

ECGs and arrhythmia identification

MC Fletcher

Institute of Veterinary, Animal and Biomedical Sciences, Private Bag 11222, Massey University, Palmerston North

Introduction

The electrocardiogram (ECG) was the first piece of monitoring equipment to be made compulsory in human medicine (Moens and Coppens 2011). In veterinary medicine it is an extremely useful pre anaesthetic, perioperative and diagnostic tool. The ECG can provide valuable information about the electrical activity of the heart.

An arrhythmia is any change in rate, rhythm or complex origin that differs from the normal pattern *for that individual patient*. They may be caused by a multitude of compounding factors including; drug administration, inhalational anaesthetic agents, heart disease, thoracic trauma, age, electrolyte and acid base disorders, and/or the type of surgery being performed. Although an arrhythmia may be detected by other means (e.g. auscultation of irregular heart sounds or palpation of an irregular pulse), placing an ECG on a patient is the only way to accurately determine, analyse and treat cardiac arrhythmias.

Electrical activity of the myocardial cell

There are two main types of myocardial cells: working cardiac muscle cells which have the ability to shorten then return to their original length (contract), and the specialised “self-excitatory” myocardial cells involved in the electrical conduction system. The latter have the ability to generate and conduct an electrical impulse, referred to as “automaticity” (Tilley 1992).

For either type of myocardial cell to do work, they need to be electrically charged. The charge is created by the transport of potassium (K^+), calcium (Ca^{2+}) and sodium (Na^+) ions across the cell membrane. The **resting membrane potential** is the voltage that exists across the cell membrane when the cell is at rest. For normal “working” ventricular cells, this equates to an overall negative charge (inside the cell relative to outside the cell) of -90mV. Two transport mechanisms are responsible for maintaining the resting membrane potential: the sodium pump and potassium pump. The sodium and potassium pump uses ATP (energy) and transports ions against their normal energy gradients by pumping Na^+ ions extracellularly and K^+ ions intracellularly (Tilley 1992).

Muscular contraction of the working myocardial muscle cells is triggered by a wave of electrical activity (i.e. a rapid voltage change known as an **action potential**) that sweeps over the cell membrane. The action potential has a **depolarising** phase (where the inside of the cell becomes more positive i.e. less negative) and a **repolarising** phase (where the resting membrane potential returns to normal). In order for an action potential to be generated within a cell (thereby allowing the cell to depolarise and thus contract), the resting membrane potential must change, increasing until it reaches a critical point called the **threshold potential**. Achievement of this critical potential causes a sudden change in the permeability of the cell membrane to Na^+ and K^+ ions. Ions flood into and out of the cell (as described below), generating an action potential and causing the cell to depolarise.

The action potential of a working ventricular myocardial cell is divided into five “phases”, dependent on the movement of ions, and changes in charge, across the cell membrane (Tilley 1992).

- Phase 0 is the period of rapid depolarisation and Na^+ enters the cell.
- Phase 1 is a period of early repolarisation, with a decrease in Na^+ entering the cell and a passive movement of chloride (Cl^-) intracellularly.
- Phase 2 is a slower period of repolarisation, with a slow movement of Ca^{2+} and Na^+ into the cell.
- Phase 3 is rapid repolarisation with K^+ ions rapidly leaving the cell and the cell returning to the negative resting membrane potential. Although repolarisation is complete at the end of phase 3, the ionic distribution across the cell membrane is different to that of just before depolarisation in phase 0.
- In Phase 4 the sodium and potassium pumps are activated transporting K^+ back into the cell and Na^+ out of the cell (Tilley 1992).

The normal cardiac cycle

Some myocardial cells have the ability to generate an electrical impulse on their own (a property known as *automaticity*). These cells are located in the sinoatrial (SA) node, the atrioventricular (AV) node, the bundle of His and the ventricular Purkinje fibres. The highest intrinsic rate (electrical firing rate) is found in the SA node cells. As we travel down the electrical conduction pathway the intrinsic rate of the myocardial cells gets slower. Since the SA node has the highest intrinsic rate, it is termed the “pacemaker” of the heart and it controls the heart rate. The heart rate is dependent on autonomic tone. Sympathetic tone increases the heart rate, while parasympathetic tone decreases the rate (Martin 2007).

P, QRS and T waves

The normal electrical activity of the heart is represented by the P, QRS, and T wave and follows a similar sequence in both dogs and cats. The P wave is initiated by the SA node. A wave of depolarisation spreads through the atrial muscle of the right and left atria, reaches the AV node and the atria contract. The atrial muscle mass is small, therefore the P wave is small in amplitude. The P-R interval starts at the beginning of the P wave and ends at the start of the QRS complex. Once the wave of depolarisation has reached the AV node, its conduction slows before it reaches the bundle of His to allow filling of the ventricles (and ensure the ventricles do not contract at the same time as the atria). The QRS complex represents the depolarisation of the ventricles and is the largest positive deflection on the trace. This depolarisation occurs as the electrical activity spreads from the bundle of His, down the right and left bundle branches to the Purkinje fibres and the ventricles contract. The T wave represents the repolarisation (re-charging) of the ventricles, and results in a deflection from the baseline that may be positive or negative in orientation (Tilley 1992, Martin 2007).

Recording the ECG

Any patient that is considered compromised or is having an anaesthetic will benefit from the placement of an ECG monitor. The ECG is a recording of the electrical activity produced by the heart. Because the entire body acts as a conductor of electrical currents, the pattern of electrical activity within the heart can be measured via electrodes attached to the skin. There are several methods available to attach the ECG electrodes: crocodile clips, adhesive pads and transcutaneous needles can all be utilised (Moens and Coppens 2011). Although the electrical activity of the heart can be displayed when any two points on the body are connected by the electrodes, specific placement of the electrodes is needed to generate the classical ECG trace and allow accurate measurement and interpretation (Martin 2007).

Most commonly, a three “lead” ECG is utilised in veterinary patients. The electrical activity is measured between any two of these electrodes – known as **leads** - with the third electrode acting as an “earth” electrode. There are three commonly utilised electrodes or “leads” in veterinary practice:

1. White electrode: Front right elbow or axillary region.
2. Black electrode: Front left elbow or axillary region.
3. Red electrode: Back left inguinal region.

A fourth electrode, (coloured green) is present on some monitors and should be placed on the back right inguinal

region. The monitor or recording device should be set to Lead II. A Lead II trace (reading between the right forelimb and left hind limb electrodes) generates a “classical” ECG and is the most commonly examined trace in small animals.

Patient positioning

Diagnostically speaking, patients should be positioned in right lateral recumbency, but an ECG can be recorded and evaluated in any position (particularly useful in our anaesthetised patients). The ECG electrodes have specific positions on the body to attach them to, as outlined above. A conductive gel, ultrasound gel or alcohol spray is used to create better contact with the patient’s skin and limit interference on the trace. All electrical equipment can interfere with the ECG and cause artefacts. Placing the patient on a thick towel or rubber mat and turning off electrical appliances and equipment at the wall socket may help reduce this (Martin 2007). Positioning the electrodes away from the chest wall and extending the legs away from the body can help reduce respiratory motion or shivering artefacts.

Paper speed

There are two commonly utilised paper speeds, 25 or 50mm/second. The slower speed allows more complexes to be seen on the paper, allowing for better analysis between beats. The faster speed makes it easier to measure and evaluate each complex. The “gain” (size) is usually set at 1cm = 1 millivolt (mV), although the ECG tracing can be made larger or smaller by increasing or decreasing the gain.

Interpreting the ECG

After recording for a suitable length of time to allow analysis of the electrical rhythm, interpretation and evaluation can begin. There are six steps to ECG interpretation:

1. Evaluate the rate.
2. Evaluate the rhythm.
3. Analyse and identify the P wave.
4. Analyse and identify the QRS complex.
5. Analyse the distance and relationship between the P and QRS complex.

The sixth step is naming the rhythm or arrhythmia. It is important to remember to follow these steps every single time an ECG recording is analysed as skipping steps may lead to a wrong analysis or interpretation.

Step 1: Evaluating the rate

The 10 or 20 method and the “6 second” rule

A quick look over the recorded strip should highlight immediately if the rate is fast (tachycardic), slow (bradycardic), or about normal for a given patient’s age, species and breed. The actual rate should then be determined. There are several simple methods for obtaining a heart rate. Some of the simplest and most effective methods are the “10 or 20 method” and the “6 second” rule. On most types of ECG recording paper there are markers printed. Each marker represents one second at a paper speed of 25mm/second. If six seconds is isolated from the recording, the number of “R to R” intervals can be counted in it and multiplied by 10 to get a heart rate in beats per minute. Likewise, if three seconds is isolated on the recording and the R to R intervals counted, the number can be multiplied by 20 to get a heart rate (Hudson 2009). At a paper speed of 50mm/second 10 large boxes represents 50mm (or one second). A heart rate can be determined by counting the R to R interval in 60 large squares (6 seconds). Most types of ECG paper mark off every 7.5cm along the upper margin. These marks correspond to a three second interval at a paper speed of 25mm/sec, and a 1.5 second interval at a speed of 50mm/sec.

Step 2: Evaluate the rhythm

Looking at the rhythm may give us clues as to whether an arrhythmia is occurring. The rhythm is analysed by looking at the distance between each R wave, and analysing the “R to R interval”. The rhythm of the heart rate can be described as being *regular*, *regularly irregular* (which is the case in a sinus arrhythmia), or *irregularly irregular* (hinting that other types of arrhythmia are occurring).

Step 3: Analyse and identify the P wave

The morphology of the P wave and its timing in the cardiac cycle should be analysed. A normal P wave should be a small positive deflection from the baseline. P waves in the dog and cat should be less than 0.04 seconds in duration, and have an amplitude of less than 0.4mV in the dog, and less than 0.2mV in the cat (Martin 2007). Abnormal P waves include negative P waves, P waves of differing sizes, missing P waves, or P waves occurring earlier or later than expected in the cardiac cycle. (Hudson 2009).

Step 4: Analyse and identify the QRS complex

The morphology of the QRS complex and its timing in the cardiac cycle should be analysed. A normal QRS complex should be positive in amplitude. A normal duration is less than 0.05 seconds in the dog, and less than 0.04 seconds in the cat (Martin 2007). Abnormal QRS complexes are usually wider than normal, indicating the electrical activity is taking longer to pass through the ventricles. The complexes are often bizarre looking, and may appear larger in amplitude than the normal QRS for an individual patient. For this reason, abnormal ventricular complexes can often be termed “big, broad and bizarre”.

Step 5: Analyse the distance and relationship between the P and QRS complex

The distance between the P and R wave represents the slowing of conduction through the AV node (which allows complete filling of the ventricles before they are depolarised). Every complex should be checked for the association between the P wave and the QRS complex. The normal association is for every P wave to be followed by a QRS complex, and likewise for each QRS complex to be preceded by a P wave. The P-R interval should remain consistent, and be between 0.06-0.13 seconds in the dog, or 0.05-0.09 seconds in the cat (Martin 2007).

Common cardiac arrhythmias

Atrial premature complexes (APCs)

When an APC occurs the atrium is depolarised early in the cardiac cycle. The rhythm is classified as irregularly irregular. The premature P wave is usually smaller than that of a normal P wave as it most commonly does not originate from the SA node, but from some other ectopic focus within the atria. It may be positive or negative in amplitude, and may be superimposed in the T wave of the previous complex. The QRS and T wave after the premature P wave usually appear normal and follow the correct electrical pathway through the heart muscle. The complex may be followed by a compensatory pause if the premature P wave occurs very early in the cardiac cycle (Tilley 1992).

AV node blocks

AV node blocks occur when the normal wave of depolarisation is unable to pass into the bundle of His and the ventricles because of a “block” in the AV node. The block can vary from a partial block (first and second degree AV block) to a full block (third degree or complete AV block).

- **First degree AV block:** The P-R interval is prolonged, but the P wave, QRS complex and T wave all appear normal in morphology (Martin 2007).
- **Second degree AV block:** The electrical impulse is intermittently unable to pass through the AV node, resulting in an occasional P wave that is not followed by a QRS complex (atrial depolarisation that is not followed by ventricular depolarisation). The rhythm is classified as irregularly irregular and a second degree AV block can be classified further as either Mobitz type 1 (when the P-R interval progressively lengthens as the electrical activity finds it harder and harder to get across the AV node) or Mobitz type 2, when the P-R interval remains

constant (Martin 2007). Second degree Mobitz type 2 blocks are commonly seen in horses under anaesthesia, or small animals that have received high dose opioids or alpha 2 agonists. They can range from intermittent and mild to frequent and severe impacting significantly on cardiac output and blood pressure.

- **Third degree (complete) AV block:** A complete block occurs when the normal electrical impulse fails to get through the AV node completely. A second “pacemaker” below the block is activated as a rescue mechanism to allow the ventricles to depolarise (although this new ventricular “heart rate” will be much slower than normal due to the slower-than-normal intrinsic rate of the new pacemaker). The P waves and the QRS complexes are completely dissociated from each other, with them only aligning in a normal looking “P, QRS and T wave” by coincidence. The ventricular pacemaker may be from the AV node below the block, the bundle of His or the ventricular bundle branches, resulting in a relatively normal QRS morphology. An abnormal morphology and much broader QRS complex is produced when the rescue pacemaker is located in the purkinje fibres (Martin 2007). A patient presenting with a third degree AV block would be markedly bradycardic on clinical examination. Most third degree blocks are caused by heart disease, and due to the severity of the disease process the patient may require a pacemaker to be implanted.

Ventricular premature complexes (VPCs)

A VPC occurs when the ventricles depolarise early in the cardiac cycle. The rhythm is commonly classified as irregularly irregular. The abnormal beat can originate from any ectopic focus within the ventricles, and can therefore take multiple different morphologies. There is no P wave before the abnormal QRS complex, and the shape of the complex is generally “big, broad and bizarre”. There may be a compensatory pause after the complex if it occurred very early in the cardiac cycle. The T wave is usually large and in the opposite orientation to the abnormal QRS complex (Martin 2007). A single ectopic focus producing one VPC morphology is called a “unifocal” VPC, two foci are called “bifocal VPCs” and three or more foci are called “multi focal VPCs”. Treatment is usually only warranted if the VPCs are occurring enough to impact on cardiac output and blood pressure. Intravenous lignocaine is used to “quieten” the area of irritation in the heart, allowing the normal sinus rhythm to take over again.

Ventricular tachycardia

A run of three or more VPCs occurring in a row is referred to as ventricular tachycardia. The rate is fast and the rhythm is irregularly irregular. Normally, the abnormal QRS complex is unifocal in appearance; however, multimorphic or multifocal ventricular tachycardia is possible (Martin 2007). P waves may still be present on the ECG trace, but they are often hidden in the abnormal ventricular complexes. Sustained ventricular tachycardia may result in a seemingly regular rhythm with regular heart sounds on auscultation and a regularly palpable pulse. The pulse will become weaker and weaker the faster the ventricular tachycardia is occurring. Intermittent ventricular tachycardia is associated with an irregularly irregular rhythm. Lignocaine intravenously can be used to treat ventricular tachycardia.

Sinus arrests

A sinus arrest occurs when the SA node temporarily fails to initiate the normal electrical conduction, resulting in a long pause in electrical activity. If the sinus arrest is longer than a normal sinus cycle for that patient a ventricular “rescue” beat will occur: this is called a ventricular escape beat. The ECG recording will show a long pause of no electrical activity (a flat line) after a T wave, followed by either a normal P, QRS and T wave or a ventricular escape beat. The rhythm may be regularly irregular or irregularly irregular, and on auscultation have long pauses between beats (Martin 2007). Vagal nerve stimulation and sick sinus syndrome are common causes for sinus arrest. An anticholinergic (atropine or glycopyrrolate) may be indicated if the arrests are impacting on cardiac output and/or blood pressure.

Ventricular escape beats

A ventricular escape beat or rhythm is a “rescue” mechanism of the heart that may occur in response to severe bradycardia or sinus arrest. If they did not occur, death would most likely result, hence their other name “rescue” beats. The ventricular escape beat may look identical to a classical VPC highlighting the need for accurate interpretation and diagnosis before any anti-arrhythmic treatment is initiated. Under no circumstances should an escape beat be suppressed by anti-arrhythmic drugs (Tilley 1992).

Ventricular fibrillation

Ventricular fibrillation is associated with cardiac arrest, and is nearly always a terminal event. The ventricles depolarise at random. There is no co-ordinated contraction and instead a chaotic and bizarre trace is observed with no discernible P, QRS or T waves present. The ventricles quiver rather than contract and therefore have little or no pumping ability. There are no audible heart sounds, no palpable pulse and no cardiac output. There are two types of ventricular fibrillation: coarse or fine. Coarse ventricular fibrillation has large oscillating waves while fine has small oscillations. Ventricular fibrillation may follow sustained ventricular tachycardia or VPCs that occur very early in the cardiac cycle or before the ventricles have repolarised fully (called “R on T” phenomenon). Treatment includes cardiopulmonary resuscitation, and if available, electrical defibrillation (Tilley 1992, Martin 2007).

Asystole

This is the classic “flat line” rhythm. If no escape rhythm develops, and there is no electrical activity of any kind on the ECG recording then asystole has occurred. This is a terminal event unless electrical activity begins again.

Conclusion

ECGs are a simple monitoring tool that allow rapid assessment of heart rate and rhythm, and diagnosis and appropriate treatment of various arrhythmias. Whenever possible, they should be utilised in the compromised patient or patients undergoing anaesthesia. An understanding of normal cardiac activity is necessary to read and correctly interpret an ECG trace. Arrhythmias are common and may be a normal (e.g. sinus arrhythmia) or abnormal finding. Although there are many causes of cardiac arrhythmias, common arrhythmias ultimately arise from one of the following abnormalities:

- An abnormality in pacemaker rhythm,
- A shift of the pacemaker from the SA node to another site in the heart,
- A block in the normal conduction pathway,
- The development of an abnormal conduction pathway, or
- The spontaneous generation of an abnormal impulse from almost any place in the heart (because all myocardial cells are excitable).

Careful diagnosis and interpretation must first take place before any form of anti-arrhythmic agent can be administered as treatment.

References

- Hudson D.** *Anesthetic complications: Arrhythmias*. VSPN online continuing education course, May 2009
- Martin M.** *Small animal ECG's an introductory guide (Second edition)*. Blackwell Publishing Ltd, Oxford UK, 2007
- Moens Y, Coppens P.** Patient monitoring and monitoring equipment. In: Seymour C; Duke-Novakovski T (eds). *BSAVA manual of canine and feline anaesthesia and analgesia (Second Edition)*. 61-78, British Small Animal Veterinary Association, Gloucester, England, 2011
- Tilley LP.** *Essentials of canine and feline electrocardiography, interpretation and treatment (third edition)*. Lea and Febiger, USA, 1992