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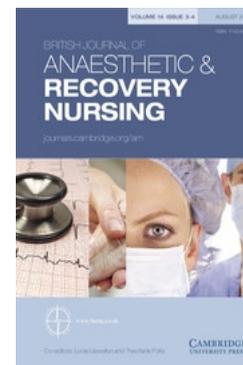
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Arrhythmia interpretation in the perioperative arena (Cont)

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British Journal of Anaesthetic and Recovery Nursing / Volume 3 / Issue 03 / August 2002, pp 12 - 18
DOI: 10.1017/S1742645600001558, Published online: 25 February 2009

Link to this article: http://journals.cambridge.org/abstract_S1742645600001558

How to cite this article:

Christine M. Spiers (2002). Arrhythmia interpretation in the perioperative arena (Cont). British Journal of Anaesthetic and Recovery Nursing, 3, pp 12-18 doi:10.1017/S1742645600001558

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ARRHYTHMIA INTERPRETATION IN THE PERIOPERATIVE ARENA (Cont)

ABSTRACT

This article continues the series on cardiac monitoring in the peri-operative area by reviewing those arrhythmias that are classified as atrial or supraventricular. Whilst these arrhythmias are rarely life threatening, their haemodynamic effects can be significant and may not be tolerated in conjunction with the concurrent operative procedures. These arrhythmias are however, very commonly seen in perioperative practice and may be associated with anaesthetic procedures, surgical techniques and the recovery phase. In this increasingly technological age, it is imperative that perioperative practitioners can recognise and respond appropriately to the rhythms observed on the cardiac monitor.



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Introduction

This article continues the series of arrhythmia interpretation in the peri-operative arena. Peri-operative arrhythmias are common and require appropriate management in the clinical setting. Arrhythmias are most likely to occur in patients with known cardiac disease, although they may be triggered in healthy individuals by a variety of factors such as hypoxia, electrolyte or fluid imbalance and increased catecholamines. The anaesthetic or operative insult may therefore easily initiate an arrhythmia; the physiological impact of the arrhythmia and its subsequent management will be dependent upon the patient's clinical status and their cardiac physiology and function (Hollenberg & Dellinger, 2000).

Cardiac arrhythmias are commonly divided into three groups determined by the site of origin of the arrhythmia; supraventricular (atrial), junctional (nodal) and ventricular. The use of the term 'supraventricular' and 'atrial' are frequently used synonymously, however 'supraventricular' will be used for this article as the term is more embracing and enables a brief review of 'normal' sinus rhythms commonly seen in the peri-operative patient.

Supraventricular arrhythmias are a frequently occurring phenomenon in peri-

operative patients and this article will review: cause, recognition, clinical significance and treatment for each of the arrhythmias identified in Table 1. The goal of management for any arrhythmia will be to establish haemodynamic stability as quickly as possible. In a tachyarrhythmia the aim will be to slow the heart rate and in a bradyarrhythmia, the ventricular response must be restored. Arrhythmias, which do not cause haemodynamic compromise, may not require urgent intervention. (Hollenberg & Dellinger, 2000).

Table 1

Supraventricular arrhythmias

- Sinus tachycardia
- Sinus bradycardia
- Sinus arrhythmia
- Atrial fibrillation
- Atrial flutter
- Premature atrial ectopics
- Atrial tachycardia

Sinus Rhythm

Recall from the first article in this series (Spiers & Stinchcombe, 2002), that the sinoatrial node is the dominant pacemaker and normally initiates each heart beat. Sinus rhythm is the normal rhythm of the heart and can be said to be present when all the component waveforms are present – P wave, PR segment, QRS complex, ST segment and T wave. Sinus rhythm originates in the sinoatrial (sinus) node and is defined as a heart rate between 60 and 100 beats per minute (bpm) in an adult.



Figure 1 Diagram of sinus rhythm to show, P, PR, QRS, ST, T

Sinus tachycardia

Sinus tachycardia is probably the most frequently observed arrhythmia in peri-operative practice. It can be defined as a normal sinus rhythm but at a rate greater than 100 bpm (Riley, 2002). In adults, due to the action of the AV node, the heart rate with sinus tachycardia is generally between 100 – 150 bpm, although it may occasionally achieve rates of up to 180 bpm.

• Recognition

Figure 2 – shows an example of sinus tachycardia. Note that the P waves are positive and each QRS complex is preceded by a P wave. At very fast rates, the P wave may merge with the preceding T wave and hence P waves and T waves may become difficult to distinguish. The QRS rate is regular and fast.

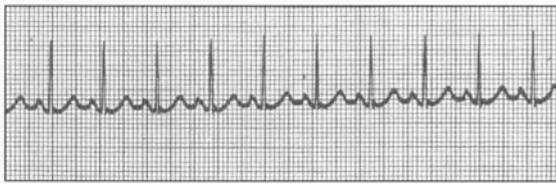


Figure 2 – Sinus tachycardia

• Causes

Sinus tachycardia occurs in any situation that increases sympathetic tone or decreases vagal tone. Thus, it may be considered a physiological response to a variety of situations including:

- Physiological - anxiety, pain, exercise
- Pathological - Fever, anaemia, septic and hypovolaemic shock, thyrotoxicosis
- Intravascular volume loss – vomiting, diarrhoea, bleeding, dehydration
- Pharmacological – epinephrine, salbutamol, pancuronium, atropine

Box 1 Causes of sinus tachycardia

Sinus tachycardia may be induced by drugs that increase sympathetic tone (eg epinephrine, dopamine, cocaine, atracurium and pancuronium) and drugs that decrease vagal tone (eg atropine and other anticholinergic drugs). Sinus tachycardia may also occur as a compensatory response to reduced cardiac output in serious pathological conditions such as left ventricular failure, acute myocardial infarction and pulmonary embolism.

• Clinical significance

Sinus tachycardia will therefore be seen in all areas of peri-operative practice. In the

anaesthetic room the patient may be extremely anxious and emotional and some anaesthetic agents may also induce sinus tachycardia. During the operative period it may result from intravascular volume loss and poor pain relief during surgery. Finally it may also be observed during post-operative recovery and may be an early indication of inadequate analgesia or hypovolaemia (Hatfield & Tronson, 2001).

• Management

As cardiac filling time is reduced at heart rates above 130 bpm, most patients cannot tolerate a sinus tachycardia for prolonged periods of time. Reduced cardiac filling time results in reduced cardiac output, hypotension and syncope, and the conscious patient may complain of significant symptoms. In addition myocardial perfusion will also be reduced during periods of tachycardia and this may result in anginal-type pain due to myocardial ischaemia.

Clinical assessment of the patient with a tachycardia should include; monitoring of trends in BP, CVP, oxygen saturation and if possible, ST segment monitoring. A patient with significant myocardial ischaemia may develop ST segment depression as shown in Figure 3.



Figure 3 – ischaemia induced by sinus tachycardia

In general no specific treatment is offered for sinus tachycardia, but considerable attention should be paid to finding and rectifying the underlying cause, for example managing hypovolaemia, giving adequate analgesia or improving oxygenation.

Sinus bradycardia

In sinus bradycardia, sinus rhythm is present at a heart rate less than 60 bpm. It is a commonly encountered ‘normal variant’-that is it is frequently seen in fit, healthy individuals, particularly during rest or sleep and it is also seen in athletes (Goldberger, 1999).

• Recognition

In Figure 4 note that all the waveforms (P,PR segment, QRS, ST segment, T wave) are present and occur regularly at a slow rate. During sinus bradycardia the pauses between complexes are lengthened and

“considerable attention should be paid to finding and rectifying the underlying cause”

hence the period for cardiac filling is enhanced. Hence at rates between 50 and 60 bpm sinus bradycardia can augment cardiac performance as cardiac output and myocardial perfusion will be optimised.



Figure 4 Sinus bradycardia

• Causes

Sinus bradycardia can occur in any situation that increases vagal tone or decreases sympathetic activity. As previously stated, it is an expected normal variant in fit healthy individuals during sleep and in trained athletes who may have a resting or sleeping heart rate as low as 35 bpm (Goldberger, 1999). It is also observed in elderly patients who may develop sinus bradycardia due to degenerative disease of the sino-atrial node or surrounding conduction tissue.

The most notable cause of sinus bradycardia relates to situations that increase vagal tone. Some anaesthetic procedures can increase vagal tone including endotracheal intubation and endotracheal suctioning. Similarly, rectal, gynaecological and oesophageal surgery can augment vagal tone, as can facial, dental and ophthalmic surgery. Sinus bradycardia is also seen in deep pain and during vomiting. Drugs that cause increased vagal stimulation (notably halothane and digoxin) or decrease sympathetic tone (particularly beta-blockers and calcium channel blockers) may also caused marked sinus bradycardia.

• Clinical significance

Sinus bradycardia will be observed frequently in perioperative practice and may be induced by certain anaesthetic procedures or surgical practices. At heart rates of 50-60 bpm sinus bradycardia may enhance cardiac output by augmenting stroke volume. The patient maintaining an adequate BP and O2 saturation will evidence this, although some patients will not tolerate reduced heart rates and light-headedness, hypotension and syncope may result. Clinical monitoring of BP, O2 saturation and peripheral perfusion is therefore mandatory.

• Management

As with sinus tachycardia, no specific treatment is offered for sinus bradycardia, but in the compromised patient Atropine 1

mg i/v will increase the heart rate quickly. During operative procedures which may enhance vagal tone, extra care should be taken with patient who are already receiving beta-blockers (Atenolol), calcium channel blockers (Diltiazem, Verapamil), or Digoxin to ensure that heart rates do not fall to dangerously low rates.

Sinus arrhythmia

In healthy people the sinus node does not always pace the heart at an entirely regular rate – a slight beat to beat variation is often present. This cyclic variation in sinus rhythm is referred to as ‘sinus arrhythmia’ and it is considered a ‘normal variant’ in young adults (Goldberger, 1999; Wiederhold, 1999).

• Recognition

Sinus arrhythmia is a sinus rhythm with normal P, QRS, ST, and T waves, but it manifests as a slightly irregular rhythm. This irregularity occurs in a regular cycle and hence the rhythm can be said to be ‘regularly irregular’.

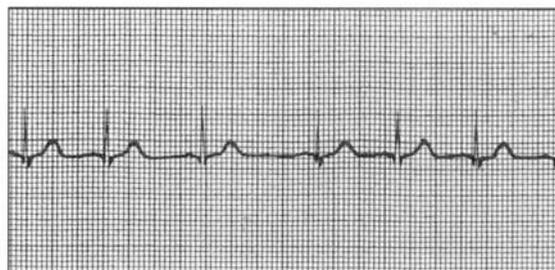


Figure 5 – sinus arrhythmia

• Causes

The cyclic variation in sinus rhythm is commonly associated with respiration. ‘Respiratory sinus arrhythmia’ occurs in response to changing thoracic pressures and is a normal result of changes in vagal tone that occurs during the different phases of respiration. Hence in sinus arrhythmia, the heart rate will increase slightly with inspiration and decrease with expiration. The effect on the heart rate can be quite marked (up to 10-20 bpm) in children and young adults (Riley, 2002).

• Clinical significance

Sinus arrhythmia is generally considered to be a very benign arrhythmia, particularly when observed in young adults and in relation to respiratory cycles. It requires no clinical intervention.

• Management

‘Respiratory sinus arrhythmia’ and sinus arrhythmia seen in young adults is entirely

“Sinus bradycardia is also seen in deep pain and during vomiting”

benign and requires no clinical intervention at all. Very occasionally sinus arrhythmia may be seen in elderly patients and, when it is not associated with the respiratory cycle it may be an early sign of sino-atrial node disease or Sick Sinus Syndrome and a permanent pacemaker may be required (Mangrum & DiMarco, 2000).

SUPRAVENTRICULAR TACHYCARDIAS

The term 'supraventricular tachycardia' can really be considered an *umbrella term* to describe any arrhythmia which arises above the level of the ventricles (Goldberger, 1999). Commonly supraventricular tachycardias can be divided into two distinct groups of arrhythmias, dependent upon whether they arise from the atria or the atrioventricular node/junction (Goodacre & Irons, 2002). Tachycardias, which arise from the atrial tissue, include:

- Atrial fibrillation
- Atrial flutter
- Atrial tachycardia

Tachycardias that arise from the atrioventricular node or junction and are commonly associated with re-entry circuits as seen in patients with the Wolff-Parkinson-White syndrome include:

- Atrioventricular re-entrant tachycardia (AVRT)
- Atrioventricular nodal re-entrant tachycardia (AVNRT)

The scope of this article does not allow consideration of AVRT and AVNRT and the subsequent discussion will consider atrial fibrillation, flutter and atrial tachycardia. Sinus tachycardia has previously been discussed.

Atrial fibrillation

Atrial fibrillation is a ubiquitous and yet diverse cardiac arrhythmia, which for many years was considered a Cinderella arrhythmia. Until recently, the arrhythmia was rarely given much credence, poorly managed and inadequately treated (Allessie et al, 2001; Pepper, 2001). Atrial fibrillation is the most common sustained arrhythmia, its prevalence increases with age and it is frequently associated with structural heart disease. It may however be seen in patients with no known heart disease and its haemodynamic and thromboembolic consequences result in significant morbidity and mortality for affected individuals (Task

Force Report, 2001). The scope of this article does not allow extensive discussion of the management of this arrhythmia, therefore discussion will be focussed upon the emergency management of paroxysmal atrial fibrillation in the peri-operative patient.

• Recognition

Atrial fibrillation results from multiple ectopic foci forming throughout the atria. These ectopic foci override sinus node function and cause the atria to 'fibrillate'. The ectopic foci are observed on the ECG as a wavy baseline or as 'f waves' and these 'f waves' may occur at a very fast rate (up to 600 bpm). The electrical activity generated by the fibrillations 'bombard' the AV node with stimuli. Due to the physiological structure of the AV node, not all impulses are conducted into the ventricles (which would of course be disastrous) and the ventricular response is irregular. Conduction to the ventricles occurs at an irregular rate and may vary from 60 bpm to 180 bpm. Atrial fibrillation is evident in figure 6 and is characterised by a fast irregularly irregular rhythm with no discernible P waves, a wavy baseline, 'f waves' and normal shaped QRS complexes. Due to the fibrillatory waves, the T waves may also become difficult to identify.

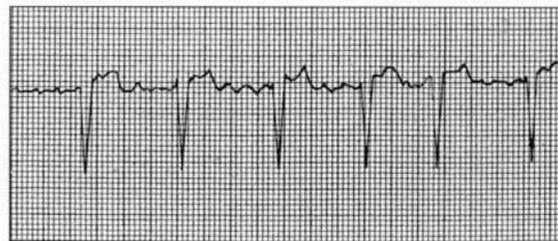


Figure 6 – atrial fibrillation

• Causes

Atrial fibrillation is commonly encountered in patients undergoing major surgical procedures and is frequently encountered in the elderly population. It is particularly prevalent in patients undergoing abdominal aortic aneurysm repair and thoractomy for lung cancer. It is also common in the cardiac surgical patient.

The causes of atrial fibrillation are numerous and are summarised in Box 2. Essentially atrial fibrillation may occur in situations which cause atrial dilatation or stimulation – such as sympathetic stimulation related to anxiety, or fluid overload due to cardiac failure or over-enthusiastic volume loading!

“causes of atrial fibrillation are numerous”

Causes of atrial fibrillation	
• Atrial enlargement – mitral or tricuspid stenosis	
• Coronary heart disease, Cardiomyopathy, Cardiac failure	
• Sympathetic stimulation – anxiety, caffeine, ethanol, anaesthetic agents	
• Hypoxia – COPD, acute pulmonary embolism, respiratory distress	
• Thyrotoxicosis, phaeochromocytoma, major thoracic and abdominal surgery	

Box 2 – Causes of atrial fibrillation

• Clinical significance

Of all the supraventricular tachycardias, atrial fibrillation is by far the most common and has the most potential for serious consequences. Fast ventricular rates in atrial fibrillation can cause significant haemodynamic compromise, atrial fibrillation causes reduced cardiac output, decreased cardiac filling time and loss of atrioventricular synchrony. Conscious patients may experience significant symptoms including palpitations, dyspnoea, syncope and chest pain; the symptoms can be both alarming and distressing. In the unconscious patient, a fall in blood pressure and oxygen saturation herald the need for urgent intervention (Bubien, 2000).

• Management

In uncompromised patients with an episode of atrial fibrillation lasting more than 15 minutes, the aim will be to establish ventricular rate control. This may be achieved pharmacologically by the use of intravenous beta-blockers (Esmolol), calcium-channel blockers (Verapamil), Digoxin or Amiodarone.

Whilst all these drugs will be efficient at achieving significant ventricular rate control (particularly Esmolol and Verapamil) caution should be applied with the use of all these drugs as they have significant contraindications and side effects (see Box 3). Assessment of the patient's medical history is therefore mandatory before pursuing this approach.

Drugs	Contraindications/side effects
Esmolol	Bronchospasm, hypotension, cardiac failure
Digoxin	Delayed onset, nausea, vomiting, diplopia
Amiodarone	Hypotension, heart block
Verapamil	Cardiac failure (extreme caution with patients with reduced systolic function)

Box 3 Intravenous drug therapy for atrial fibrillation

“a fall in blood pressure and oxygen saturation herald the need for urgent intervention”

If the patient is stable and uncompromised, there is time to identify the rhythm and to establish a diagnosis. In a compromised patient with atrial fibrillation, particularly when the ventricular rate is fast (more than 130 bpm), and poor perfusion is evident, prompt electrical conversion may be indicated. The Resuscitation Council (2000) recommends cardioversion using an algorithm of 100J, 200J, 360J as indicated. If atrial fibrillation recurs it is recommended that intravenous Amiodarone 300mg is administered over an hour before cardioversion is reattempted.

Atrial flutter

Atrial flutter is a rare arrhythmia in adults, although it is commonly seen in children with congenital heart disease. It is due to a macro re-entry circuit in the right atrium causing the atria to depolarise at a fast rate of 300 bpm.

• Recognition

Due to the physiological action of the AV node, transmission to the ventricles is blocked in a regular manner. Consequently ‘F’ waves are seen on the ECG (from the macro re-entry circuit) and the ventricular response may be observed as 2:1, 3:1 or 4:1. Hence the ventricular rate may be 150 bpm, 100 bpm or 75 bpm. When 1:1 conduction occurs – theoretically a rate of 300 bpm would be observed. At the slower rates, the ‘F’ waves are easily observed on the ECG as a *sawtooth appearance*.

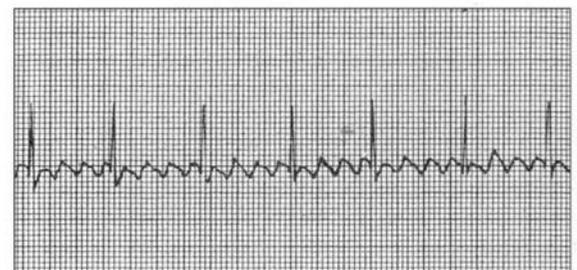


Figure 7 – Atrial flutter

• Causes

The causes of atrial flutter are essentially the same as for atrial fibrillation and occasionally atrial flutter may be idiopathic and occur in the absence of any precipitating factors.

• Clinical significance

Atrial flutter will rarely be observed in the perioperative clinical setting, but its physiological impact will once again be determined by the duration of the arrhythmia, ventricular rate and underlying cardiac function (Hollenberg & Dellinger, 2000).

• Management

The management of atrial flutter is the same as suggested for atrial fibrillation – pharmacological or electrical cardioversion. However remember, management of the patient is principally determined by the patient’s haemodynamic response to the arrhythmia.

Premature atrial complexes (extrasystoles)

Premature atrial complexes (PACs) arise from irritable atrial myocardium causing early depolarisation of the atrial and subsequent early stimulation of the ventricles. They may be considered a *normal variant* and some individuals will have PACs as part of the normal sinus rhythm. Occasionally PACs can precipitate atrial fibrillation or supraventricular tachycardia.

• Recognition

As the electrical stimulus arises from the atrial myocardium (rather than the SA node), the P wave will have an abnormal configuration and will be discernibly different to the normal P waves of the sinus beats. The P-R interval may also be shorter with a PAC, but the ventricular response (QRS) is likely to be normal.

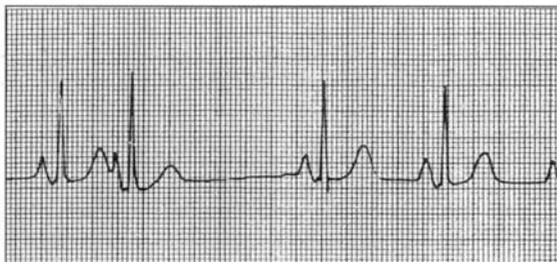


Figure 8 – Premature atrial complexes

• Causes

PACs may be a normal variant or may be triggered by mechanisms that cause sympathetic stimulation (anxiety, pain, hypoxia) and can occur with electrolyte imbalances (hypokalaemia).

• Clinical significance

PACs are generally clinically insignificant as they tend to result in minimal haemodynamic derangement. Occasionally, when PACs occur frequently, and the underlying sinus rhythm is fast, atrial fibrillation or supraventricular tachycardia may be precipitated.

• Management

No treatment is indicated for PACs although efforts should be taken to establish, and if

necessary correct, the electrolyte, fluid balance and O₂ saturation status of the patient.

Atrial tachycardia

Atrial tachycardia can be very simply defined as three or more consecutive PACs arising from an ectopic focus in either the right or left atrium (Goldberger, 1999). The ectopic focus fires at a rate faster than the sinus node and hence tends to trigger a tachycardia in the range of 100-250 bpm.

• Recognition

Atrial tachycardia arises from an ectopic source in either of the atria and produces an atrial rate of 100-250 bpm (slower than atrial flutter). The P waves therefore differ from that seen in sinus rhythm, but do not have the commonly described sawtooth appearance characteristic of atrial flutter.

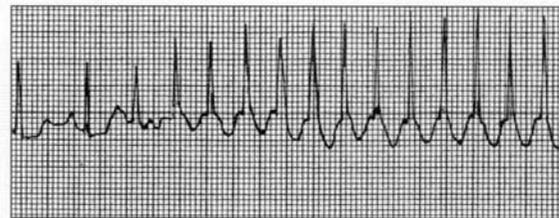


Figure 9 – Atrial tachycardia

• Causes

Atrial tachycardia may be a benign arrhythmia in children, young adults and the elderly. It tends to be paroxysmal in nature and has an abrupt onset and cessation. It may be triggered by sympathetic activation, digoxin toxicity or the stress response. Caffeine, tobacco and alcohol are all common precipitators. Atrial tachycardia may be observed in patients without known structural heart abnormality, but also is seen with virtually any type of heart disease.

• Clinical significance

As with any other supraventricular tachycardia, a rapid rate may induce hypotension, reduced cardiac output, cerebral and myocardial ischaemia, acute cardiac failure and syncope.

• Management

Atrial tachycardia tends to occur in paroxysms and short episodes may not require treatment. Longer episodes, which result in symptoms, may require intervention. Clinical options include; vagal manoeuvres such as carotid sinus massage (caution in elderly patients and those with known vascular disease), electrical

“Atrial tachycardia may be a benign arrhythmia in children, young adults and the elderly”

cardioversion or anti-arrhythmic therapy such as Adenosine.

Conclusion

The first principle of managing arrhythmias is to treat the patient, not the ECG. Therefore, once an arrhythmia is recognised it is imperative that assessment is undertaken to establish whether the patient is compromised by the arrhythmia or not.

The physiological impact of an arrhythmia will depend upon the ventricular rate (too fast or too slow), the duration of the arrhythmia (paroxysmal or sustained) and the pre-existing morbid state of the patient.

Peri-operative arrhythmias are more likely to occur in a patient with known cardiac disease, although arrhythmias may arise in any situation where there is a transient imbalance such as; hypoxia, electrolyte/fluid imbalance, increased catecholamine release or ischaemia. Peri-operative procedures including anaesthetic techniques and surgical interventions can also induce arrhythmias. Finally, arrhythmias may arise in the recovery phase as the anaesthetic and analgesic agents wear off and breakthrough pain is seen.

All arrhythmias may cause haemodynamic disruption and in some patients this may be catastrophic. Conversely some individuals will tolerate sustained arrhythmias with little or no signs or symptoms. **Always remember**, the cardiac monitor is an adjunct to your care, but it is only one of the vast array of assessment tools available to ensure that your patients' journey through their peri-operative experience is a safe one. As anaesthetic and recovery room practice becomes more technological nurses have a professional responsibility to ensure that their practice is evidence-based and founded upon sound physiological principles. Learning to read the ECG is one of the many skills that will be required in the 21st century for all peri-operative practitioners.

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