5 • Arrhythmias associated with ectopia

'Ectopia' literally means 'in an abnormal place'. In reference to the heart this means outside the SA node, the dominant pacemaker. Ectopic beats arise as a result of various mechanisms due to a number of causes (see Chapter 12).

Ventricular premature complexes

Ventricular premature complexes (VPCs) are a common finding in dogs and cats. VPCs arise from an ectopic focus or foci within the ventricular myocardium. Depolarisation therefore occurs in an abnormal direction through the myocardium and the impulse conducts from cell to cell (not within the conduction tissue) as described in the preceding chapter.

ECG characteristics

The QRS complex morphology is abnormal, i.e. unlike a QRS that would have arisen via the AV node. It is usually:

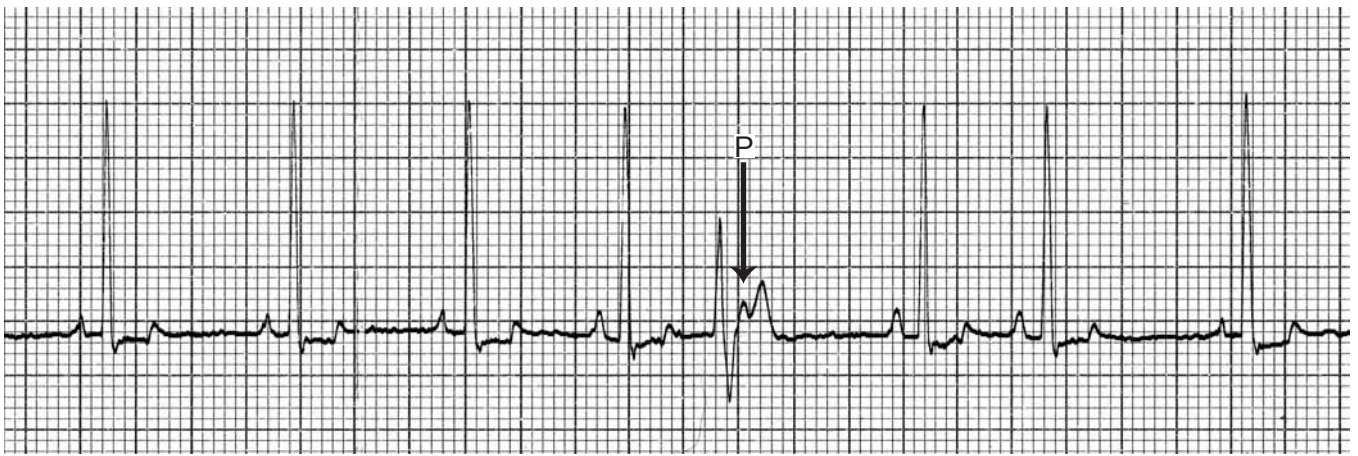

Figure 5.1 ECG from a 10-year-old Springer spaniel showing a single VPC. Note the P wave within the ST segment (arrowed). However this normal sinus depolarisation arrived when the ventricles were still refractory (25 mm/sec and 10 mm/mV).

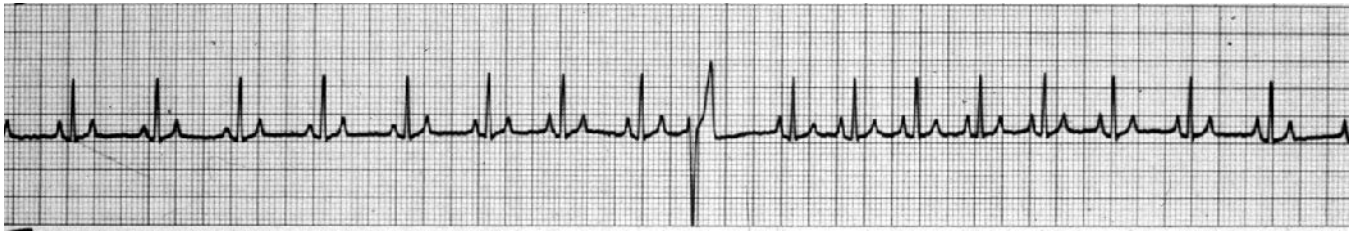

Figure 5.2 ECG from a 10-year-old Labrador dog with a single ventricular premature complex (25 mm/sec and 10 mm/mV).

- abnormal (bizarre) in shape;
- wide (prolonged), typically by ~50%;

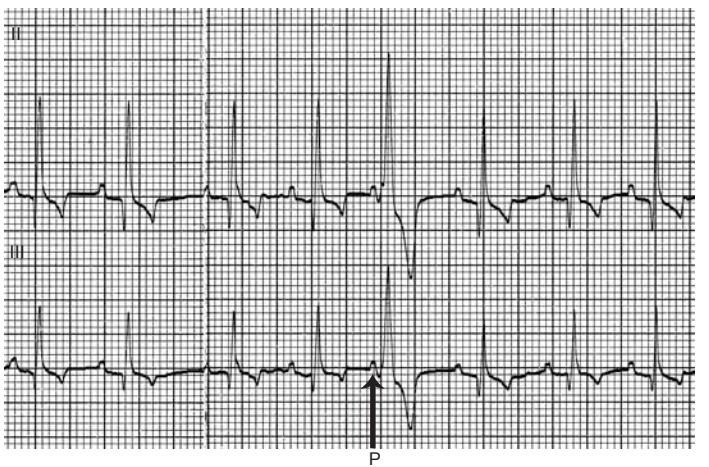

Figure 5.3 ECG from a Boxer with right ventricular arrhythmogenic cardiomyopathy showing a single VPC with a positive QRS morphology. (Note the P wave, arrowed, just preceding the QRS complex with a very short P–R interval. However the VPC occurred before the normal sinus deplorisation.) (Leads 2 and 3, 25 mm/sec and 10 mm/mV.)

- the T wave of a VPC is often large and opposite in direction to the QRS.

Figures 5.1–5.3 show examples of single VPCs in dogs and Fig. 5.4 shows an example in a cat. Additionally a VPC is not associated with a preceding P wave (except by coincidence). Since a VPC occurs prematurely, a normal atrial depolarisation (i.e. a P wave) arriving at the AV node will meet ventricles that are refractory (Figs 5.1 and 5.3), thus the P wave is usually hidden by the ventricular premature complex.

Occasionally a normal sinus complex QRS will occur at the same time as an ectopic VPC, resulting in fusion of the two wavefronts. This is seen on an ECG as a change in QRS & T morphology from the normal sinus QRS & T and the QRS & T of the VPC. Usually the combination tends to neutralise the deflections and the QRS is much smaller in comparison (Figs 5.5a, b).

When a VPC is so premature that it is superimposed on the T wave of the preceding complex (sinus or ectopic), i.e. the ventricles are depolarised before they have completely repolarised from the preceding contraction, this is termed **R-on-T** (Fig. 5.6).

A run of three or more VPCs is termed a **ventricular tachycardia (VT)** (Figs 5.7a, b, c). In the vast majority of cases VT is fairly uniform and regular, however occasionally it can be multiform (Fig. 5.8).

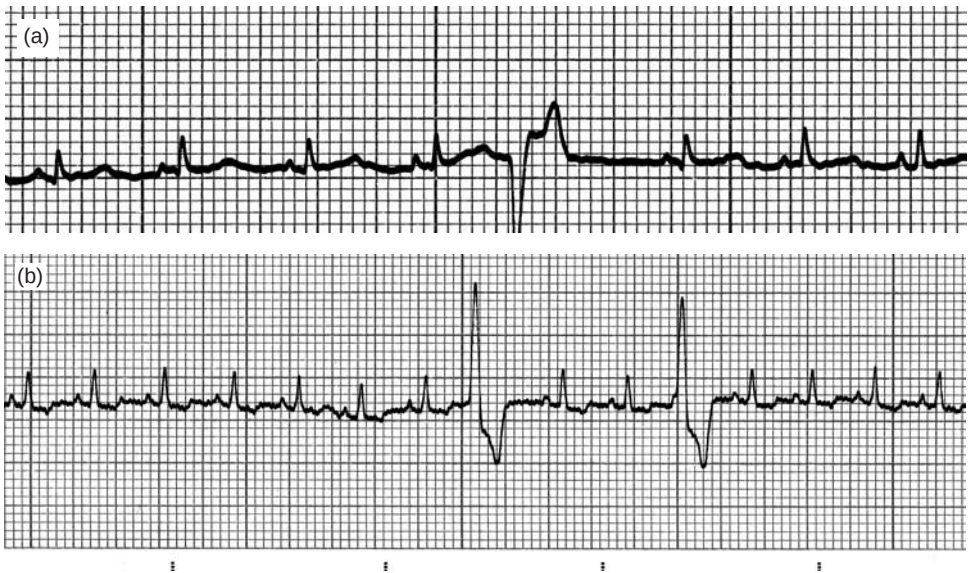

Figure 5.4 ECGs from two cats each showing a VPC, but of differing morphologies: (a) has a negative QRS morphology and (b) has two positive QRS morphology VPCs (25 mm/sec and 10 mm/mV).

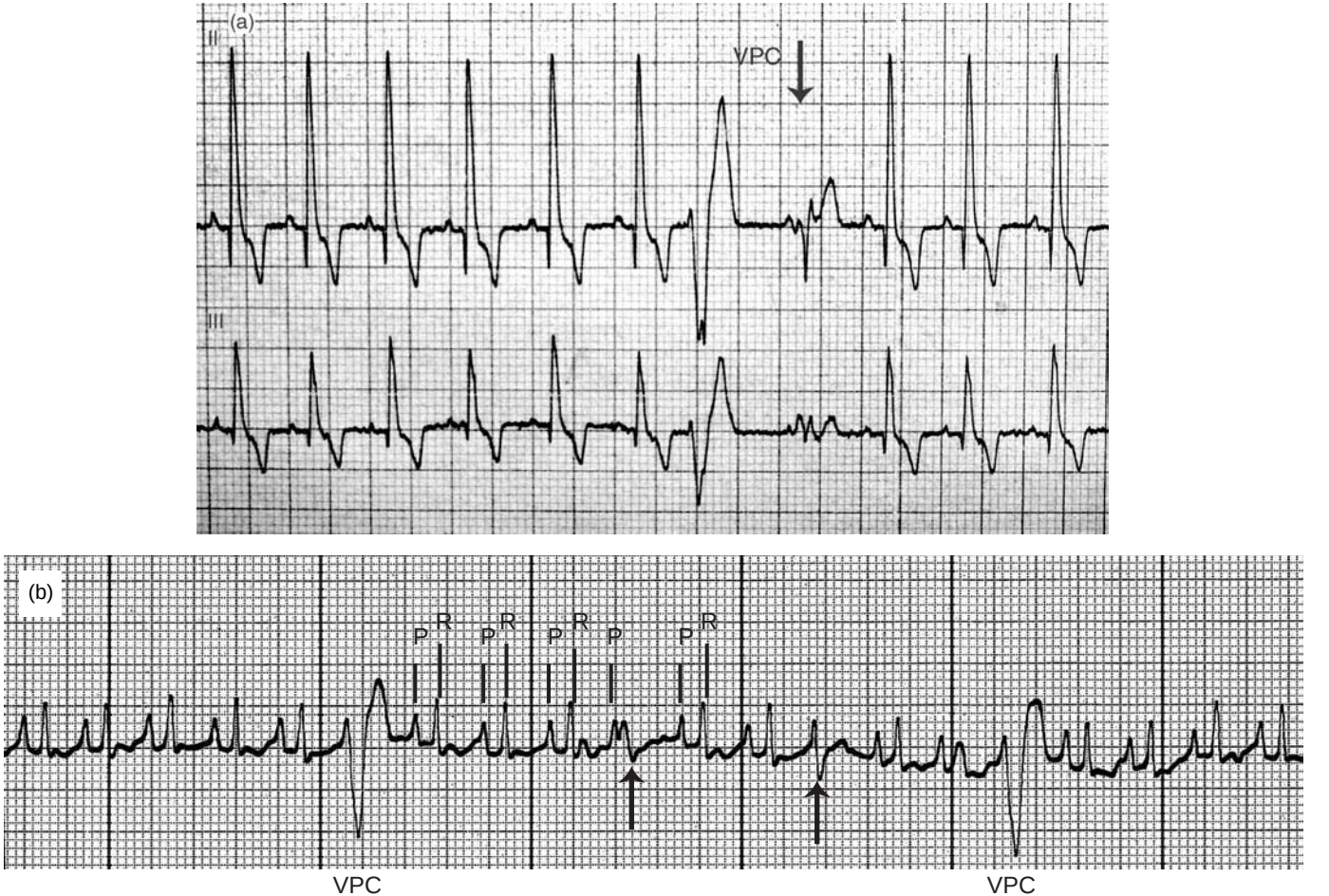

Figure 5.5 ECGs from a dog (a) and cat (b) showing fusion complexes (arrowed). Note the different morphology of the second VPC in each example; this may be a combination of a normal QRS complex and a VPC – this is termed a fusion complex. (a) is from a German Shepherd dog with a splenic mass (Leads 2 and 3, 25 mm/sec and 10 mm/mV) and (b) is from a 6-year-old domestic short-haired cat with hypertrophic cardiomyopathy (25 mm/sec and 20 mm/mV).

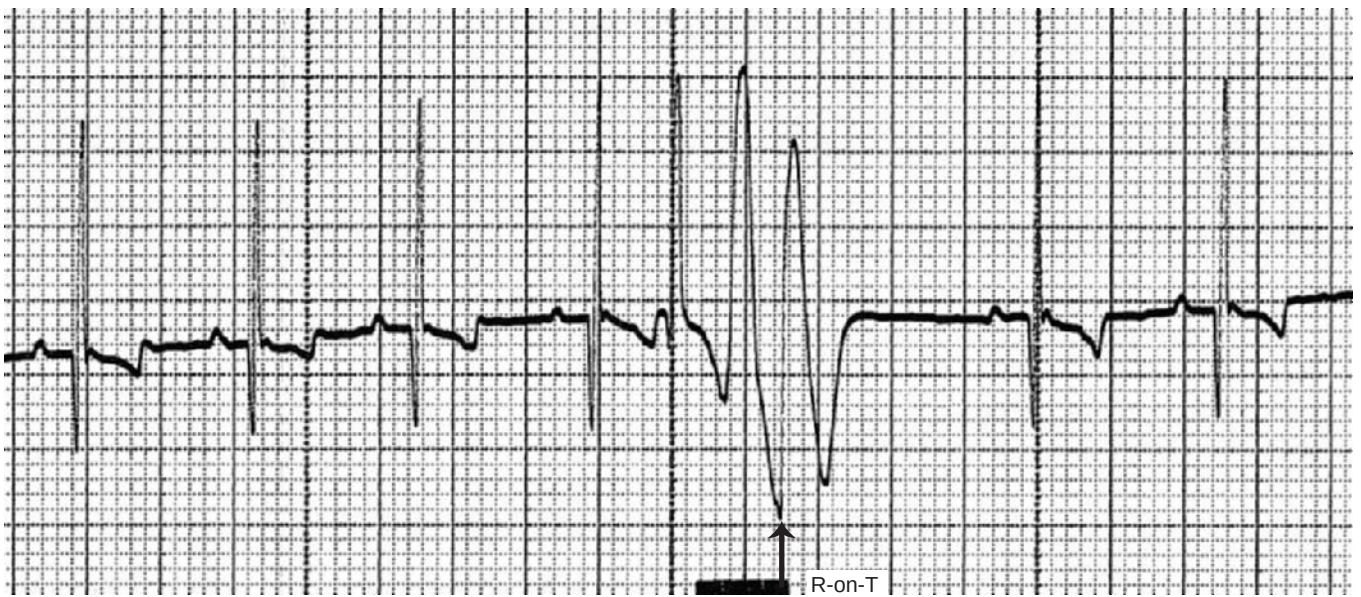

Figure 5.6 ECG from a dog showing a two ventricular premature complex (VPC) occurring very quickly, such that the QRS complex of the second is virtually superimposed on the T wave of the previous VPC (arrowed) – this is termed R-on-T (25 mm/sec and 10 mm/mV).

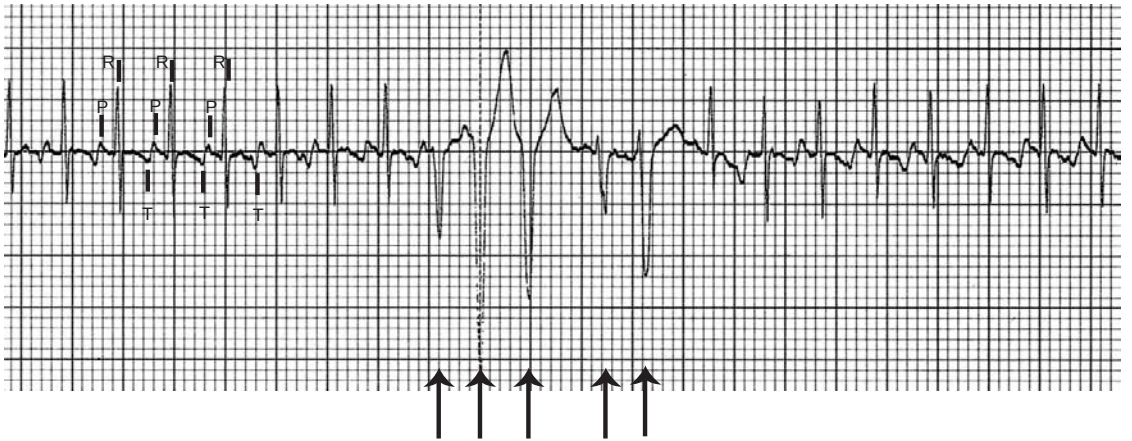

Figure 5.7 (a) ECG showing a paroxysmal ventricular tachycardia (arrows) (25 mm/sec and 10 mm/mV).

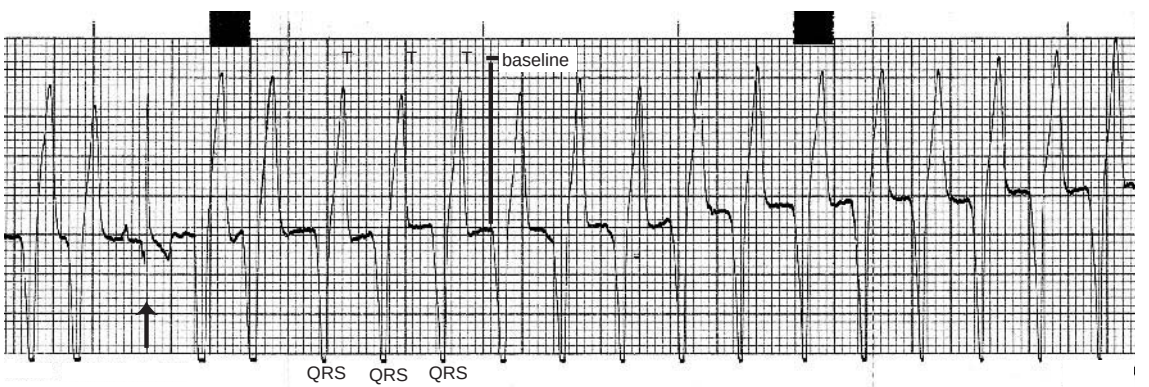

Figure 5.7 (b) ECG showing a ventricular tachycardia (VT) at 200/min with a negative QRS morphology. There is one normal sinus complex (arrowed). From a 10-year-old Labrador with liver neoplasia (25 mm/sec and 10 mm/mV).

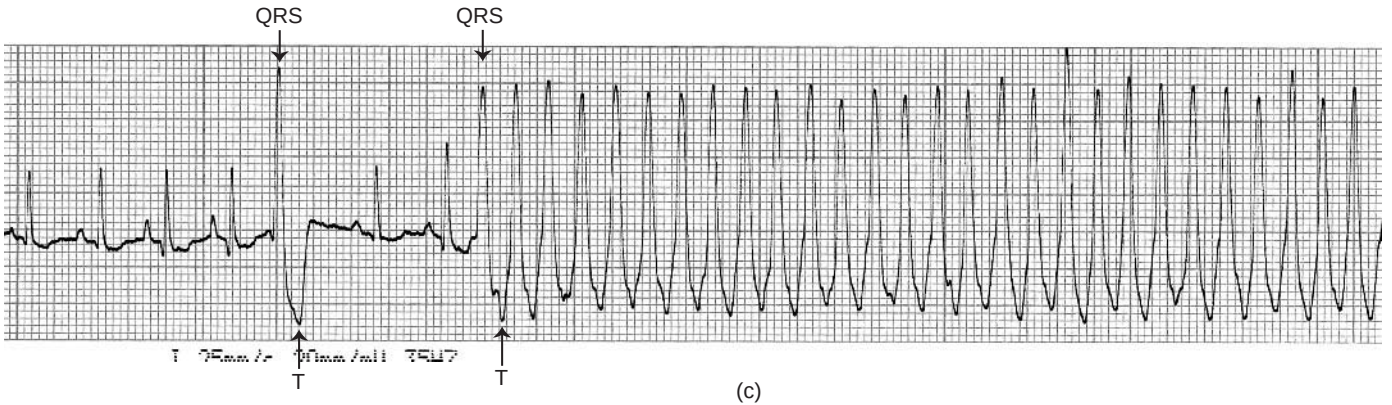

Figure 5.7 (c) ECG from a dog showing the start of a sustained VT, with a positive QRS morphology, preceded by a sinus rhythm and a single VPC (25 mm/sec and 20 mm/mV).

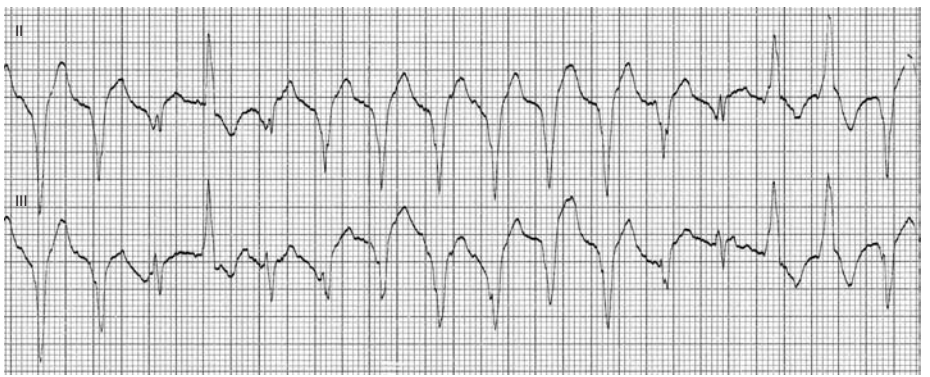

Figure 5.8 (a) ECG from a 14-year-old Jack Russell terrier showing a multimorphic VT (50 mm/sec and 10 mm/mV).

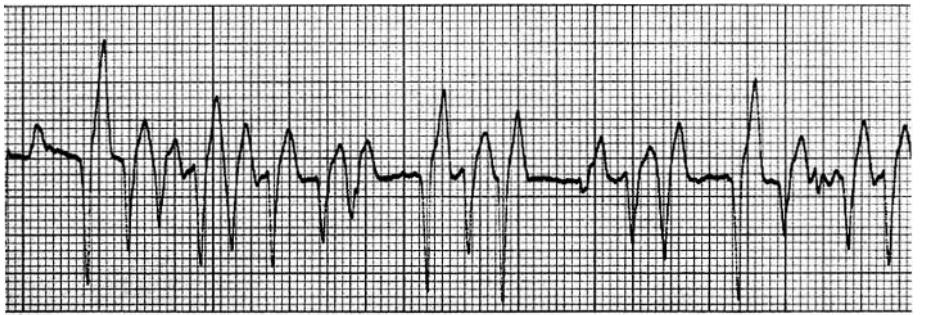

Figure 5.8 (b) ECG from a 10 year old Labrador showing a multimorphic VT (25 mm/sec and 10 mm/mV).

Clinical findings

Occasional premature beats will sound like a 'tripping in the rhythm'. Depending upon how early the beat occurs – the 'extra' premature beat may be heard or it might be 'silent' (and sound like a brief pause in the rhythm). There will be little or no pulse associated with the premature beat (i.e. a pulse deficit). If the premature beats are more frequent, the tripping in the rhythm will start to make the heart rhythm sound more irregular. With very frequent premature beats, the heart rhythm can sound quite chaotic, and with a pulse deficit for each premature beat, the pulse rate will be much slower than the heart rate. During a sustained ventricular tachycardia however, the heart rhythm will sound fairly regular – pulses will probably be palpable, but reduced in strength, becoming weaker with faster heart rates.

Supraventricular premature complexes

Supraventricular premature complexes (SVPCs) arise from an ectopic focus or foci above the ventricles, i.e. in either the atria, the AV node or bundle of His. The ventricles are then depolarised, normally producing a normal-shaped QRS complex with a normal duration.

ECG characteristics

QRS–T complexes, which have a normal morphology, are seen to occur prematurely (Fig. 5.9). The ECG features are:

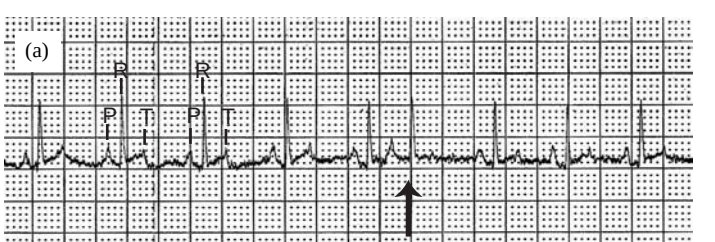

Figure 5.9 (a) ECG showing a single supraventricular premature complex (arrowed). Note the premature timing and absence of an obvious P wave. An incidental finding from a 9-year-old Newfoundland dog (25 mm/sec and 10 mm/mV).

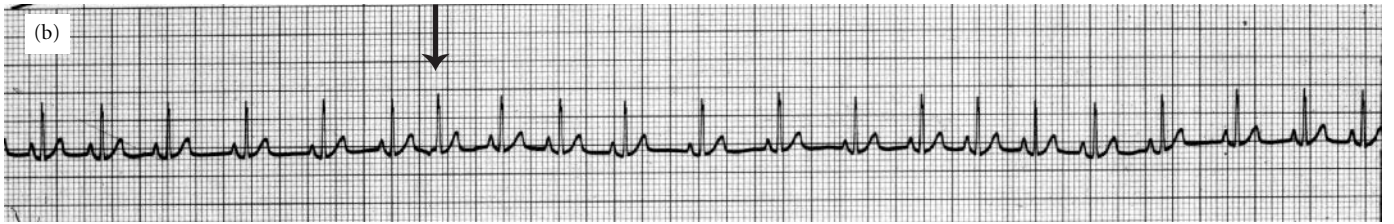

Figure 5.9 (b) ECG from a dog. Note the premature complex (arrowed) with a normal QRS–T morphology – this is a supraventricular premature complex (SVPC) (25 mm/sec and 10 mm/mV).

- normal QRS morphology;[1]
- QRS are seen to occur prematurely;
- P waves may or may not be identified; and
- if P waves are seen, they are usually of an abnormal morphology (i.e. non-sinus) and the P–R interval will differ from a normal sinus complex.

A run of three or more SVPCs is termed a **supraventricular tachycardia (SVT)**; it is usually at a rate in excess of 200/min (but can be as high as 400/min) and regular (Fig. 5.10). SVT needs to be distinguished from a sinus tachycardia.

Clinical findings

On auscultation it is not possible to distinguish ventricular premature beats from supraventricular premature beats (see p. 18). Occasional premature beats will sound like a 'tripping in the rhythm', with little or no pulse associated with the premature beat. If the premature beats are more frequent, the tripping in the rhythm will start to make the heart rhythm sound more irregular. With very frequent premature beats, the heart rhythm can sound quite chaotic, and with a pulse deficit for each premature beat, the pulse rate will be much slower than the heart rate. During a sustained supraventricular tachycardia however, the heart rhythm will sound fairly regular – pulses will probably be palpable, but reduced in strength, becoming weaker with faster heart rates.

Escape rhythms

When the dominant pacemaker tissue (usually the SA node) fails to discharge for a long period, pacemaker tissue with a slower intrinsic rate (junctional or ventricular) may then discharge, i.e. they 'escape' the control of the SA node. This is commonly seen in

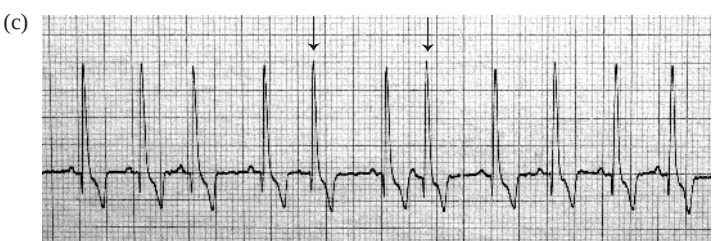

Figure 5.9 (c) ECG from a German Shepherd dog showing two SVPCs (arrowed).

[1] Except when there is aberrant conduction, see Chapter 10.

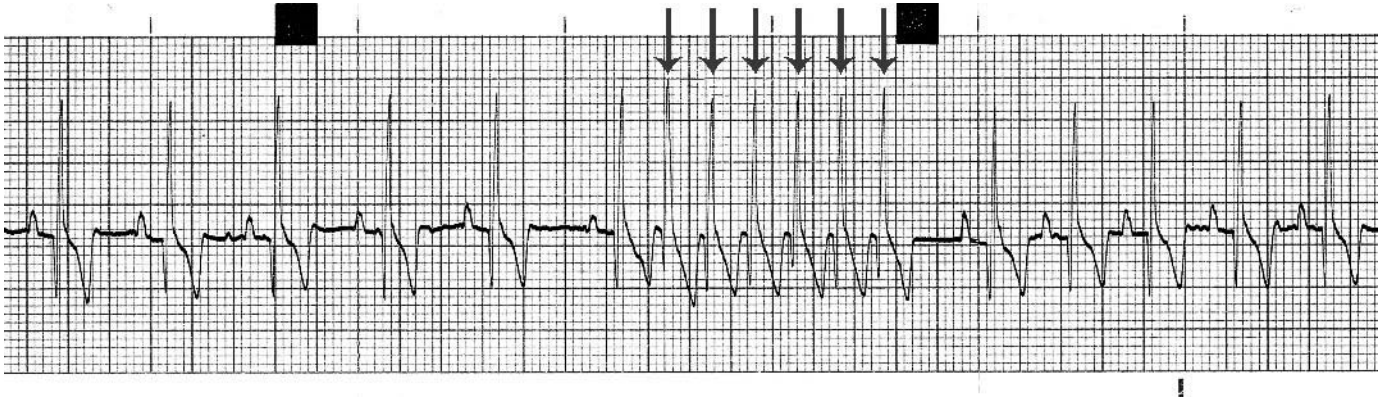

Figure 5.10 (a) ECG showing a paroxysmal supraventricular tachycardia (arrowed) (25 mm/sec and 10 mm/mV).

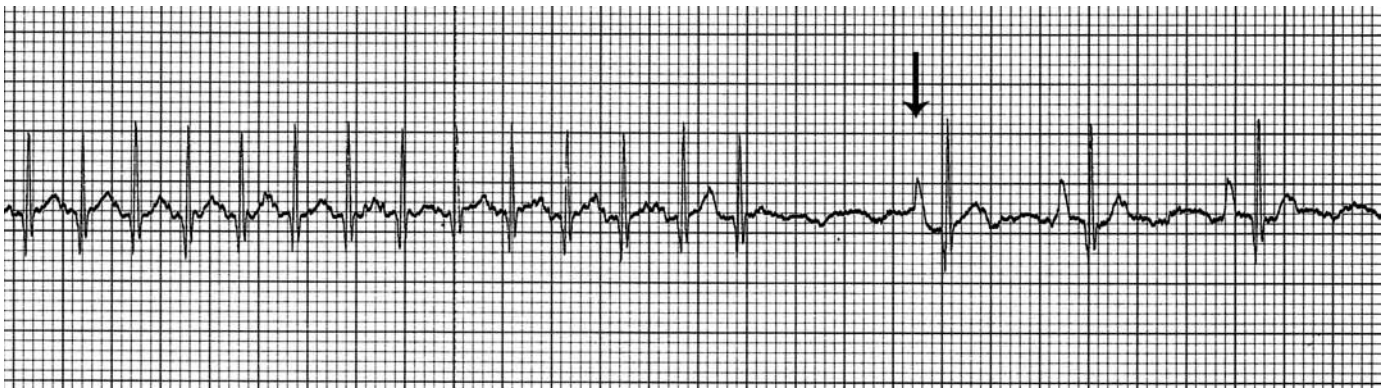

Figure 5.10 (b) ECG showing initially a supraventricular tachycardia at 280/min, which then breaks to a normal sinus rhythm at 90/min (the first sinus complex is arrowed). From a 6-year-old Irish Wolfhound with occult dilated cardiomyopathy (25 mm/sec and 10 mm/mV).

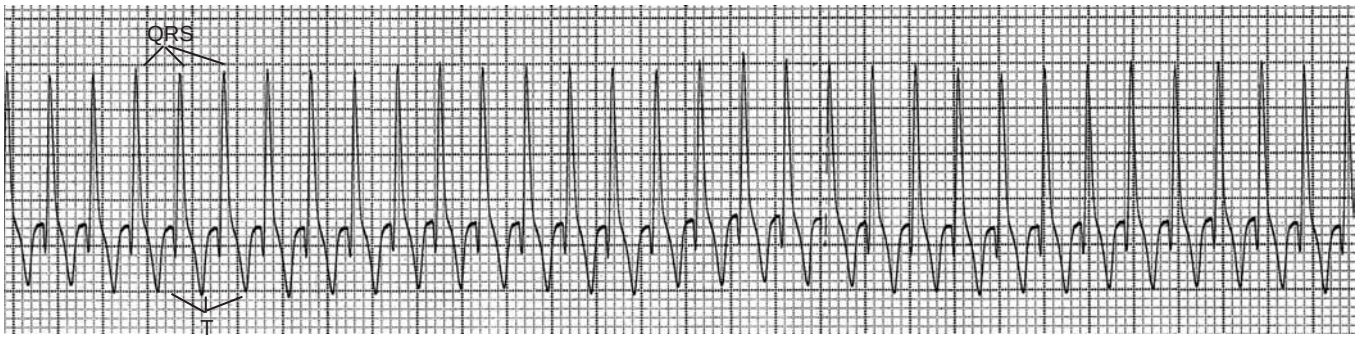

Figure 5.10 (c) ECG showing a sustained supraventricular tachycardia at 320/min. From a 9-year-old Weimaraner with myocarditis (25 mm/sec and 5 mm/mV).

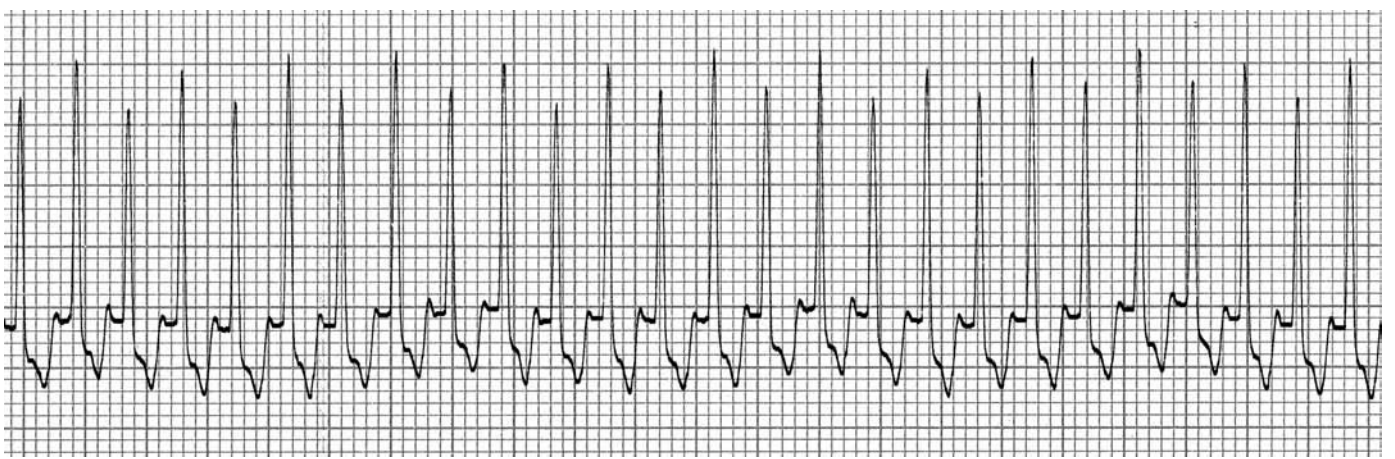

Figure 5.10 (d) ECG from a 7-month-old Labrador with a sustained SVT at 350/min (25 mm/sec and 10 mm/mV).

association with bradyarrhythmias (e.g. sinus bradycardia, sinus arrest, AV block). Escape complexes are sometimes referred to as rescue beats, because if they did not occur death would be imminent. Since they are rescue beats they should not be suppressed by any form of treatment. Treatment should be directed towards the underlying bradyarrhythmia.

If no escape rhythm has developed, i.e. there was no electrical activity of any kind, then this is termed **asystole**. It would not be dissimilar to sustained sinus arrest if no escape rhythm developed. This is a terminal event unless electrical activity returns. If there is a failure of an escape rhythm during complete heart block, i.e. there are P waves but no QRS complexes, then this is termed **ventricular standstill**. Again, if ventricular electrical activity does not return death is imminent.

Junctional escapes are fairly normal in shape (the same as a supraventricular ectopic), whereas ventricular escapes are abnormal and bizarre (the same as a ventricular ectopic): see Figs 5.11 and 6.2. A continuous junctional escape rhythm occurs at a rate of 60–70/min and a continuous ventricular escape rhythm occurs at a rate of less than 50/min. Either may be seen in complete AV block.

AV dissociation

AV dissociation describes the situation when the atria and ventricles are depolarised by separate independent foci. This may occur due to an accelerated junctional or ventricular rhythm, disturbed AV conduction or depressed SA nodal function.

ECG characteristics

The ECG shows a ventricular rate that is usually very slightly faster than the atrial rate. The P waves may occur before, during or after the QRS complex. The P waves and QRS complexes are independent of each other with the QRS complexes appearing to 'catch up' on the P waves (Fig. 5.12). Complete AV block is one form of AV dissociation, but AV dissociation does not mean there is AV block.

Clinical findings

The heart rhythm will sound fairly normal and the pulse should match the heart rate.

Fibrillation

Fibrillation means rapid irregular small movements of fibres.

Atrial fibrillation

This is probably one of the most common arrhythmias seen in small animals. In atrial fibrillation (AF) depolarisation waves occur randomly throughout the atria (Fig. 5.13). Since AF originates

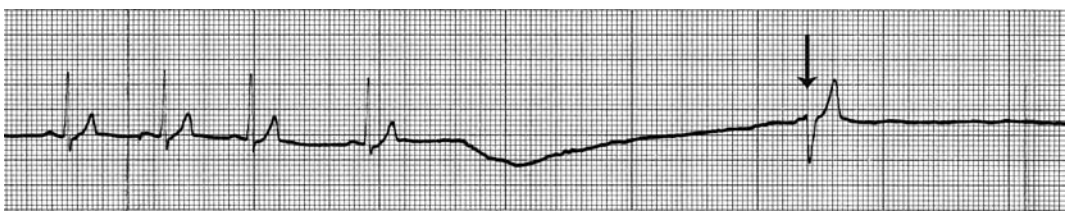

Figure 5.11 (a) ECG from a Springer spaniel with sinus arrest. Following the long flatline, there is a ventricular escape complex (arrowed) (25 mm/sec and 10 mm/mV).

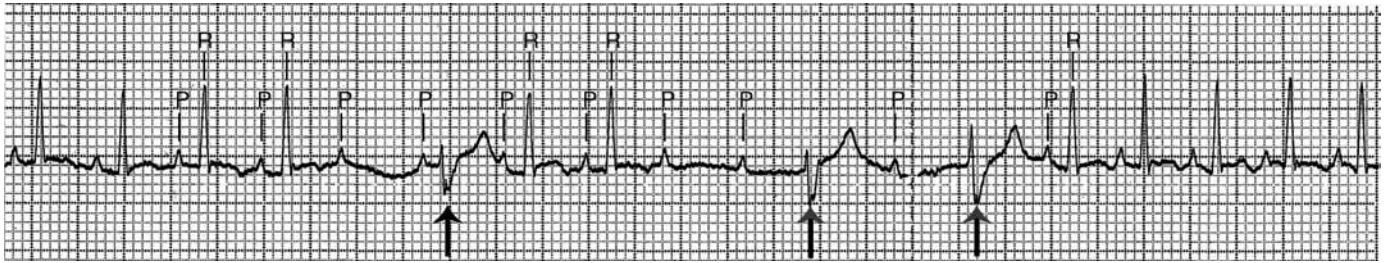

Figure 5.11 (b) ECG from a 7-year-old cat with intermittent failure of AV nodal conduction through to the ventricles (non-conducted P waves), i.e. second degree heart block (see later). Following the consequential pauses in ventricular depolarisation, ventricular escape complexes occur (arrowed) (25 mm/sec and 20 mm/mV).

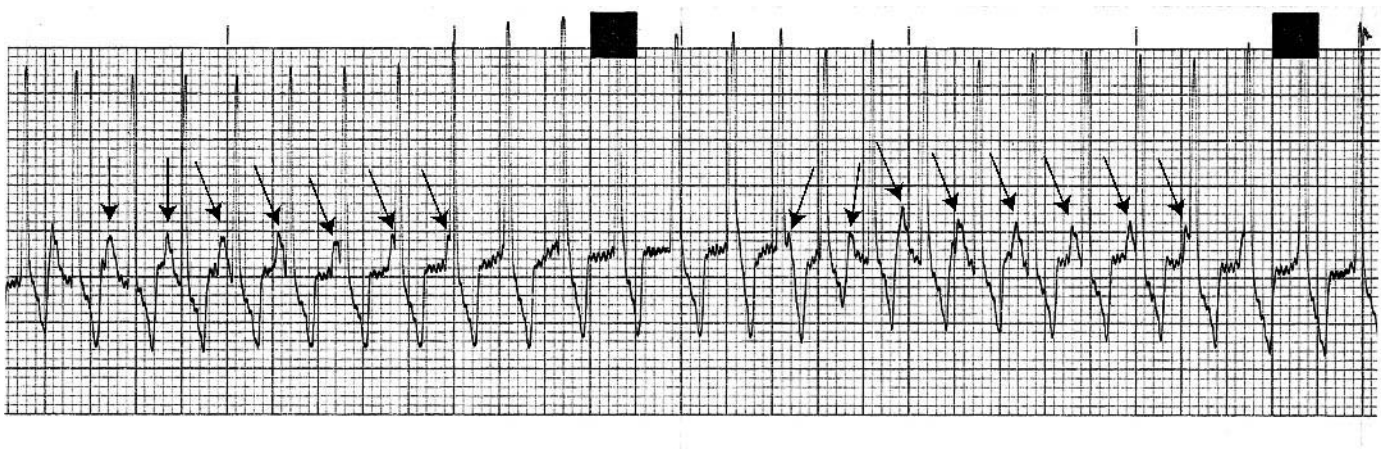

Figure 5.12 (a) ECG showing AV dissociation. Note how the P waves (arrows) appear to drift in and out of the QRS complexes (the P–R interval is variable). Incidental finding from a 13-year-old Samoyed dog (25 mm/sec and 10 mm/mV).

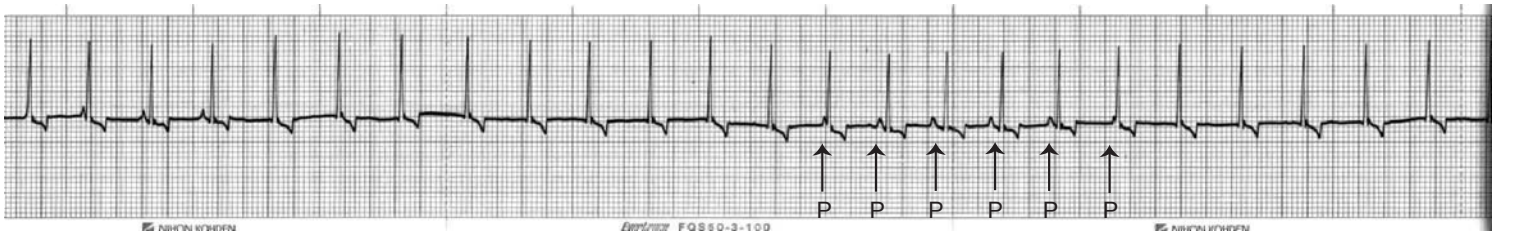

Figure 5.12 (b) ECG from a 6-year-old Labrador dog showing AV dissociation. This was an incidental finding. P waves are arrowed (25 mm/sec and 10 mm/mV).

above the ventricles, it could also be classified as a supraventricular arrhythmia.

ECG characteristics

The QRS complexes have a normal morphology (similar to supraventricular premature complexes described previously) and occur at a normal to fast rate (Fig. 5.14). The ECG features are:

- normal QRS morphology;[2]
- the R–R interval is irregular and chaotic (this is easier to hear on auscultation);
- the QRS complexes often vary in amplitude;
- there are no recognisable P waves preceding the QRS complex; and

[2] Except when there is bundle branch block (see Chapter 10).

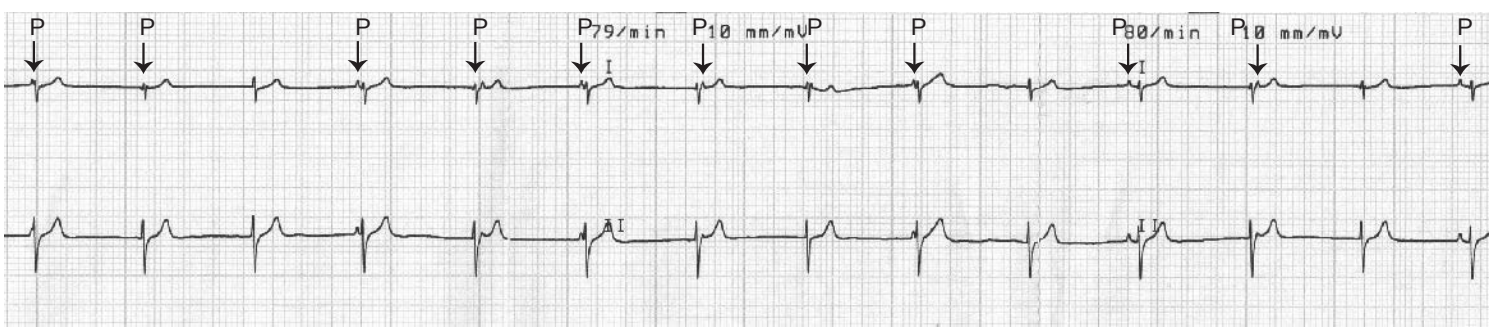

Figure 5.12 (c) ECG from a cat following sedation with medetomidine with a bradycardia (80/min) and AV dissociation. P waves are arrowed (Leads 1 and 2, 25 mm/sec and 10 mm/mV).

Tip

As a guideline[3]: Supraventricular QRS morphology + Irregular (chaotic) R–R intervals + Absence of P waves = Atrial fibrillation

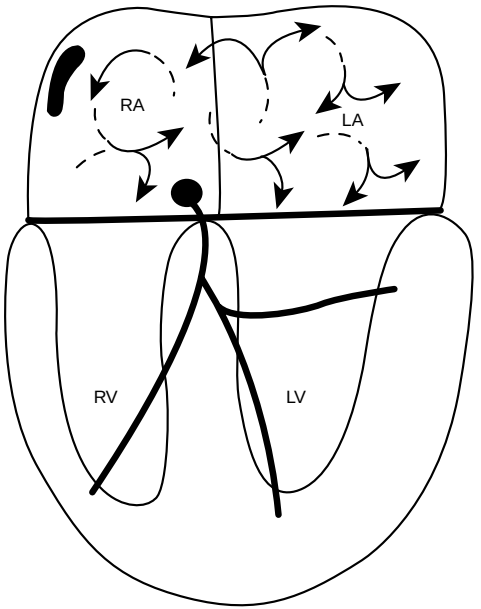

Figure 5.13 Diagram illustrating the wavelet theory for atrial fibrillation. Note the multiple small wavelets that randomly depolarise small portions of the atria. This is easiest to sustain when the atria are large such as in giant-breed dogs or animals in which there is atrial dilation.

- sometimes fine irregular movements of the baseline are seen as a result of the atrial fibrillation waves – referred to as **'f' waves**, however these are frequently indistinguishable from baseline artifact (e.g. muscle tremor) in small animals.

Clinical findings

The heart rhythm sounds chaotic and the pulse rate is often half the heart rate, especially with fast atrial fibrillation. This is a very common arrhythmia in dogs, and can be strongly suspected on auscultation by its chaotic rhythm and 50% pulse deficit. However, very frequent premature beats (ventricular or supraventricular) can mimic it.

Ventricular fibrillation (VF)

This is nearly always a terminal event associated with cardiac arrest. The depolarisation waves occur randomly throughout the ventricles (Fig. 5.15). There is therefore no significant coordinated contraction to produce any cardiac output. If the heart is visualised or palpated, fine irregular movements of the ventricles are evident – likened to a 'can of worms'. VF can often follow ventricular tachycardia.

ECG characteristics

The ECG shows **coarse** (larger) or **fine** (smaller) rapid, irregular and bizarre movement with no normal wave or complex (Fig. 5.16).

Clinical findings

No heart sounds are heard. No pulse is palpable.

[3]Except when there is bundle branch block, see Chapter 10.

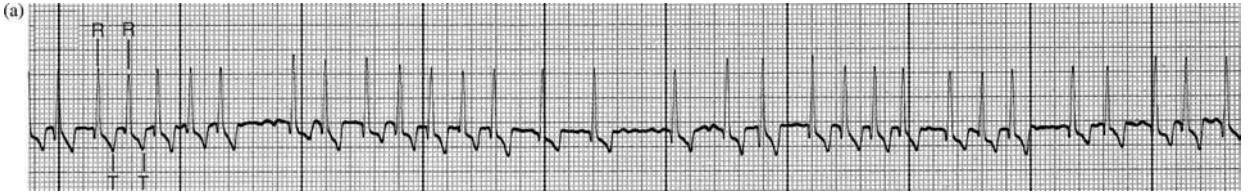

Figure 5.14 (a) ECG showing atrial fibrillation with an average ventricular response rate of 180/min. The QRS complexes are chaotic – this is usually easier to hear on auscultation of the heart. There are no P waves discernible, although the fine undulations of the baseline may be fibrillation waves in this instance (25 mm/sec and 10 mm/mV).

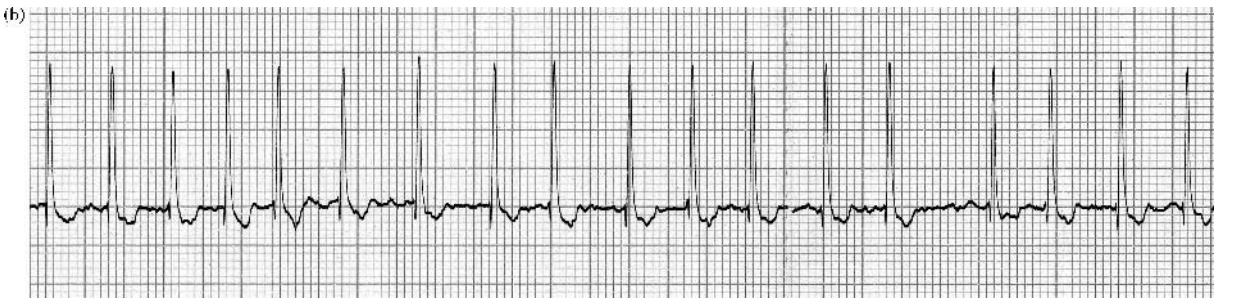

Figure 5.14 (b) ECG showing atrial fibrillation with an average ventricular response rate of 180/min. From a 10-year-old Whippet with mitral valve disease (25 mm/sec and 10 mm/mV).

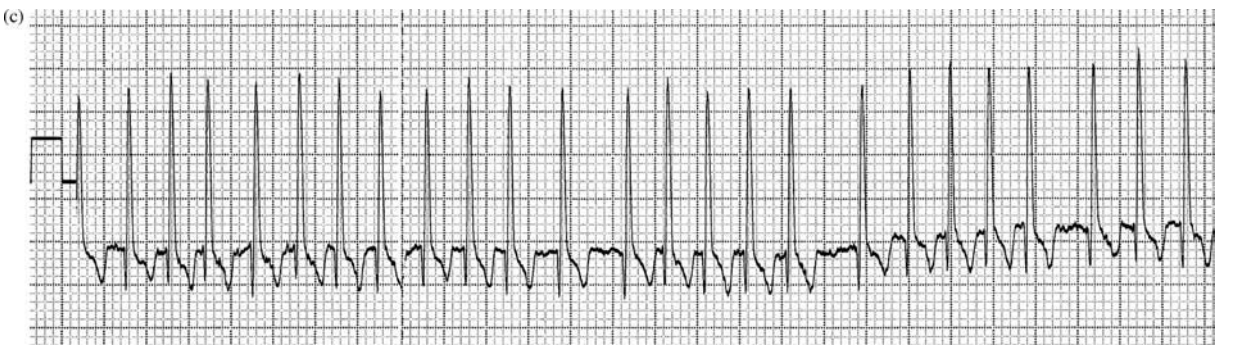

Figure 5.14 (c) ECG showing atrial fibrillation with an average ventricular response rate of 280/min. Such a fast ventricular rate is usually seen when the dog is in congestive heart failure. From a 7-year-old Dobermann with pulmonary oedema due to dilated cardiomyopathy (25 mm/sec and 5 mm/mV).

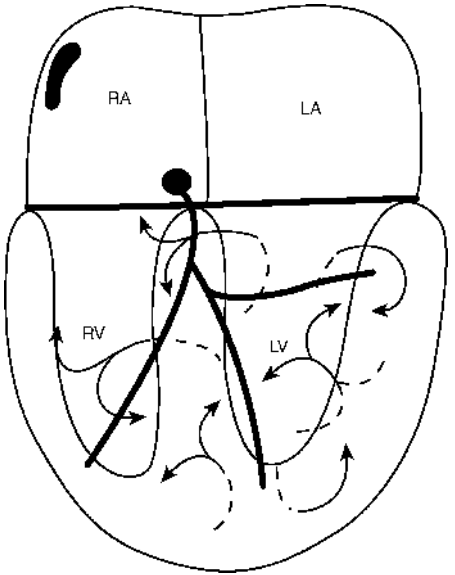

Figure 5.15 Diagram illustrating the wavelet theory for ventricular fibrillation (VF). VF initially consists of large wavelets (coarse VF), that progress over a short period of time to a greater number of smaller wavelets (fine VF).

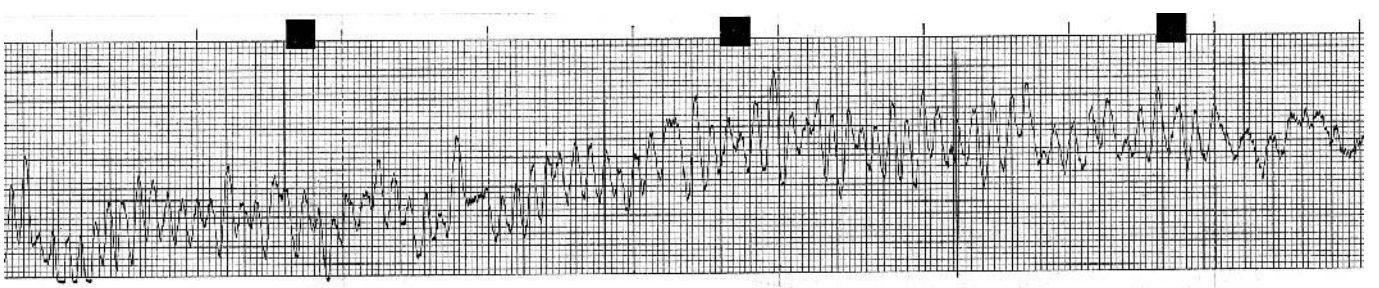

Figure 5.16 ECG showing ventricular flutter/fibrillation. From an 11-year-old German Shepherd dog that died on arrival with advanced cardiac tamponade due to pericardial haemorrhage (25 mm/sec and 10 mm/mV).