Electrocardiogram and Heart Sounds
The body depends on the heart pumping oxygenated blood to the tissues.

Unoxygenated blood enters the right side of the heart and is pumped to the lungs (pulmonary circulation).

From the lungs, the newly oxygenated blood enters the left side of the heart where it is pumped to the organs and tissues via the systemic circulation.
Conduction System

Electrical impulses are transmitted through the heart via specialized conduction cells in the following sequence:

- Sinoatrial node.
- Interatrial and internodal conduction tracts.
- Atrioventricular (AV) node.
- Bundle of His (the region of the AV node and the Bundle of His is called the AV junction).
- The left and right bundle branches.
- Purkinje fibers.
Five physiologic properties of cardiac muscle

• Automaticity: SA node is the primary pacemaker of the heart, but any cells in the conduction system can initiate their own impulses under the right circumstances.

• Excitability: Cardiac muscle is excited when the electrical stimulus reduces the resting potential to the threshold potential. The degree of resting potential within the cell determines its excitability and obey the all or none law.

• Refractoriness: Heart muscle will not respond to external stimuli during its period of contraction.
Five physiologic properties of cardiac muscle

• Conductivity: Activation of an individual muscle cell produces activity in the neighboring muscle cells. Conduction velocity varies in the different portions of the specialized conduction system and muscle fibers. Velocity is greatest in the Purkinje fibers and least in the mid portion of the AV node.

Activation sequence is so arranged that the maximum mechanical efficiency is provided from each corresponding contraction.

Contractility: Occurs in response to electrical current. Remember that ECG only measures the stimulus for contraction – not the actual contraction itself. Echocardiography is the tool of choice for assessing contractility.
Formation of the normal P–QRS–T complex

- All cells within the heart have the potential to generate their own electrical activity, however the sinoatrial (SA) node is the fastest part of the electrical circuit to do so and is therefore the ‘rate controller’, termed the pacemaker. The rate of the SA node is influenced by the balance in autonomic tone, i.e. the sympathetic (increases rate) and parasympathetic (decreases rate) systems. The electrical discharge for each cardiac cycle (Fig. 2.1) starts in the SA node. Depolarisation spreads through the atrial muscle cells. The depolarisation wave then spreads through the atrioventricular (AV) node, but it does so relatively slower, creating a delay.
Formation of the normal P–QRS–T complex

- Conduction passes through the AV ring (from the atria into the ventricles) through a narrow pathway called the bundle of His. This then divides in the ventricular septum into left and right bundle branches (going to the left and right ventricles). The left bundle branch divides further into anterior and posterior fascicles. The conduction tissue spreads into the myocardium as very fine branches called Purkinje fibres. The SA node is therefore the start of the electrical depolarisation wave. This depolarisation wave spreads through the atria (somewhat like the ripples in water created by dropping a stone into it). As the parts of the atria nearest the SA node are depolarised (Fig. 2.2), this creates an electrical potential difference between depolarised atria and parts not yet depolarised (i.e. still in a resting state).
Fig. 1–5. Diagram of the action potential of a nonpacemaking cell (on the left) with the relationship of the electrocardiogram (on the bottom). The action potential of a pacemaking cell is illustrated on the right.
If negative (−ve) and positive (+ve) electrodes were placed approximately in line with those as shown on the diagram (Fig. 2.2), then this would result in the voltmeter (i.e. the ECG machine) detecting the depolarisation wave travelling from the SA node, across the atria, in the general direction of the +ve electrode. On the ECG recording, all positive deflections are displayed as an upward (i.e. positive) deflection on the ECG paper, and negative deflections are displayed downwards. The atrial depolarisation wave therefore creates an upward excursion of the stylus on the ECG paper. When the whole of the atria become depolarised then there is no longer an electrical potential difference and thus the stylus returns to its idle position – referred to as the baseline. The brief upward deflection of the stylus on the ECG paper creates the P wave, representing atrial electrical activity (Fig. 2.3). The muscle mass of the atria is fairly small and thus the electrical changes associated with depolarisation are also small.
• **The P–R interval**
• During the course of atrial depolarisation, the depolarisation wave also depolarises the AV node. The speed with which the electrical depolarisation wave travels through the AV node is deliberately slow so that ventricular contraction will be correctly coordinated following atrial contraction. Once the depolarisation wave passes through the AV node, it travels very rapidly through the specialised conduction tissues of the ventricles, i.e. the bundle of His, the left and right bundle branches and Purkinje fibres.
• **The Q waves**

• Initially the first part of the ventricles to depolarise is the ventricular septum, with a small depolarisation wave that travels in a direction away from the +ve electrode (Fig. 2.4). This creates a small downward, or negative, deflection on the ECG paper – termed the Q wave.
• The R wave
• Then the bulk of the ventricular myocardium is depolarised. This
• creates a depolarisation wave that travels towards the +ve electrode
• (Fig. 2.5). As it is a large mass of muscle tissue, it usually creates a
• 6 large deflection – this is termed the R wave.
• **The S wave**

• Following depolarisation of the majority of the ventricles, the only remaining parts are basilar portions. This creates a depolarisation wave that travels away from the +ve electrode and is a small mass of tissue (Fig. 2.6). Thus, this creates a small negative deflection on the While the different parts of the QRS waveform can be identified, it is often easier to think of the whole ventricular depolarisation waveform as the QRS complex. This will avoid any confusion over the correct and proper naming of the different parts of the QRS complex.
• *The T wave*

• Following complete depolarisation (and contraction) of the ventricles they then repolarise in time for the next stimulus. This phase of repolarisation creates a potential difference across the ventricular myocardium, until it is completely repolarised. This results in a deflection from the baseline – termed the T wave (Fig. 2.7). The T wave in dogs and cats is very variable, it can be negative or positive or even biphasic (i.e. a bit of both). This is because repolarisation of the myocardium in small animals is a little random, unlike in humans, for example, in which repolarisation is very organised and always results in a positive T wave. Thus, the diagnostic value obtainable from abnormalities in the T wave of small animals is very limited, unlike the very useful features of abnormal T waveforms seen in humans.
Generation of the Electrocardiogram

The following illustrations depict the genesis of the ECG on the basis of:

✓ Initiation of the impulse in the primary pacemaker (sinoatrial node).

✓ Transmission of the impulse through the specialized conduction system of the heart.

✓ Activation or depolarization of the atrial and ventricular myocardium.

✓ Recovery or repolarization of the preceding three areas.
Electrocardiographic Recording Technique

Patient cable—a single lead or 6-lead patient cable can be attached to a hand-held recorder/monitor or any standard ECG strip chart recorder, and the ECG can be recorded with the patient in lateral or standing position. The electrode clips are attached directly to the skin and moistened with alcohol or gel to assure good contact.

- Record lead II for 30-60 seconds at 25 mm/sec to assess arrhythmias
- Record a brief tracing at 50 mm/sec for ease of measurement of P-QRS-T waveforms.
Standardization Signal

Without a standardization signal, the ECG paper is merely graph paper. By entering a signal, you place value on each of the smallest squares.

At 1 cm = 1 mv each tiny square represents 0.1 mv in height (amplitude) and 0.02 seconds in width (duration) at a paper speed of 50 mm/sec. It wouldn’t be such a bad idea to memorize this.
Calculating the Heart Rate

The heart rate (beats/minute) can be calculated easily by:

1. Using the ECG ruler on the next page. The ruler allows you to use 25 mm/sec or 50 mm/sec paper speed.

2. Or, by counting the number of beats (R-R intervals) between two sets of marks in the margin of the ECG paper (3 seconds at 50 mm/sec) and multiplying by 20.

Normal feline ECG: 11 beats \( \times 20 = 220 \) beats/minute

Regardless of which system you use it is important to know the correct paper speed!
Canine ECG normal value

**Rate**
- 70 to 160 beats/min for adult dogs
- 60 to 140 beats/min for giant breeds
- Up to 180 beats/min for toy breeds
- Up to 220 beats/min for puppies

**Rhythm**
- Normal sinus rhythm/Sinus arrhythmia
- Wandering sinus pacemaker

**P wave**
- **Width:** maximum, 0.04 sec (2 boxes wide)
  - maximum, 0.05 sec (2½ boxes wide) in giant breeds
- **Height:** maximum, 0.4 mv (4 boxes tall)

**P-R interval**
- **Width:** 0.06 to 0.13 sec (3 to 6½ boxes)

**QRS complex**
- **Width:** maximum, 0.05 sec (2½ boxes) in small breeds
  - maximum, 0.06 sec (3 boxes) in large breeds
- **Height of R wave:** maximum, 3.0 mv (30 boxes) in large breeds
  - maximum, 2.5 mv (25 boxes) in small breeds
S-T segment
No depression: not more than 0.2 mv (2 boxes)
No elevation: not more than 0.15 mv (1 1/2 boxes)

T wave
Can be positive, negative, or diphasic
Not greater than 1/4 amplitude of R wave

Q-T interval
Width: 0.15 to 0.25 sec (7 1/2 - 12 1/2 boxes) at normal heart rate

* Lead II:
  50 mm/sec,
  1 cm = 1 mv
Feline ECG normal value

Rate
120 to 240 beats/min

Rhythm
Normal sinus rhythm
Sinus tachycardia (physiologic reaction to excitement)

P wave
Width: maximum, 0.04 sec (2 boxes wide)
Height: maximum, 0.2 mv (2 boxes tall)

P-R interval
Width: 0.05 to 0.09 sec (2½ to 4½ boxes)

QRS complex
Width: maximum, 0.04 sec (2 boxes)
Height of R wave: maximum, 0.9 mv (9 boxes)

S-T segment
No depression or elevation
**T wave**
Can be positive, negative, or diphasic; most often positive
Maximum amplitude: 0.3 mv (3 boxes)

**Q-T interval**
Width: 0.12 to 0.18 sec (6 to 9 boxes) at normal heart rate

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*Lead II: 50 mm/sec, 1 cm = 1 mv*
Quick & Dirty Guide to ECG Enlargements & Bundle Branch Blocks

- **R wave increased in amplitude = Left Ventricular Enlargement**
- **P wave increased in amplitude or duration = Atrial Enlargement**
- **S wave increased in amplitude = Right Ventricular Enlargement**
- **S wave increased in duration = Right Bundle Branch Block**
Definition of Arrhythmias:
1. An abnormality in the rate, regularity, or site of origin of the cardiac impulse.
2. A disturbance in conduction of the impulse such that the normal sequence of activation of the atria and ventricles is altered.

Classification of Arrhythmias
Abnormalities of impulse formation or impulse conduction are the basis for the following classification:

Normal sinus impulse formation
- Normal sinus rhythm
- Sinus arrhythmia

Disturbances of sinus impulse formation
- Sinus bradycardia
- Sinus tachycardia

Disturbances of supraventricular impulse formation
- Atrial premature complexes
- Atrial tachycardia
- Atrial fibrillation
- Atrioventricular junctional premature complexes
- Atrioventricular junctional tachycardia

Disturbances of ventricular impulse formation
- Ventricular premature complexes
- Ventricular tachycardia
- Ventricular asystole
- Ventricular fibrillation

Disturbances of impulse conduction
- Sinus arrest or block
- Sick sinus syndrome
- Atrial standstill
- Ventricular pre-excitation
- First-degree atrioventricular block
- Second-degree atrioventricular block
- Third-degree atrioventricular block
- Left bundle branch block
- Right bundle branch block
HEART SOUNDS

• First heart sound
  The first heart sound (SI) signals the onset of ventricular systole, is synchronous with the apex beat and is temporally associated with closure of the mitral and tricuspid valves. The area for maximal audibility of the mitral valve in the horse is on the left fifth intercostal space, at a level midway between a horizontal line drawn through the point of the shoulder and one drawn at the sternum at the caudal edge of the triceps muscle. With cattle, sheep, goats and swine the sound is located at a similar level but at the fourth intercostal space.
HEART SOUNDS

• The area for maximal audibility of the tricuspid valve is on the right side of the chest slightly ventral to the equivalent level for the mitral valve and at the fourth intercostal space in the horse; and at the level of the costochondral junction at the third intercostal space for the other species.
Second heart sound

• The second heart sound (S2) is associated with aortic and pulmonic valve closure and is synchronous with the end of systole and the beginning of cardiac diastole. The aortic component is most audible just ventral to a horizontal line drawn through the point of the shoulder and in the left fourth intercostal space in horses and the left third in the other species. The pulmonic component is most audible ventral and anterior to the aortic valve area in the left third intercostal space in horses and the left second or third intercostal space close to the costochondral junction in the other species.
Second heart sound

• These two components of the second heart sound have the same temporal occurrence on auscultation but tonal differences can frequently be detected at the two areas of maximal audibility. Splitting of the second sound in the horse can be detected on phonocardiographic examination but cannot be detected on auscultation and there is no respiratory-associated splitting, as occurs with some other species.
Third heart sound

• The third heart sound (S3) is associated with rapid filling of the ventricle in early diastole and is heard as a dull thudding sound occurring immediately after the second sound. It is usually most audible on the left side just posterior to the area of maximal audibility of the first heart sound. However it is frequently heard over the base and also over the area of cardiac auscultation on the right side. Phonocardiographically there are two components to this heart sound but these are not usually detectable on clinical auscultation.
Third heart sound

• The third heart sound is very common in horses and can be detected in the majority of fit racing animals. It is more audible at heart rates slightly elevated above resting normal. The third heart sound is very common in slightly excited cattle (heart rates 80-100 beats/min) but becomes more difficult to hear when the heart rate exceeds 100 beats/min.
Fourth heart sound

• The fourth heart sound (S4) is associated with atrial contraction. It is also called the 'a' sound. It occurs immediately before the first heart sound and is a soft sound most audible over the base of the heart on the left- and right-hand side. It is also common in horses but its clear separation from the first heart sound is dependent upon the length of the P-R interval, which varies between horses. At resting heart rates the S4 sound is detectable on clinical examination in at least 60% of horses.
Sequence of heart sounds

• The sequence of heart sound occurrence is thus 4-1-2-3. The intensity of the third and fourth sounds is less than that of the first and second and the complex can be described as du LUBB DUP boo. In some horses, the third or fourth sound may be inaudible so that 1-2, 4-1-2 and 1-2-3 variations occur.
Sequence of heart sounds

• The name gallop rhythm is frequently applied when these extra sounds occur. Gallop rhythms also occur in cattle and may be due to the occurrence of a fourth or third sound or to true splitting of the components of the first heart sound. In sheep, goats and pigs only two heart sounds are normally heard. The occurrence of a third or fourth heart sound in horses and cattle is not an indication of cardiovascular abnormality, as it is in other species.