The rate and character of the arterial pulse has been used for millennia for the diagnosis of a wide range of disorders. Perhaps more useful, however, is the direct cannulation of an artery, which allows quantitative information to be extracted.

**What is the Windkessel effect?**

During systole, the LV ejects around 70 mL of blood into the aorta (the SV). The elastic aortic walls expand to accommodate the SV, moderating the consequent increase in intra-aortic pressure from a DBP of 80 mmHg to an SBP of 120 mmHg. The ejected blood possesses kinetic energy, whilst there is storage of potential energy in the stretched aortic wall. In diastole, recoil of the aortic wall converts the stored potential energy back into kinetic energy. This maintains the onward flow of blood during diastole, thereby maintaining DBP; this is known as the ‘Windkessel effect’. This effect converts the sinusoidal pressure wave generated in the heart into a positive and constant pressure at the tissues, much like converting AC to DC electricity. With advancing age, there is degeneration of elastin in the wall of the aorta. The aortic wall becomes less compliant, and its ability to accommodate SV without a large increase in pressure reduces. This accounts for the development of systolic hypertension in the elderly.

**What is the arterial pressure wave?**

Ejection of blood into the aorta generates both an arterial pressure wave and a blood flow wave. The arterial pressure wave is caused by the distension of the elastic walls of the aorta during systole. The wave propagates down the arterial tree at a much faster rate (around 4 m/s) than the mean aortic blood velocity (20 cm/s). It is the arterial pressure wave that is felt as the ‘radial pulse’, not the blood flow wave.

**Describe the arterial pressure waveform for the aorta**

Starting from end-diastole (Figure 33.1), the pressure generated by the LV ejects the SV into the aorta. The intra-aortic pressure rises to a peak value, the SBP, and then falls to a trough, the DBP. The smooth decent of the curve is interrupted at the dicrotic notch, when the aortic valve closes.

**How does the arterial pressure waveform differ at peripheral arteries?**

The morphology of the arterial pressure waveform differs depending on where it is measured (Figure 33.2). As the site of measurement moves more distally:

![Figure 33.1 The arterial waveform.](https://www.cambridge.org/core/terms._animation.https://doi.org/10.1017/CBO9781139226394.035)
The arterial upstroke is steeper and SBP is increased. DBP is decreased. Crucially, MAP is relatively constant wherever it is measured; this is another reason why MAP is the most important measure of blood pressure.

The morphology of the dicrotic notch changes:
- The dicrotic notch is positioned further down the pressure curve.
- Rather than being a sharp interruption in the pressure descent, the dicrotic notch becomes more of a dicrotic wave.

**Figure 33.2** Arterial pressure waveform at different sites.
The change in shape and position of the dicrotic wave is due to it being caused by reflections of the arterial pressure wave rather than aortic valve closure.

Can any other information be gathered from the arterial pressure waveform?

Although the arterial pressure waveform is often only used for measuring SBP, DBP, MAP and HR, it has many other clinical uses:

- **Myocardial contractility.** The slope of the waveform upstroke is a reflection of myocardial contractility: an increased upstroke gradient suggests a greater pressure generated per unit time. However, a reduced upstroke gradient is sometimes seen in aortic stenosis (Figure 33.3a). In contrast, aortic regurgitation is usually associated with normal myocardial contractility but often has a pressure wave with a bisferens appearance.

- **SVR.** The downstroke of the arterial pressure waveform gives information about SVR: a steep downstroke with a low dicrotic notch indicates a low SVR – the arterial waveform looks thin and pointed (Figure 33.3a). Likewise, a high dicrotic notch implies a high SVR.

- **Hypovolaemia.** In positive-pressure ventilated patients, a ‘respiratory swing’ in the arterial pressure waveform is an indicator of hypovolaemia. There is beat-to-beat variation in the systolic pressure of the waveform, caused by the variation in preload throughout the respiratory cycle (Figure 33.3b).

- **Arterial pulse contour analysis.** SV is proportional to the area under the systolic portion of the arterial pressure waveform; arterial pulse contour analysis allows calculation of the CO (see Chapter 28). SVV is calculated by dividing the minimum SV (Area 2) by the maximum SV (Area 1).

Further reading


