displacement of the carotids, which may be visible from a distance (Corrigan’s pulse). The large-amplitude pulsation may be felt in more peripheral vessels as well and reflects the increased change in wall tension secondary to increased stroke volumes. The low basal tension due to the low diastolic pressure, low peripheral resistance, and vasodilatation is also associated with an increase in the pulse pressure (Figs. 4 and 8).

In the presence of a large stroke volume associated with low diastolic pressure as in aortic regurgitation or its mimickers (aortic sinus rupture or communication with another low-pressure cardiac chamber such as the right atrium, aorto-pulmonary window, persistent ductus arteriosus), the arterial pulse feels strong and bounding with large volume of expansion. Therefore the amplitude is often casually referred to as the volume of the pulse. In patients with significant hypertension, in the presence of severe vasoconstriction, the peripheral pulses may even be difficult to palpate due to the decreased change in radius and wall tension.

In patients with combined aortic stenosis and regurgitation where both are significant, the amplitude of the carotid pulsation often would reflect the aortic stenosis, whereas the aortic regurgitation will show its effects on the amplitude more peripherally, such as in the popliteal arteries. The amplified systolic pressures due to reflection in these more muscular and peripheral vessels together with the low diastolic pressure secondary to vasodilatation and retrograde flow into the left ventricle would cause a wide change in tension between diastole and systole, causing more prominent pulsation.
Upstroke

The arterial pulse upstroke is best judged at the carotid. This is a palpatory assessment of the rate of rise of the pulse from its onset to the peak. The normal rate of rise of the carotid arterial pulse is usually sharp and rapid and indicates unobstructed ejection by a healthy ventricle. This in essence rules out a significant fixed type of aortic stenosis. The normally rising carotid pulse transmits a sharp tapping sensation to the palpatting finger. When the upstroke is delayed due to significant aortic stenosis, the rise is slow and gives a more sustained, gentle type of pushing sensation reflecting the gradual rise. In fact the sensation in some patients may be jagged and simulates a “thrill” or “shudder.” In normals, most of the stroke volume is ejected during the first third of systole, causing a rapid rise in the aortic pressure giving rise to a rapid upstroke. In aortic stenosis, this rapid ejection cannot occur. In fact, it takes all of systole to eject the same volume. The decreased mass or volume ejected per unit time leads to a considerable decrease in ejection momentum despite increased velocity of ejection. In addition, the increased velocity of flow caused by the significant pressure gradient between the left ventricle and the aorta caused by the stenosis produces a Venturi effect on the lateral walls of the aorta. This has the effect of significantly reducing the net pressure rise in the aorta. Thus the rate of rise of the arterial pressure pulse is slow in aortic stenosis. The net effects on the arterial pulse in valvular aortic stenosis are diminished amplitude (small), slow ascending limb (parvus), and a late and poorly defined peak (tardus). When the stenosis is very severe and accompanied by failing ventricle, the upstroke and the pulse may be poorly felt, if felt at all (pulsus tardus et parvus, meaning late, slow, and small). When the carotid pulse amplitude is judged to be very low, one will have extreme difficulty in assessing the rate of rise accurately.

The decreased momentum of ejection, particularly in severe cases with decreased stroke volume, will result in a low mean arterial pressure. This may be accompanied by slow pulse wave velocity, which in rare instances may actually be felt as an appreciable delay between the brachial and the radial artery pulses (brachio-radial delay). In the normals, simultaneous palpation of the brachial and the radial arterial pulse of the same side will not show any delay at the onset of the pulses over the two vessels. In fact an appreciable delay between the two pulses would indicate a slow transmission of the pulse wave. This is a rare sign, which may be present in severe aortic stenosis (49).

In aortic stenosis, the peripheral amplification of pressures is diminished but may be still present. This must be taken into account in assessing the gradient of pressure between the left ventricle and a peripheral artery, especially in children.

In elderly patients with stiffened arteries, even significant aortic stenosis may fail to be detected by the assessment of the upstroke. When compliance of the aorta is decreased, as seen in the elderly, even the small volume that is ejected in the early systole will cause a significant rise in pressure, thus increasing the tension in the carotids quickly enough to hide the expected effect of the stenosis on the pulse.

In some elderly patients with stiff vessels with rapid pulse wave velocity and reflection, there may be a hump or shoulder felt in the upstroke (anacrotic shoulder). This may mimic a delayed upstroke. These patients may be falsely diagnosed as having aortic stenosis, especially when the findings are associated with aortic sclerosis and ejection murmurs. Therefore, in the elderly, interpretation of carotid pulse upstroke should take these factors into account because of rapid transmission of the incident and the reflected pulse waves.
Very rapid or “brisk” upstroke of the carotid pulse when felt in association with a large-amplitude pulse suggests a hyperdynamic state such as due to aortic regurgitation or aortic regurgitation mimickers (aortic sinus rupture or communication with another low-pressure cardiac chamber such as the right atrium, aorto-pulmonary window, persistent ductus arteriosus). When the upstroke is brisk and the pulse amplitude is normal or low, hypertrophic cardiomyopathy with or without subvalvular muscular dynamic mid systolic obstruction must be considered (Fig. 10). In severe mitral regurgitation, the upstroke may tend to be brisk due to a Starling effect on the left ventricle secondary to the volume overload. However, the amplitude will tend to be normal (43).

In supravalvular aortic stenosis, the right brachial pulse and the carotid may be stronger than the left brachial. This will be reflected in a greater pulse pressure in the right arm than in the left. It has been attributed to a Coanda effect (the tendency of a jet of fluid, when properly directed, to attach to a convex surface instead of moving in a straight line) (43). The obstruction in the supravalvular aortic stenosis is such that the high-velocity jet is directed towards the right innominate artery and gets carried by this Coanda effect. It probably means that the direct impact pressure at the center of the jet is received by the right innominate artery. There may be actually a Venturi effect of lowered lateral pressure in the aorta distal to the stenosis transmitted to the left subclavian and the left brachial artery.

**Contour Abnormalities**

The normal arterial pulse has a single impulse with each cardiac systole. Occasionally in certain abnormal states, the pulse may be felt as a double impulse. This abnormality of the contour of the impulse is termed “pulsus bisferiens” or simply bifid pulse. Bifid pulse contours may be felt and recorded in arterial pulse tracings.

The conditions that may exhibit bifid pulse contours are (1) hypertrophic cardiomyopathy with obstruction (Fig. 11) and (2) severe aortic regurgitation with some aortic stenosis (Fig. 8). The mechanisms are different in these two conditions and have been dealt with previously. The arterial pulse will tend to have an increased volume and amplitude in combined aortic stenosis and regurgitation compared to hypertrophic cardiomyopathy. The effect of aortic regurgitation on the amplitude will be particularly evident in the periphery, whereas the aortic stenosis effect may be more appreciable in the carotid. The bisferiens of aortic stenosis and regurgitation also is altered in peripheral arteries, where they may not be recognizable as such. It is always best detected in the carotids, and the bisferiens is usually such that the late peak is always higher than the initial peak. This is different in hypertrophic cardiomyopathy, where the initial peak is often brisk and higher that the late peak.

Dicrotic wave, when exaggerated, may become palpable and cause a bisferiens effect on the pulse. The difference lies in the appreciation of the fact that the second impulse is diastolic and will occur after the second heart sound, as judged by simultaneous auscultation and palpation. The exaggerated dicrotic wave is usually associated with low momentum of ejection due to low stroke volume as in severe heart failure, cardiomyopathy, and cardiac tamponade. In these instances, the pulse amplitude will be expected to be low. The mechanisms and factors favoring the development of prominent dicrotic waves have been discussed previously.

**Bruits**

Bruits are audible noises, often systolic, caused by turbulence in flow usually resulting from partial obstruction of the lumen of the arteries and, very occasionally, from high
flow. These are often heard over large arteries and detected by auscultation with a stethoscope. Routine assessment of the arterial pulse must therefore include auscultation over all large peripheral arteries such as the carotid, subclavians, vertebrals, abdominal aorta, the renals, the iliacs, and the femorals. Often a systolic murmur originating from the aortic valve and occasionally from the mitral valve may radiate to the carotids. This can be differentiated by noting the location of maximal loudness of the bruit murmur. The carotid bruit is maximally loud, as expected, over the carotids as opposed to a radiating murmur, which should be maximally loud over the precordium. In high-flow states, continuous bruit lasting throughout systole and diastole may be heard over the intercostal vessels. This could occur in situations that lead to development of collateral flow such as seen in patients with aortic coarctation. Peripheral arteriovenous shunts (congenital or acquired A-V fistulae) may also produce continuous bruits over the vessels involved.

**Pulsus Alternans**

When the pulse amplitude changes beat by beat, alternating between higher and lower pulse amplitude as a result of alternating stroke volume, the resulting pulse is termed *pulsus alternans*. The alternating weaker and stronger pulses can be both felt and recorded. In such patients when the intra-arterial pressures are monitored, similar changes in both systolic and pulse pressures are noted. It can be detected by palpation and can be confirmed by blood pressure recording. *Korotkoff sounds* will be heard to double in rate once the cuff pressure is lowered and the lower systolic peaks of the weaker beats are detected. Pulsus alternans, if detected clinically, usually indicates a severe degree of myocardial dysfunction and left ventricular failure.

Pulsus alternans is, however, a normal phenomenon in as much as it can be induced after a premature beat. The effect of alternans in normal subjects can only be demonstrated by measurements of the systolic time intervals (Fig. 12). Systolic time intervals consist of the following:

1. The duration of the total electromechanical systole is measured by the interval QS2 between the onset of the QRS in the ECG and the end of systole as depicted by the onset of the second heart sound (S2) on the simultaneously recorded phonocardiogram.
2. The left ventricular ejection time (LVET) is measured from the onset of the upstroke of the simultaneously recorded carotid artery pulse tracing to the dicrotic notch.
3. The third interval, which is derived from these two measurements, is the pre-ejection period (PEP). This is obtained by subtracting the LVET from the QS2.

The stronger beat has better stroke output and therefore has longer left ventricular ejection time. The increased contractility of the stronger beat is reflected in a shorter pre-ejection period. The weaker beat has the opposite, namely a longer pre-ejection period. The effect of the alternans after an extrasystole in normal subjects lasts for two beats. As left ventricular function deteriorates, the alternans effect is more pronounced and tends to persist for a longer period. When the left ventricular function is severely depressed, the alternans becomes more pronounced and may become clinically noticeable and palpable in the arterial pulse (Fig. 13). This may sometimes last for long periods of time, such as several hours or days. Pulsus alternans is often initiated by an extrasystole. Although the mechanism is not fully elucidated, it is perhaps related in some ways to the phenomenon of postextrasystolic potentiation (57,58).

It is well known that the beat following an extrasystole is associated with increased contractility and stroke volume. This *postextrasystolic potentiation* is partially related
Fig. 12. Simultaneous recordings of ECG, phonocardiogram, and carotid pulse tracings showing measurement of systolic time intervals. QS2, Total electromechanical systole; LVET, left ventricular ejection time; PEP, pre-ejection period. All intervals are in milliseconds.

Fig. 13. (A) Carotid pulse recording from a patient with cardiomyopathy showing pulsus alternans. (B) In the same patient, intra-aortic pressures showing alternating levels of increased and decreased systolic pressures and pulse pressures.
to the compensatory pause, which follows the extrasystole, providing increased time for
diastolic filling, which in turn through the Starling effect would help increase the con-
tractility and stroke volume. The long pause will also allow more time for peripheral
runoff and drop in diastolic pressure. This will in turn help the postpremature beat by
decreasing the afterload. However, the postextrasystolic potentiation can be demon-
strated even when there is no compensatory pause, thereby keeping both the filling and
the afterload constant by pacing studies. In this instance, the increased contractility is
probably due to increased levels of intracellular calcium availability for the actin-myos-
in interaction. The premature depolarization caused by an extrasystole is thought to
release more intracellular calcium from the sarcoplasmic reticulum (SR) before all the
calcium released during the previous beat could be taken back up by SR. The increased
calcium is therefore available for the postpremature beat, thereby increasing its contrac-
tile force by more actin–myosin interaction. The increased calcium uptake and release may actu-
ally fluctuate alternatingly from beat to beat reaching the steady state in the normals after
a couple of beats. However, in the severely diseased myocytes of patients with severe
cardiomyopathy or end-stage heart failure, the steady state may not be reached for
prolonged periods of time (59). This becomes manifest as alternating strong and weak
contractions of pulsus alternans.

Peripheral Signs of Aortic Regurgitation

The peripheral signs of aortic regurgitation are:

- Quincke’s sign
- Corrigan’s pulse
- Water-Hammer pulse
- Pistol-shot sounds
- Duroziez’s sign
- de Musset’s sign
- Hill’s sign

Most of these peripheral signs of aortic regurgitation (60) are related to the large
stroke volume, increased ejection velocity, and momentum together with decreased
peripheral resistance, and widened pulse pressure with low diastolic pressure secondary
to retrograde flow into the left ventricle and peripheral vasodilatation. Some of these
signs may therefore be present in aortic regurgitation mimickers (aortic sinus rupture or
communication with another low-pressure cardiac chamber such as the right atrium,
aorto-pulmonary window, persistent ductus arteriosus) as well as other conditions ful-
filling the same pathophysiological requirements (Paget’s disease, arterio-venous com-
munications, severe anemia).

Quincke’s Sign

This sign refers to the capillary pulsation as detected in the nailbed. This sign is
elicited by applying enough pressure at the tip of the fingernail to cause blanching of the
distal nail bed while shining the penlight through the pulp of the fingertip. The observer
should look for movement of the proximal edge of the blanched area. This sign is not
diagnostic of aortic regurgitation and can occur in many other states with increased pulse
pressure and may even be detected in normal young individuals.

Corrigan’s Pulse

This term refers to the visible large-amplitude carotid pulsation.
WATER-HAMMER PULSE

This term refers to a toy, which consists of a sealed tube of vacuum partially filled with water so that when it is turned upside down the water falls with a palpable slap. The peripheral arterial pulse, such as the radial, in patients with aortic regurgitation and other similar states as mentioned above, is usually peaked and with rapid rise, which is poorly sustained followed by a rapid fall. When the radial artery is palpated with the palm of the hand while the arm is held raised, which helps to lower the diastolic pressure further in the arm palpated, the sharp slapping quality may be exaggerated.

PISTOL-SHOT SOUNDS

The phenomenon responsible for water-hammer effect upon auscultation over large vessels such as the femorals produces loud slapping sounds, which mimic pistol shots. These are short loud sounds. The mechanism has not been clearly established, but it is thought to be due to shock waves generated when the flow velocities exceed pressure velocities locally (35). It is also conceivable that they may be associated with actual reflection at these sites.

DUROZIEZ’S SIGN

This is also termed the intermittent femoral double murmur of Duroziez (60). It is elicited by compressing the femoral artery by applying gradual pressure over the stethoscope, which is placed over the femoral artery. At a certain moment of pressure a double murmur will be heard. The second method is to listen over the femoral artery while applying pressure with the finger in succession first 2 cm upstream (meaning proximal to) and then 2 cm downstream (meaning distal to) of the stethoscope. The upstream pressure will produce a systolic murmur, and the downstream pressure will produce a diastolic murmur. The mechanism involved is the turbulent flow caused by the partial obstruction. The turbulence gives rise to bruit, and its presence in relation to the site of obstruction should suggest the direction of flow. The systemic bruit is easy to understand because flow is toward the periphery during systole; one would expect the bruit to be distal to site of obstruction. Because diastolic bruit is heard proximal to the obstruction, the turbulent flow must also be proximal to the obstruction, indicating retrograde flow. Retrograde flow in some major arteries, including the coronaries during diastole, has been documented by angiography in aortic regurgitation.

Duroziez’s sign may be falsely positive in other high-output states such as thyrotoxicosis, severe anemia, fever, persistent ductus and arteriovenous fistula. In high-output states, the diastolic component of the bruit may be due to forward flow. The specificity of the sign can be increased by eliciting the sign slightly differently by applying the partial compression of the femoral artery with the proximal or the distal edge of the stethoscope while listening for the loudest diastolic component. In retrograde flow states (such as aortic regurgitation and persistent ductus arteriosus), as opposed to high-output states, the diastolic components are louder when the distal edge of the stethoscope is pressed.

DEMusSET’S SIGN

This sign is best elicited by watching the patient’s upper body and head while seated at the edge of the examining table. The upper body and head will be seen to move back and forth rhythmically with each systole. This is the result of the exaggerated ballisto-
graphic effect of the large stroke volume and wide pulse pressure together with low peripheral resistance. Ballistocardiography, an old physiological method (61), involves recording of the reaction of the whole body to the action of ejection of blood into the aorta (Newton’s third law of motion). The recoil of the aorta itself may play a part in contributing to the movement. Special instruments and bed are required to detect these in normal subjects with normal stroke volume. However, it is so exaggerated in aortic regurgitation that it becomes visible and detectable clinically.

**Hill’s Sign**

This sign is elicited by measurement of the systolic blood pressure in the arm and the leg simultaneously or in very quick succession (3,62). It must be emphasized that the *Hill’s sign* refers to systolic blood pressure differential as obtained by indirect blood pressure measurements using the traditional cuff. The pressure is obtained in the usual way at the arm over the brachial artery. The pressure in the leg can be elicited over the popliteal artery or at the ankle by palpation of the posterior tibial artery. Intra-arterial pressure recordings at the femoral level are not the way to look for this difference accurately. The reason for this is that the femoral artery is probably not peripheral enough to show this exaggerated effect.

Normally the peripheral pressures are usually amplified due to the muscular nature of these vessels, allowing rapid transmission of the pulse wave in both directions resulting in summation of the reflected with the peak of the incident wave. This peripheral amplification is usually more pronounced in the leg than in the arm. In normal subjects this difference in peak pressure between the arm and the leg is in the order of 15–20 mmHg. This difference may be markedly exaggerated in patients with significant aortic regurgitation (Fig. 14).
A difference of 20–40 mmHg in peak pressure can easily be seen in other conditions with wide pulse pressures (e.g., thyrotoxicosis, anemia, fever, or Paget’s disease). A difference of between 40 and 60 mm Hg is associated with a moderate degree of aortic regurgitation, whereas an excess of 60 mmHg is usually indicative of moderately severe aortic regurgitation. Hill’s sign is therefore somewhat related to the degree of aortic regurgitation and useful in following the patients with aortic regurgitation assuming, of course, there is no significant peripheral arterial disease.

The blood pressure differential between the arm and the leg is probably multifactorial in origin even in normal subjects:
1. The reflecting sites in the lower extremities are probably more than in the arm.
2. The vessels of the lower extremities are probably more muscular.
3. It is known that the age-related change in the compliance of the arteries is less in the upper limb vessels than in the lower limb vessels (36,37).
4. The upper arm vessels tend to arise anatomically at approximately 90° angle from the aortic arch. The diameter of these vessels being smaller than the aorta, the relative rapid flow in the aortic arch may cause a Venturi effect of relative suction on these cephalo-brachial vessels. This may tend to reduce the net effect of peripheral amplification of pressures caused by reflection. This effect of suction can be demonstrated in the side arm of a tap by running water through it when the side arm is of a smaller diameter and at right angles to the direction of water flow.

   This concept derives support from the fact that when direct impact pressure gets transmitted preferentially to the orifice of the innominate artery, as it happens in supravalvular aortic stenosis, the pressure is actually higher in the right arm supplied by that vessel (43). Presumably here, the Venturi effect of reduction in lateral pressure does not apply since the direct impact pressure of the jet gets directed preferentially towards the orifice due to the anatomical nature of the stenosis.

5. In aortic regurgitation, the increased momentum of ejection will produce larger-amplitude incident pressure wave. The increased momentum of ejection as well as the increased duration of ejection may in fact alter the harmonic components of the wave. It has been shown that peripheral amplification is less with lower frequencies than with higher frequencies of the pressure pulse wave. It has been suggested, therefore, that peripheral amplification is generally less in aortic regurgitation (36,38). While these may be valid, it is known that in echo Doppler measurements of pure aortic regurgitation, the aortic outflow velocity is quite variable. Sometimes it can be quite high without the presence of any stenosis. In patients with aortic regurgitation and high velocities of flow in the aortic arch, one can expect exaggerated result from the Venturi effect. This may explain variations seen in the sensitivity of the Hill sign in patients with aortic regurgitation. In patients who have a positive Hill’s sign, it becomes useful in their long-term follow-up.

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**PRACTICAL POINTS IN CLINICAL ASSESSMENT OF THE ARTERIAL PULSE**

In the clinical assessment of the arterial pulse, it is worth remembering the following points:

1. The three features of the arterial pulse that should be diligently sought for are the amplitude, the upstroke, and the pulse contour abnormalities.
2. Other features include the determination of rate, rhythm, pulse deficit, symmetry, radio-femoral delay, bruits, and specifically looking for the peripheral signs of aortic regurgitation when indicated.
3. Palpation of the carotid arterial pulsation is necessary to determine the pulse upstroke and for the detection of abnormal contours. The amplitude may be judged by palpation of the carotid as well as the more peripheral vessels.

4. The amplitude of the pulse is assessed by determining the displacement felt by the palpating fingers. The displacement is dependent on the change in tension in the artery between diastole and systole. The amplitude of the pulse must be judged as to whether it is normal, low, or increased.

5. The low-amplitude (the low-volume) pulse usually indicates low momentum of ejection due to decreased stroke volume and low pulse pressure as seen in significant obstructive lesions of the outflow or the inflow tracts (severe aortic or mitral stenoses), poor ventricular pump function as in severe cardiomyopathy or heart failure, and severe reductions in left ventricular filling as in cardiac tamponade or significant loss in the blood volume or the extracellular fluid volume.

6. In severe systemic hypertension with excessive vasoconstriction, the peripheral pulse amplitude may be poor due to decreased change in radius and wall tension.

7. If the pulse amplitude is considered increased or exaggerated (“bounding pulses”), then conditions associated with large stroke volume and low peripheral resistance must be considered. These include aortic regurgitation and aortic regurgitation mimickers (aortic sinus with a communication to a low-pressure chamber such as the right atrium, aorto-pulmonary window, and persistent ductus arteriosus).

8. The pulse amplitude will also be exaggerated in conditions associated with vasodilatation and low peripheral resistance, since in these states (e.g., septic states, drugs causing vasodilatation, arterio-venous communications congenital or iatrogenic or due to pathological processes in systemic organs as in cirrhosis of the liver, chronic renal disease, chronic pulmonary disease, Paget’s disease, and beriberi) the diastolic pressure in the vessel is low and therefore the change in tension is better appreciated.

9. In patients with combined aortic stenosis and regurgitation where both are significant, the amplitude of the carotid pulsation often would reflect the aortic stenosis, whereas the aortic regurgitation will show its effects on the amplitude more peripherally such as in the popliteal arteries.

10. In the elderly with decreased compliance of the large arteries, ejection of normal stroke volume may cause significant systolic hypertension. The pulse amplitude will be high due to rapid pulse wave velocity caused by the increased stiffness and reduced compliance of the arteries, resulting in increased augmentation of central systolic and pulse pressures due to reflection. In fact, even the presence of aortic stenosis may be masked in such patients.

11. The upstroke of the pulse is best assessed over the carotid artery. The normal rate of rise is felt as a sharp tap by the palpating finger. The delayed upstroke is felt as a gentle sustained push. In some, the sensation may be jagged, simulating a "thrill" or "shudder." A normally rising carotid pulse rules out significant fixed aortic stenosis. The delayed carotid upstroke, on the other hand, is indicative of fixed left ventricular outflow obstructive lesions. Rare exceptions are, of course, elderly patients. Delayed upstroke may be masked in the elderly with stiff aorta where the pressure rise may be steep due to the decreased compliance of aorta. The elderly may also have a hump on the upstroke due to an exaggerated anacrotic shoulder mimicking a delayed upstroke without the presence of any significant aortic stenosis.
When the amplitude of the pulse is low as in low stroke output, then the rate of the rise of the pulse is often difficult to judge.

12. When the upstroke of the pulse is thought to be brisk or rapid and if the pulse amplitude is normal or low, then conditions associated with rapid left ventricular ejection such as hypertrophic cardiomyopathy with or without subvalvular dynamic muscular obstruction must be considered. If the upstroke is brisk and the pulse amplitude is normal, then rapid ejection with normal stroke volume as in significant mitral regurgitation must be considered.

13. Bifid pulse contours or pulsus bisferiens must lead one to consider hypertrophic cardiomyopathy with possible obstruction as well as combined aortic regurgitation and stenosis where the aortic regurgitation is more dominant and hemodynamically significant than the aortic stenosis. In hypertrophic cardiomyopathy with obstruction, amplitude of the pulse is low while the upstroke will be brisk and the initial peak will be brisk and taller than the late peak. In combined aortic stenosis and regurgitation with bisferiens, the amplitude will be large and the upstroke will be relatively normal. The second peak will be larger than the first peak.

14. Exaggerated dicrotic wave may occasionally be the cause of a bifid pulse contour. The second impulse in this instance will occur in diastole as timed by auscultation. The exaggerated dicrotic wave is usually associated with low momentum of ejection, usually the result of low stroke volume as in severe heart failure, cardiomyopathy, and cardiac tamponade. In these instances, the pulse amplitude will be expected to be low.

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2007, XVIII, 412 p. 251 illus., 12 illus. in color.,
Hardcover
ISBN: 978-1-58829-776-1
A product of Humana Press