7 • Changes in the P–QRS–T morphology

Wandering pacemaker

This occurs as a result of the dominant pacemaker shifting from the SA node to other pacemaker cells with a high intrinsic rate within the atria. This is sometimes referred to as a wandering atrial pacemaker. This is a normal variant and not uncommon in dogs. It is thought to be associated with high vagal tone. Its significance is therefore similar to sinus arrhythmia (see page 9).

ECG characteristics

P waves can vary in morphology, i.e. there is a variation in amplitude, varying from positive, negative or biphasic, or they can even be isoelectric (i.e. be so small that they are difficult to identify) (Fig. 7.1).

Changes associated with chamber enlargement

The ECG should not be viewed as being a means to diagnose heart enlargement in small animals, but as an additional diagnostic test that might help to support such a clinical suspicion. Chest radiographs are often considered a better indicator of heart enlargement. Echocardiography is the best means to assess chamber size and morphology.
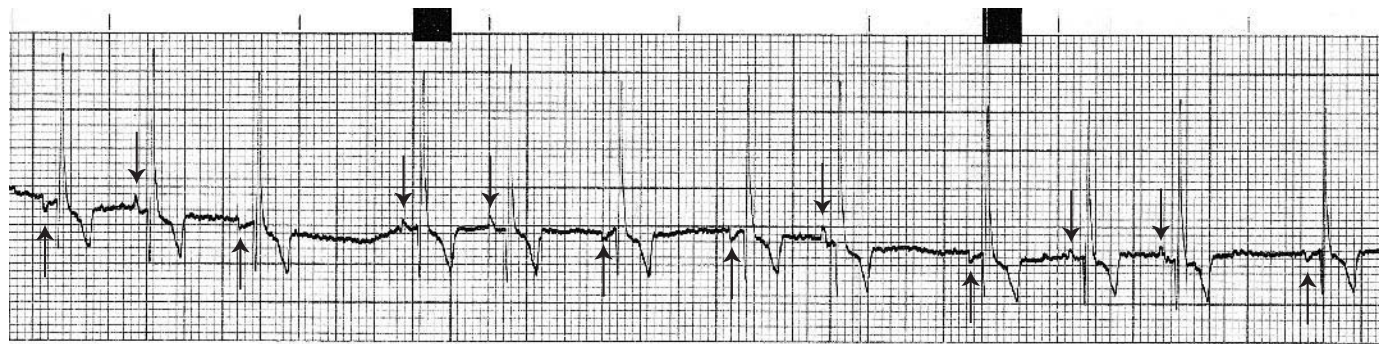

Figure 7.1 ECG from a dog showing a wandering pacemaker. Note how the P wave morphology changes (arrows) (Lead II, 25 mm/sec and 10 mm/mV).

Table 7.1. Guidelines on normal values for cats and dogs (Tilley 1992).

		Dog	Cat
Heart rate	Adult	70–160	120–240
	Puppy	70–220	
Measurements			
P wave duration		<0.04 sec	<0.04 sec
	Giant breeds	<0.05 sec	
P wave amplitude		<0.4 mV	<0.2 mV
P–R interval		0.06–0.13 sec	0.05–0.09 sec
QRS duration		<0.05 sec	<0.04 sec
	Giant breeds	<0.06 sec	
R wave amplitudes		<2.0 mV	<0.9 mV
	Giant breeds	<2.5 mV	
S–T segment	Depression	<0.2 mV	No depression
	Elevation	<0.15 mV	No elevation
T wave		<0.25 of normal R wave amplitude	<0.3 MV
Q–T interval		0.15–0.25 sec	0.12–0.18 sec
Mean electrical axis		+40° to +100°	0° to + 160°

Figure 7.2 ECG illustrating prolonged (0.06 sec) and notched P waves; this is termed P-mitrale. From an 8-year-old Dobermann with dilated cardiomyopathy (50 mm/sec and 10 mm/mV).

Note that in 'ECG-speak', 'enlargement' is commonly used to encompass either hypertrophy or dilation, as these can rarely be distinguished reliably on an ECG.

Table 7.1 lists normal values giving ECG complex durations and amplitudes. Measurements are usually measured in lead II at 50 mm/sec, unfiltered.

Left atrial enlargement

When there is left atrial (LA) enlargement (or dilation) the P wave is often prolonged and sometimes also notched (Fig. 7.2). A prolonged and notched P wave is referred to as **P-mitrale** (as LA enlargement is often associated with mitral valve disease). The notching occurs as a result of asynchronous depolarisation of the atria, the dilated left atrium depolarising fractionally later than the right atrium. Note: giant breeds often normally have slightly prolonged P waves.

Right atrial enlargement

When there is right atrial (RA) enlargement (or dilation) the P wave is increased in amplitude (Fig. 7.3). Such tall P waves are referred to as **P-pulmonale** (as RA enlargement may be associated with cor pulmonale). Note that P-pulmonale is commonly seen in breeds that are predisposed to chronic airway disease.

Figure 7.3 ECG illustrating tall P waves (0.5 mV); this is termed P-pulmonale. There is fine muscle tremor artifact affecting the baseline. From a 10-year-old Yorkshire terrier with long-standing tracheal collapse (25 mm/sec and 10 mm/mV).

Left ventricular enlargement

Tall R waves are suggestive of left ventricular (LV) enlargement (Fig. 7.4). An R wave in lead I greater than leads II or aVF, may be associated with hypertrophy. An increase in R waves in leads I, II and III may be associated with dilation. Other ECG features that may be associated with LV enlargement are: prolongation of the QRS duration, S–T segment sagging/coving (see page 53) or a shift in the mean electrical axis (MEA) to the left.

Right ventricular enlargement

Deep S waves are suggestive of right ventricular (RV) enlargement (Fig. 7.5). Other ECG features that may be associated with RV en-

Figure 7.4 ECG illustrating tall R waves (6.0 mV) and prolonged QRS complex duration (0.06 sec), which is suggestive of left ventricular enlargement. From a 1-year-old German Shepherd dog with a patent ductus arteriosus (50 mm/sec and 5 mm/mV).

largement are: prolongation of the QRS duration or a shift in the MEA to the right.

Abnormal mean electrical axis (MEA)

A **right axis** may suggest right ventricular enlargement (Fig. 7.5), but may be due to displacement of the heart within the chest to the right side or might even be a normal-variation. A conduction disturbance such as right bundle branch block (see Chapter 10) also produces a right axis deviation.

A **left axis** may be due to left ventricular enlargement, but may be due to displacement of the heart within the chest to the left or it may be a normal-variation. A conduction disturbance such as left anterior fascicular block (see Chapter 10) also produces a left axis deviation.
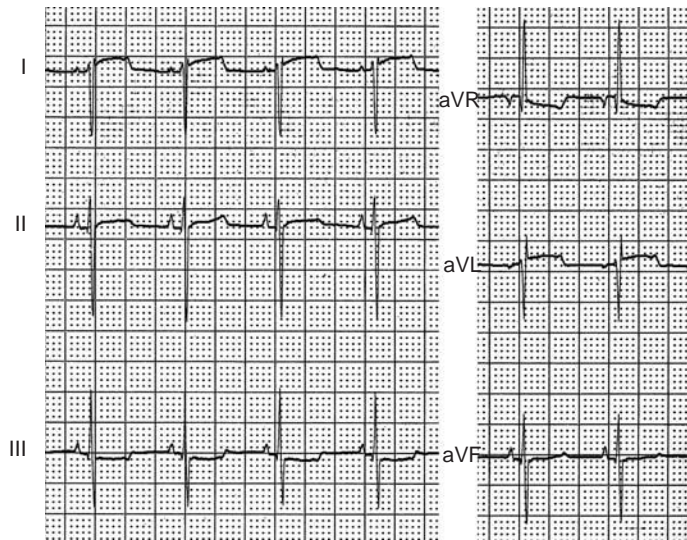
Figure 7.5 ECG illustrating deep S waves in Leads I and II and an axis shift towards aVR, i.e. to the right. From a 2-year-old West Highland White terrier with pulmonic stenosis (25 mm/sec and 5 mm/mV).

Low-voltage QRS complexes

QRS complexes will be smaller the further the electrodes are from the heart and depend on the resistance to electrical conduction between the heart and the electrodes. For example, the ECG complexes are larger in precordial chest leads, which are very close to the heart. However, complexes can be small in limb leads in obese animals. Heavy filtering on the ECG machine can also reduce the amplitude of the ECG complexes significantly.

Small complexes in dogs may be due to obesity, effusions (pericardial, pleural, ascites), hypothyroidism, hyperkalaemia, pneumothorax, some respiratory diseases, hypovolaemia or it may be a normal-variation.

ECG characteristics

An R wave amplitude less than 0.5 mV in the limb leads is considered small in dogs (Fig. 7.6). QRS complexes are usually small in normal cats.
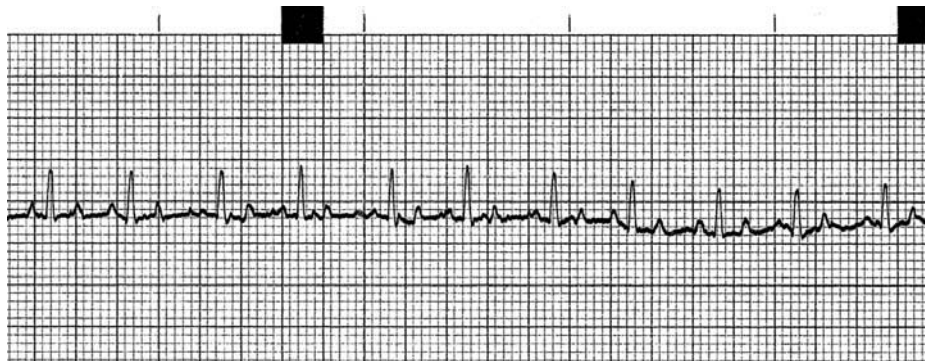

Figure 7.6 ECG illustrating small ECG complexes in a dog (25 mm/sec and 10 mm/mV).

Figure 7.7 (a) ECG illustrating electrical alternans – note the alternating amplitude of the R waves. From a Golden Retriever with pericardial effusion due to idiopathic pericarditis (25 mm/sec and 10 mm/mV).
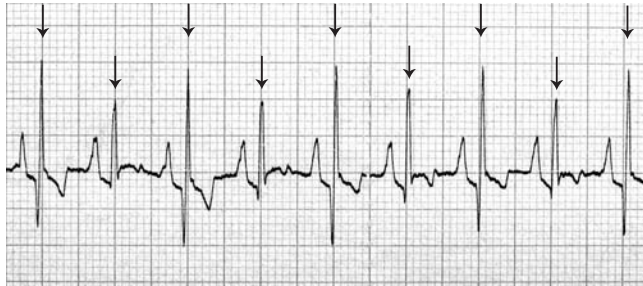

Figure 7.7 (b) ECG from a German Shepherd dog with perciardial effusion secondary to a right atrial haemangiosarcoma illustrating electrical alternans. The QRS complexes are arrowed. Note the alternating amplitudes of the R waves, S waves and T waves in this example (25 mm/sec and 10 mm/mV).

Electrical alternans

This is an alternation in QRS amplitude that occurs nearly every other beat (Fig. 7.7).

Electrical alternans is associated with movement of the heart within pericardial effusion, which is evident on echocardiography where the heart can be seen to 'bounce' from side to side within pericardial fluid as it beats. This movement of the heart causes a slight alternating change in the cardiac axis and is seen on the ECG as an alternating variation in QRS amplitude. Note: this should not be confused with the more gradual variation in amplitude seen with respiration in some animals, nor the variation seen with a supraventricular tachycardia or atrial fibrillation.

Notching in the R wave

Although these abnormalities can be seen commonly in heart disease in small animals, the significance of notches is debatable – it is the 'heart disease' that is of greater importance than trying to analyse every minutia. Notches in the QRS complex are reported to occur with microscopic intramural myocardial infarction or are associated with areas of myocardial fibrosis (Fig. 7.8). Notches in the
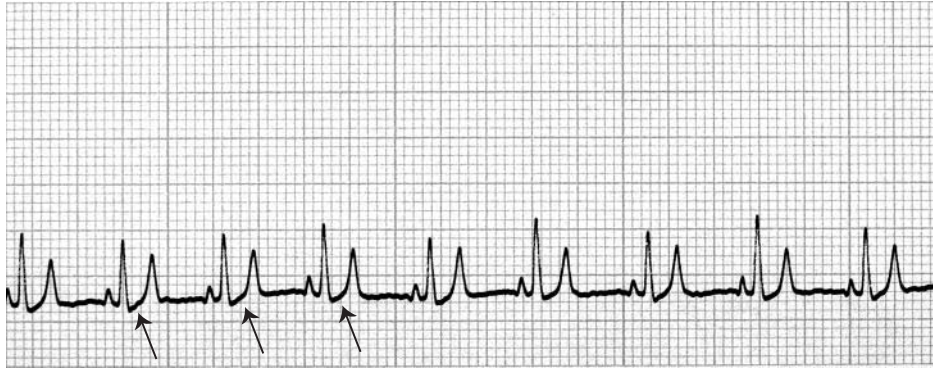
Figure 7.7 (c) ECG from a German Pointer with pericardial effusion. In addition to the electrical alternans, also note the rising S–T segment sometimes seen with pericardial disease (arrowed) (25 mm/sec and 10 mm/mV).

QRS complex are also seen with intraventricular conduction defects (see Chapter 10) and a slight notch is sometimes also seen with ventricular pre-excitation in the upstroke of the R wave (see Chapter 11). Notches can also be produced artifactually in tracings in which there is excessive muscle tremor or electrical interference.

Q–T interval abnormalities

The Q–T interval varies a little, inversely with heart rate, so it is difficult to accurately define what is exactly abnormal.

Prolonged Q–T intervals may be seen in:
- hypocalcaemia
- hypokalaemia
- hypothermia
- quinidine
- ethylene glycol poisoning.

Shortened Q–T interval may be seen in:
- hyperkalaemia

- hypercalcaemia
- digitalis
- atropine
- beta-blockers and calcium channel antagonists.

S–T segment abnormalities

S–T elevation is seen in:
- pericarditis (pericardial effusion) (Fig. 7.7c)
- severe ischaemia/infarction, e.g. full wall thickness.

S–T depression is seen in (Fig. 7.9):
- endomyocardial ischaemia (e.g. cardiomyopathy, trauma)
- potassium imbalance
- digitalis toxicity.

Abnormalities of the T wave

The morphology of T waves in small animals is very variable and the diagnostic value of T wave changes is very limited compared

Figure 7.8 ECG illustrating notching in the QRS complex. From a cat with dilated cardiomyopathy (50 mm/sec and 20 m/mV).
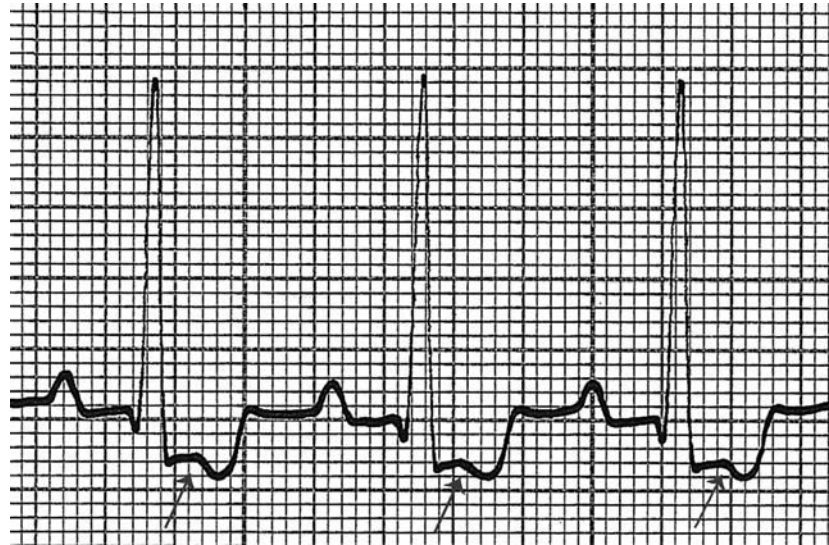

Figure 7.9 ECG illustrating depression of the S–T segment. From a 4-year-old Staffordshire Bull Terrier with mitral valve dysplasia (50 mm/sec and 10 mm/mV).
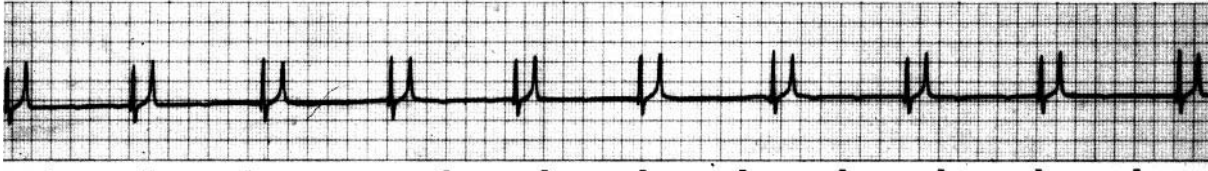
Figure 7.10 (a) ECG illustrating a bradycardia at 50/min, the absence of P waves (atrial standstill) and tall peaked T waves from a dog with hyperkalaemia (25 mm/sec and 10 mm/mV).

with that of humans. A higher value might be placed on T wave changes compared with a previous recording in the same animal. The most common abnormal change is the development of large T waves (Fig. 7.10). This can be associated with hyperkalaemia (see below) or myocardial hypoxia.

Hyperkalaemia

Hyperkalaemia is a well known cause of ECG abnormalities (Fig. 7.10), but it must be remembered that a normal ECG would not exclude hyperkalaemia (e.g. Addison's disease) and serum electrolyte levels should always be measured (and an adrenocorticotrophic hormone test performed) if this is suspected.

Hyperkalaemia may be associated with Addison's disease, acute renal shutdown (e.g. feline urethral obstruction syndrome), diabetic ketoacidosis and severe skeletal muscle damage.

ECG characteristics

The ECG changes vary with increasing severity of the hyperkalaemia as follows:

- there is a progressive bradycardia
- increased amplitude of the T wave, appearing narrow and spiked
- progressive decrease in amplitude of the R wave
- progressive reduction in amplitude of the P wave
- disappearance of the P wave, i.e. atrial standstill, with a slow junctional (nodal) rhythm
- finally, ventricular fibrillation or asystole.

Figure 7.10 (b) ECG from a young dog with Addison's disease and atrial standstill. Note that, unlike in Figure 7.10(a), the T waves are not tall and peaked. The absence of 'characteristic T wave morphology' on an ECG tracing does not exclude hyperkalaemia (25 mm/sec and 10 mm/mV).