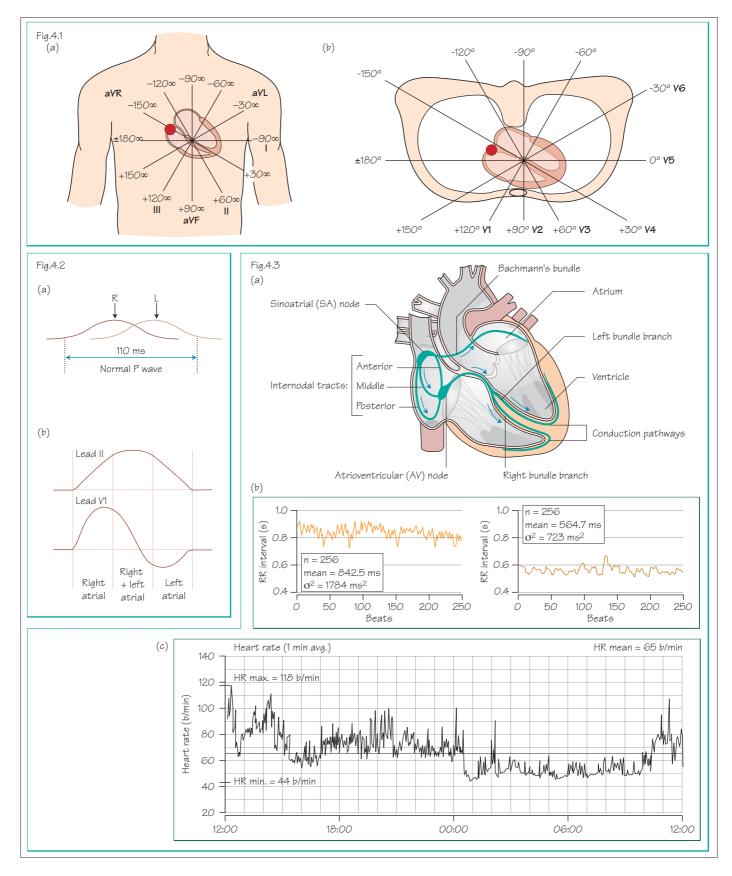


The normal P wave



 \oplus

16 The normal ECG The normal P wave

The P wave reflects the electrical activation of the atria, and allows one to: • Have some idea of where atrial depolarization started and whether the atria are enlarged, as P wave shape relates to where depolarization starts and the route it takes.

• Assess many properties of the sinus node, including heart rate variability, as the P wave reflects sinus node function.

The key points are:

• The best leads to look at the P wave are those directly in or away from the path of atrial depolarization, i.e. lead II and lead V1 (Fig. 4.1a,b).

• The direction of travel of the depolarizing wave through both atria determines the exact shape of the P wave.

• Depolarization starts at the sinus node (Fig. 4.2a,b), then travels directly into the right atria and, via specialized conducting tissue known as the bundle of Bachmann, into the left atria. The time taken for electricity to travel down the bundle of Bachmann means that left atrial depolarization starts a little while after right atrial depolarization, and accordingly goes on for a little while after right atrial depolarization has finished (Fig. 4.3a–c).

The duration of the P wave reflects how long atrial depolarization lasts. The duration is increased if the wave of electricity travels slower than normal (e.g. some cardiomyopathies), or if the wave travels at the normal speed but the atria is enlarged (see Chapter 7). In the former the P wave size is usually diminished, whereas in the latter the P wave is often of a good or better size. The size of the P wave reflects both the volume of electrically active tissue and the insulation between the atria and the observing electrode. If the atria have more/larger myocytes, then the size of the P wave increases; conversely if the number/size of myocytes decreases, or there is more insulation between the heart and the ECG electrode (e.g. pericardial effusion, obesity) then the P wave size diminishes.

Sinus node function

The P wave rate reflects sinus node activity, which is more complex than imagined. The easiest way to assess sinus node function is from a 24-h ECG (Fig. 4.3c) and, though it is possible to look at PP intervals, it is more usual to look at RR intervals (predicated on assuming the PR interval is fixed, or changes only slowly). Important measures of sinus node function include:

• Heart rate which responds to activity (e.g. slows during sleep, increases during exercise), psychological influences and disease.

• Heart rate variability. Heart rate fluctuates over very short time periods (seconds and minutes) in response to autonomic influences. These heart rate fluctuations do not change the average heart rate, but do change the instantaneous heart rate. The sympathetic nervous system is believed to alter heart rate with a periodicity of about 0.1 Hz, and the vagus with a periodicity of about 0.25 Hz.

Fig. 4.1 The position of the atria in the chest in the frontal and horizontal planes, illustrating why leads II and V1 are best for examining the P wave. From the frontal plane (a) it can be seen, as depolarization starts superiorly and spreads inferiorly, that the wave of depolarization, and hence the current flow, is largely directed towards lead II, completely so for the right atrium, largely so for the left atrium. From the horizontal view (b) it can be seen that as the sinus node lies high up in the right atria, and to its back, the wave of right atrial depolarization passes directly towards lead V1. However, the wave of left atrial depolarization passes largely away from lead V1. It is easy to see why in left atrial enlargement the late depolarization phase of the P wave is prolonged and negative in lead V1. Fig. 4.2 (a) Timing and size of the contribution of right and left atria to the shape of the P wave. Right atrial depolarization occurs first, and occupies the first two-thirds of the P wave; left atrial depolarization onset is delayed by about one-third of the duration of the P wave, and then occupies the remaining two-thirds. In health, both contribute equally to the size of the P wave. Thus the first and last thirds of atrial depolarization are exclusively the domain of the right and left atria. Both atria contribute to the middle third of the P wave and hence in health the overall P wave is largest in the middle of the P wave. (b) P wave shape in leads II and V1; both left and

right atrial depolarization are directed towards lead II. Right atrial depolarization is directed towards lead V1, though left atrial depolarization is largely away, accounting for the appearance of a late but small negative P wave deflection in lead V1. Fig. 4.3 (a) Atrial depolarization, which starts at the sinus node, spreads down internodal and interatrial ('bundle of Bachmann') pathways, respectively allowing for right and left atrial depolarization. The impulse then proceeds into the atrioventricular [AV] node and the rest of the heart. (b) Heart rate variability. Two traces of RR interval (essentially the same as PP interval) plotted out against beat number (i.e. instantaneous heart rate). Left lying, right tilted up. Vagal tone, higher when lying, increases high-frequency heart rate variability (σ^2 , measured in milliseconds squared), seen as instantaneous increases/decreases in RR interval (sharp 'spikes' on the tachogram). Standing increases sympathetic tone, increasing heart rate (i.e. lessening RR interval) as vagal tone is withdrawn, lessening the high-frequency changes seen when lying (n = number of beats assessed, μ = average RR interval). (c) Trace of heart rate (HR) plotted out against time from a normal 24-h ECG; normal heart rate variability, with a clear decrease in heart rate at night when asleep.