

See discussions, stats, and author profiles for this publication at: <https://www.researchgate.net/publication/254738470>

# ECG interpretation in the horse

Article *in* In Practice · September 2001

DOI: 10.1136/inpract.23.8.454

---

CITATIONS

9

---

READS

4,979

1 author:



Nicola Menzies-Gow

Royal Veterinary College

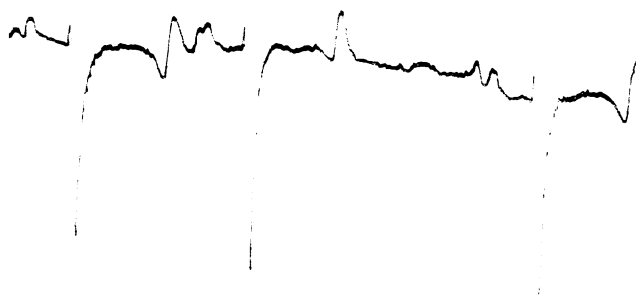
84 PUBLICATIONS 1,085 CITATIONS

SEE PROFILE

Some of the authors of this publication are also working on these related projects:



Predisposition to EMS-associated laminitis [View project](#)



If an arrhythmia is suspected on cardiac auscultation, electrocardiography will enable a definitive diagnosis to be made and the need for therapy to be determined

## ECG interpretation in the horse

NICOLA MENZIES-GOW



**Nicola Menzies-Gow** graduated from Cambridge in 1997 and worked in equine practice in Essex for three years. In March 2000, she joined the Royal Veterinary College as a senior training scholar in equine medicine.

**CARDIAC** arrhythmias are common in horses, although the majority do not require antiarrhythmic therapy. If an arrhythmia is suspected on cardiac auscultation, electrocardiography will enable a definitive diagnosis to be made. However, in each case, the significance of the electrocardiographic findings must be interpreted in the light of the clinical signs exhibited by the horse. This involves evaluation of the cardiac rate and rhythm and detection of the presence of abnormal complexes. Unlike in small animals, little information can be obtained about enlargement of the cardiac chamber(s) in horses. This is because simultaneous depolarisation of the entire ventricular myocardium via the extensive Purkinje system means that the amplitude of the QRS complex is not related to chamber size. This article describes the key features which may be present on an electrocardiographic trace and discusses how these might be interpreted.

### OBTAINING AN ECG

The resting equine electrocardiogram (ECG) is conventionally recorded using a base-apex system. This results in large, easy-to-read complexes that are relatively unaffected by movement artefact. Conventionally, for a base-apex ECG, the right arm (red or negative) electrode is placed over the right jugular groove, while the left arm (yellow or positive) electrode is placed over the left heart base. The right leg (black or earth) electrode is placed at any convenient location, ideally distant from the heart; if an elastic surcingle is used around the thorax to support the wires and electrodes, then it may be easier to position this electrode under it. However, the precise positioning of the electrodes is not critical.

The ECG is recorded with the electrocardiograph switched to the settings for 'lead I' to record from the right arm lead to the left arm lead. This is not, however, a recording of lead I of the Einthoven triangle in the frontal plane. A paper speed of 25 mm/second is generally used. A faster speed may be necessary if tachycardia is present, while a slower speed may be useful for intermittent arrhythmias. The standard deflection used is 10 mm/mV, but 5 mm/mV may be preferable in order to fit large QRS complexes onto the paper.

Radiotelemetry and a Holter monitor can also be used to obtain a resting, exercising or ambulatory ECG by placing electrodes (three for radiotelemetry and two for Holter monitoring) in a vertical configuration under a girth. However, this results in a P wave of smaller amplitude.

### INTERPRETATION OF THE ECG

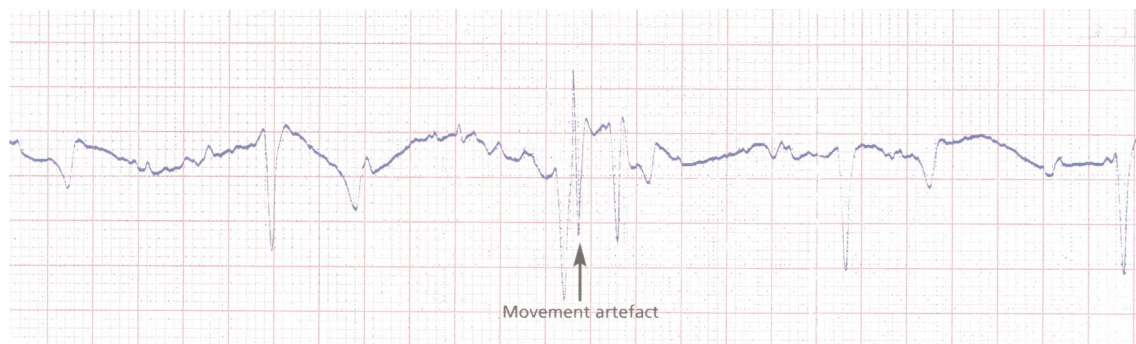
#### ECG QUALITY

The recorded ECG should be assessed in terms of its diagnostic quality. The paper speed, the amplitude of the deflection, whether or not the filter was on, and the presence of artefacts should be noted. The most common artefacts are due to movement of the horse or the electrode leads or clips, or due to poor electrical contact which results in AC interference with many machines. If resentment of the use of clips leads to excessive movement, then alternatives such as adhesive electrodes are equally acceptable.

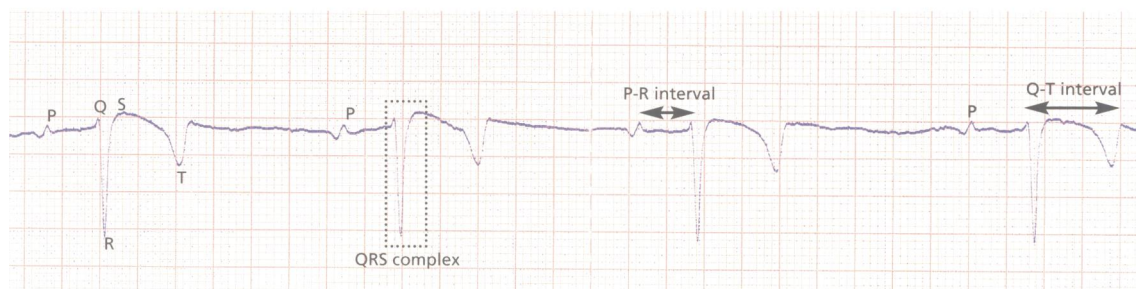
Movement artefacts are seen as sharp deflections of the baseline, which are haphazard but may occasionally resemble a QRS complex. However, the apparent QRS complex will not be followed by a T wave. Alternatively, there may be large undulations in the baseline caused by respiratory movement. Interference from electrical mains may also cause artefacts, and is seen as regular waves at a frequency of 50 Hz.

#### HEART RATE

The heart rate should be calculated. At a paper speed of 25 mm/second, each small box represents an interval of 0.04 seconds and each large box represents 0.2 seconds. The heart rate can be calculated by dividing 60 by the R-R interval in seconds, or by counting the number of complexes in a known time period. A normal heart rate ranges from 28 to 44 beats/minute (bpm); a horse with



ECG from a normal horse demonstrating movement artefact



ECG from a normal horse illustrating P wave, QRS complex and T wave morphology

tachycardia will have a heart rate of more than 44 bpm, while bradycardia results in a heart rate of less than 28 bpm.

## RHYTHM

The rhythm of the ECG should be assessed to determine whether it is regular or irregular. If it is irregular, it should be examined to establish whether or not there is an obvious cyclical pattern which may be predictable, and whether the arrhythmia is intermittent or continuous.

## COMPLEXES

The shape, duration, amplitude and interval duration of each complex should be assessed.

■ THE P WAVE represents atrial depolarisation and is a small deflection of a few millivolts. The normal morphology is very variable. It may be notched (biphasic) or have two separate deflections (biphasic), but is usually positive. Successive P waves are often not identical in the normal horse (so-called wandering pacemaker).

■ THE QRS COMPLEX is usually larger (up to 3 to 4 mV) and represents ventricular depolarisation. The first negative deflection is the Q wave, the first positive deflection is the R wave, and the second negative deflection is the S wave.

■ THE T WAVE follows the QRS complex and represents ventricular repolarisation. It is very variable in size and orientation which means that T wave morphology cannot be reliably used as an indicator of cardiac disease, electrolyte disturbance or systemic disease.

The principal purpose of evaluating interval duration is to determine conduction disturbances. Normal values are given in the table below.

### DURATIONS OF ELECTROCARDIOGRAPHIC COMPLEXES AND INTERVALS IN THE BASE-APEX LEAD IN NORMAL HORSES

	Duration (seconds)
P wave	< 0.16
P-R interval	< 0.5
QRS complex	< 0.14
Q-T interval	< 0.6

## COMPLEX RELATIONSHIPS

The relationship between complexes should be assessed. For example, it should be determined whether a QRS complex follows each P wave, and whether each QRS complex is preceded by a P wave.

## ARRHYTHMIAS

Cardiac arrhythmias are classified according to where they originate from (for instance, supraventricular arrhythmias originate in the atria, atrioventricular node or proximal junctional tissue, while ventricular arrhythmias originate in the ventricle) and their rate (ie, whether they are bradyarrhythmias or tachyarrhythmias). Alternatively, they can be grouped according to whether the arrhythmia is physiological or pathological. Generally, in horses, most bradyarrhythmias are physiological and are associated with high resting vagal tone, while most tachyarrhythmias are pathological.

Isolated abnormal beats can be classified as premature or escape complexes (ie, occur early or late) and according to whether they occur singly or in runs of more than four (ie, paroxysms).

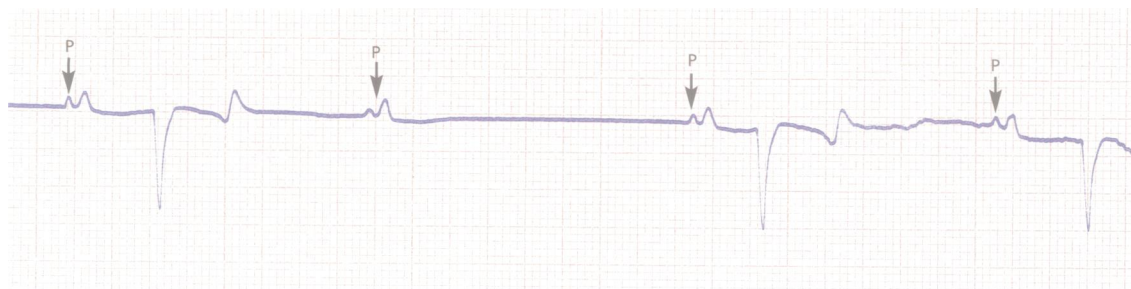
## BRADYARRHYTHMIAS

Bradyarrhythmias usually occur as a result of high resting vagal tone and should disappear following decreased parasympathetic tone or increased sympathetic tone (eg, as a result of exercise, excitement or nervousness).

### Second degree atrioventricular block

Second degree atrioventricular block – the most commonly detected equine bradyarrhythmia – is physiological and disappears with exercise. The heart rate is slow to normal (ie, 20 to 40 bpm) and the rhythm regular with intermittent pauses. The complexes are all normal in appearance; however, the normal rhythm is regularly interrupted by a normal P wave that is non-conducted and therefore not followed by a QRS complex. Most commonly, there is only one period of block before a normal P-QRS-T occurs, although a second P wave may also be blocked.





**ECG demonstrating second degree atrioventricular block with one non-conducted P wave**



**ECG showing second degree atrioventricular block with two successive non-conducted P waves**

### **Advanced second degree and third degree atrioventricular block**

In advanced second degree atrioventricular block, the heart rate is slow to normal, with normal QRS complexes interspersed by periods where P waves are not followed by QRS complexes. The P-P interval is regular. However, the periods of atrioventricular block are prolonged (ie, last for more than two P-P intervals) and do not disappear at exercise.

Third degree atrioventricular block is characterised by complete dissociation of the atria and ventricles. The atrial rate is usually rapid, while the ventricular rate is slow and independent of the atria. The appearance of the QRS complexes will depend on their site of origin. If they are supraventricular and originate from an idionodal pacemaker, they will have a normal QRS configuration. However, those that originate from an idioventricular pacemaker will be wide and bizarre in appearance. If the QRS complexes all originate from the same pacemaker, they should be similar in appearance and regular in their rhythm. None of the QRS complexes are associated with a P wave and the P-R interval will vary. The independent atria result in a regular P-P interval, but many more P waves than QRS complexes.

Both advanced second degree and third degree atrioventricular block may be caused by underlying electrolyte imbalances, digitalis toxicity or atrioventricular nodal disease (inflammatory or degenerative) and treatment is necessary.

**ECG showing third degree atrioventricular block with non-conducted P waves and wide and bizarre QRS complexes originating from the ventricles**



### **Sinus arrhythmia and sinus bradycardia**

Sinus arrhythmia and sinus bradycardia are physiological arrhythmias and are associated with high resting vagal tone. Sinus arrhythmia is seen as a normal heart rate, but the rhythm varies with the R-R interval cyclically increasing and decreasing. Sinus bradycardia is seen as a slow heart rate with a regular rhythm. The R-R interval is prolonged. In both cases, the P wave, QRS complex and T wave morphologies and associations are normal.

### **Sinoatrial arrest/block**

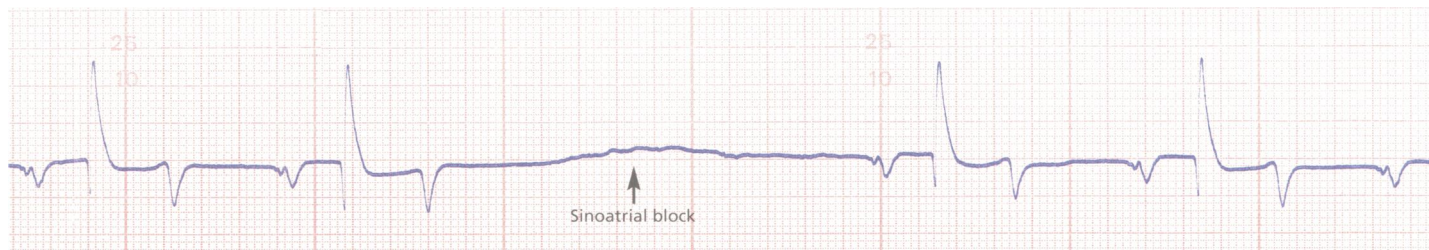
Sinoatrial arrest/block is a vagally mediated arrhythmia which results in a slow to normal heart rate and a regular rhythm with pauses. However, if there is concurrent sinus arrhythmia, then the rhythm may not be regular. The QRS complex configuration is normal and is associated with a preceding P wave. The P-P interval is regularly irregular and the R-R interval is regularly irregular with pauses equal to (block) or greater than (arrest) two P-P intervals.

### **TACHYARRHYTHMIAS**

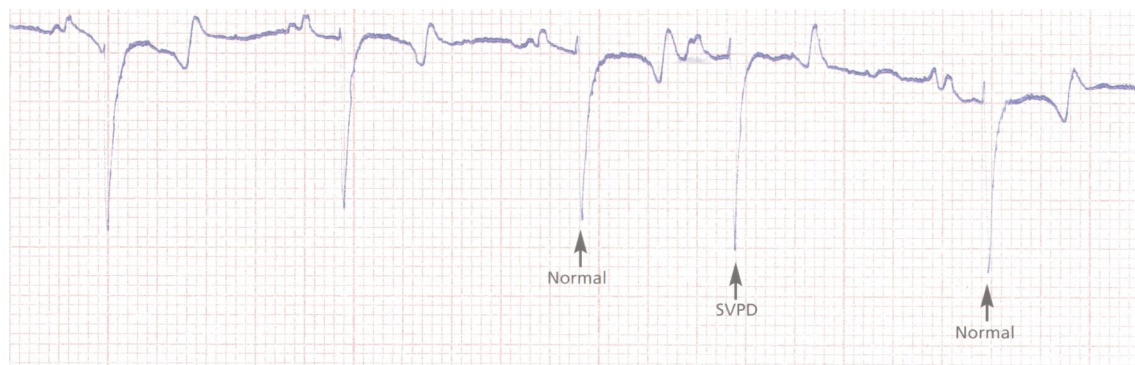
#### **Supraventricular premature depolarisations and tachycardia**

Supraventricular premature depolarisations (SVPDs) are the result of electrical activity that originates in the atria, atrioventricular node or the proximal junctional tissue before sinoatrial nodal discharge that may or may not be conducted to the ventricles. The ECG reveals a regular

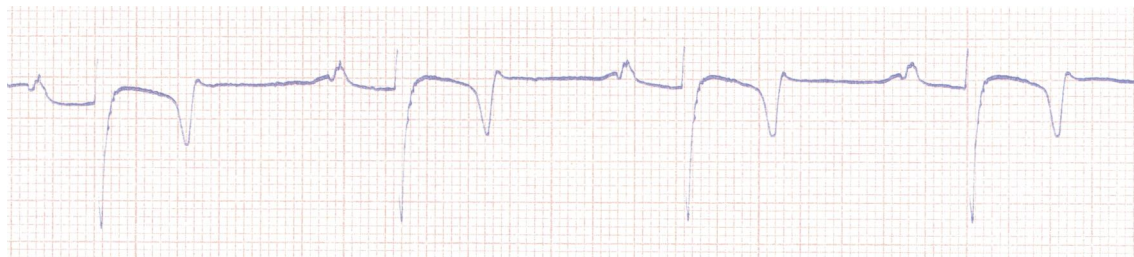




ECG showing sinoatrial block with a pause equal to two P-P intervals



ECG showing a supraventricular premature depolarisation (SVPD)



ECG demonstrating sinus tachycardia



ECG showing atrial fibrillation with fibrillatory 'f' waves between normal QRS complexes

rhythm interrupted by a QRS complex, which is normal in appearance, occurring prematurely if the P wave is conducted. If the P wave is not conducted, the ECG may reveal a bizarre P wave; alternatively, the P wave may be hidden in the preceding T wave.

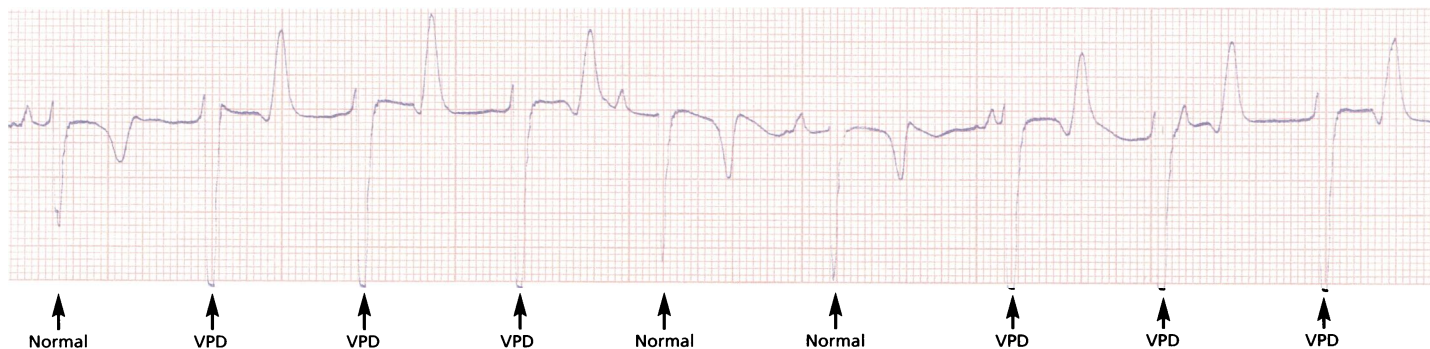
Supraventricular tachycardia (SVT) is defined as more than four premature depolarisations occurring in sequence. Infrequent SVPDs can be detected in normal horses. However, frequent SVPDs or SVT may be indicative of myocardial disease, or may occur in association with atrial enlargement that is secondary to atrio-ventricular valvular insufficiency or congenital heart disease. SVT must be distinguished electrocardiographically from sinus tachycardia. Sinus tachycardia is a normal rhythm that is associated with increased sympathetic tone. The ECG reveals that the heart rate is increased, but the rhythm and waveform are normal.

Sinus tachycardia tends to be gradual in onset, compared with the abrupt onset of SVT, and there is usually an obvious underlying cause (eg, nervousness, excitement or pain).

#### Atrial fibrillation

Horses, especially large breeds, are at an increased risk of developing atrial fibrillation due to their high resting vagal tone and their large atrial size. Atrial fibrillation may therefore occur in horses with no apparent cardiac pathology; alternatively, it may be associated with underlying cardiac disease, such as left atrial enlargement secondary to mitral regurgitation. The heart rate may be slow, normal or rapid and QRS complexes are normal. The R-R interval is irregularly irregular, and P waves are absent. Rapid baseline fibrillation 'f' waves may be seen between the QRS complexes.

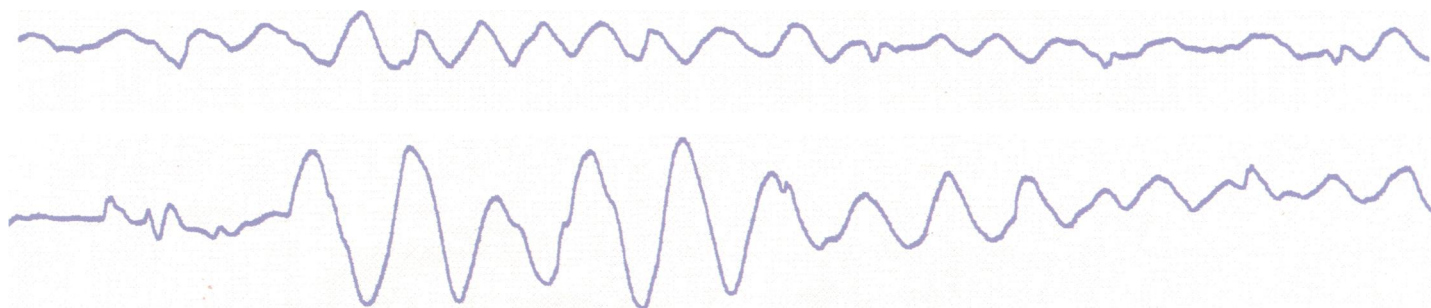




ECG showing wide and bizarre monomorphic ventricular premature depolarisations (VPDs)



ECG showing monomorphic ventricular tachycardia spontaneously converting to normal sinus rhythm



ECG showing wide and bizarre undulations of the baseline with no clear QRS complexes, typical of ventricular fibrillation

### Ventricular premature depolarisations

Ventricular premature depolarisations (VPDs) are the result of abnormal electrical activity originating in the ventricles. A premature QRS complex which is abnormal in appearance interrupts the normal rhythm. Commonly, the abnormal QRS complex is wide and bizarre and followed by a T wave that is orientated in the opposite direction. Multiple VPDs may be identical (ie, monomorphic) or may vary in appearance (ie, polymorphic). Polymorphism implies either that they originate from more than one site within the ventricles or that there is varying conduction from the same focus.

Infrequent VPDs can be detected in normal horses at rest and following exercise. However, if they are frequent, polymorphic or occur during exercise, they are considered to be abnormal. Underlying causes include myocardial inflammation, degeneration, necrosis or fibrosis, electrolyte abnormalities, hypoxia and endotoxaemia.

### Ventricular tachycardia

Ventricular tachycardia is defined as more than four VPDs in sequence and is usually indicative of primary myocardial disease. The ECG reveals a rapid heart rate with numerous wide, bizarre QRS complexes unrelated to

P waves in sequence. The QRS complexes may all be similar (monomorphic) or variable in appearance (polymorphic). The R-R interval is regular in monomorphic ventricular tachycardia, but variable in polymorphic ventricular tachycardia. The P-P interval is regular, but the P waves are often buried in the QRS and T complexes. Polymorphic ventricular tachycardia and the 'R on T phenomenon' (ie, where a QRS complex occurs within the preceding T wave) indicate increased electrical instability; affected horses are at an increased risk of developing ventricular fibrillation.

### Ventricular fibrillation

Ventricular fibrillation results in no clear QRS complexes and there is a bizarre undulating baseline. This rapidly progresses to death.

### SUMMARY

An ECG can be obtained in practice and, in the majority of cases, can be interpreted in order to achieve a definitive diagnosis for an arrhythmia. Possible underlying causes can then be investigated and the need for therapy determined.

### Further reading

PATTESON, M. (1996) *Equine Cardiology*. Ed M. Patteson. Oxford, Blackwell Science  
PATTESON, M. (1999) *Electrophysiology and arrhythmogenesis*. In *Cardiology of the Horse*. Ed C. Marr. Philadelphia, W. B. Saunders. pp 51-71  
REEF, V. B. (1999) *Electrocardiography and echocardiography in the exercising horse*. In *Cardiology of the Horse*. Ed C. Marr. Philadelphia, W. B. Saunders. pp 150-161

In Practice



## ECG interpretation in the horse

Nicola Menzies-Gow

*In Practice* 2001 23: 454-459

doi: 10.1136/inpract.23.8.454

---

Updated information and services can be found at:

<http://inpractice.bmj.com/content/23/8/454>

---

*These include:*

**Email alerting  
service**

Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

---

### Notes

---

To request permissions go to:

<http://group.bmj.com/group/rights-licensing/permissions>

To order reprints go to:

<http://journals.bmj.com/cgi/reprintform>

To subscribe to BMJ go to:

<http://group.bmj.com/subscribe/>