

ECG MONITORING IN THEATRE

Dr Juliette Lee, Royal Devon And Exeter Hospital, Exeter, UK - Previously: Ngwelezana hospital, Empangeni, Kwa-zulu Natal, RSA

Cardiac arrhythmias during anaesthesia and surgery occur in up to 86% of patients. Many are of clinical significance and therefore their detection is of considerable importance. This article will discuss the basic principles of using the ECG monitor in the operating theatre. It will describe the main rhythm abnormalities and give practical guidance on how to recognise and treat them.

The continuous oscilloscopic ECG is one of the most widely used anaesthetic monitors, and in addition to displaying arrhythmias it can also be used to detect myocardial ischaemia, electrolyte imbalances, and assess pacemaker function. A 12 lead ECG recording will provide much more information than is available on a theatre ECG monitor, and should where possible, be obtained pre-operatively in any patient with suspected cardiac disease.

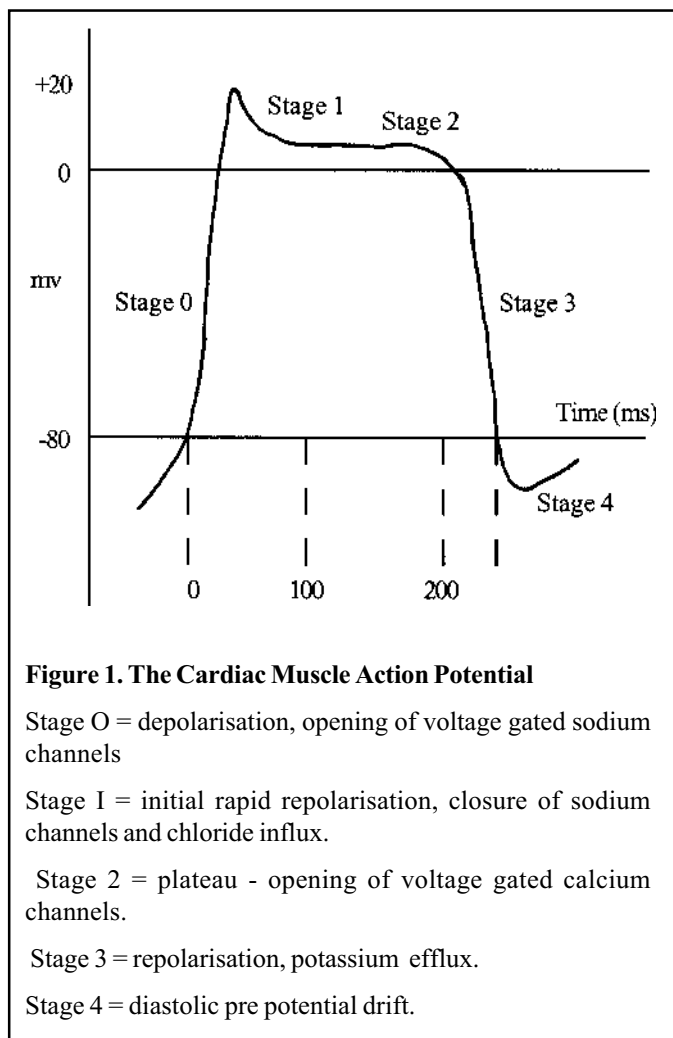
The ECG is a recording of the electrical activity of the heart. It does not provide information about the mechanical function of the heart and cannot be used to assess cardiac output or blood pressure. Cardiac function under anaesthesia is usually estimated using frequent measurements of blood pressure, pulse, oxygen saturation, peripheral perfusion and end tidal CO₂ concentrations. Cardiac performance is occasionally measured directly in theatre using Swan Ganz catheters or oesophageal Doppler techniques, although this is uncommon.

The ECG monitor should always be connected to the patient before induction of anaesthesia or institution of a regional block. This will allow the anaesthetist to detect any change in the appearance of the ECG complexes during anaesthesia.

Connecting an ECG monitor

Although an ECG trace may be obtained with the electrodes attached in a variety of positions, conventionally they are placed in a standard position each time so that abnormalities are easier to detect. Most monitors have 3 leads and they are connected as follows:

- **Red** - right arm, (or second intercostal space on the right of the sternum)
- **Yellow** - left arm (or second intercostal space on the left of the sternum)
- **Black** (or Green) - left leg (or more often in the region of the apex beat.)



This will allow the Lead I, II or III configurations to be selected on the ECG monitor. Lead II is the most commonly used. (See page 18 for other lead positions and their uses).

The cables from the electrodes usually terminate in a single cable which is plugged into the port on the ECG monitor.

A good electrical connection between the patient and the electrodes is required to minimise the resistance of the skin. For this reason gel pads or suction caps with electrode jelly are used to connect the electrodes to the patients skin. However when the skin is sweaty the electrodes may not stick well, resulting in an unstable trace. When electrodes are in short supply they may be reused after moistening with saline or gel before being taped to the patient's chest. Alternatively, an empty 1000ml iv infusion bag may be cut open to allow it to lie flat (in the form of a flat piece of plastic) on the patient's chest. If 3 small

holes are made in 3 of the corners electrodes may be stuck on one side of the plastic allowing the electrode gel to make contact with the skin. This device can be cleaned at the end of the operation and laid on the next patient allowing electrodes to be used repeatedly.

Principles of the ECG

The ECG is a recording of the electrical activity of the heart. An electrical recording made from one myocardial muscle cell will record an action potential (the electrical activity which occurs when the cell is stimulated). The ECG records the vector sum (the combination of all electrical signals) of all the action potentials of the myocardium and produces a combined trace.

At rest the potential difference across the membrane of a myocardial cell is -90mv (figure 1). This is due to a high intracellular potassium concentration which is maintained by the sodium/potassium pump. Depolarisation of a cardiac cell occurs when there is a sudden change in the permeability of the membrane to sodium. Sodium floods into the cell and the negative resting voltage is lost (stage 0). Calcium follows the sodium through the slower calcium channels resulting in binding between the intracellular proteins actin and myosin which results in contraction of the muscle fibre (stage 2). The depolarisation of a myocardial cell causes the depolarisation of adjacent cells and in the normal heart the depolarisation of the entire myocardium follows in a co-ordinated fashion. During repolarisation potassium moves out of the cells (stage 3) and the resting negative membrane potential is restored.

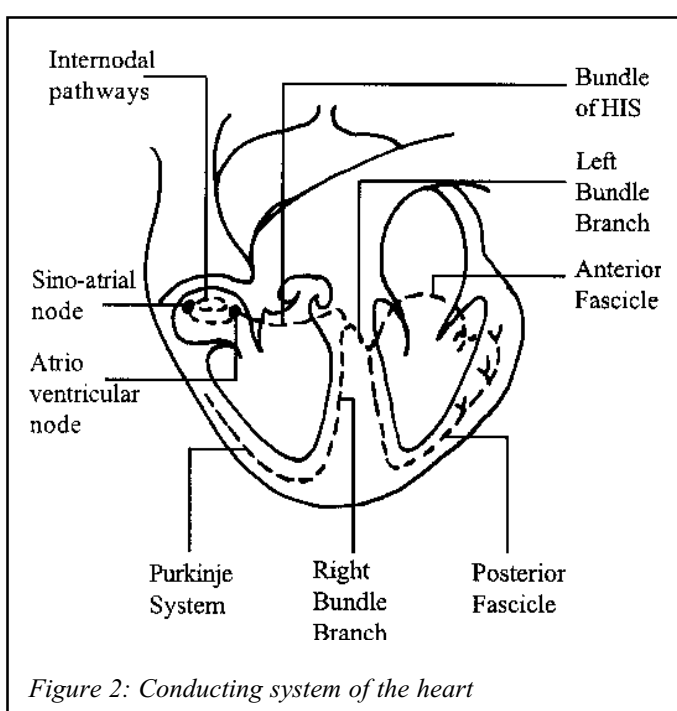


Figure 2: Conducting system of the heart

THE CONDUCTING SYSTEM OF THE HEART

The specialised cardiac conducting system (figure 2) consists of:

The Sinoatrial (SA) node, internodal pathways, Atrioventricular (AV) node, bundle of HIS with right and left bundle branches and the Purkinje system. The left bundle branch also divides into anterior and posterior fascicles. Conducting tissue is made up of modified cardiac muscle cells which have the property of automaticity, that is they can generate their own intrinsic action potentials as well as responding to stimulation from adjacent cells. The conducting pathways within the heart are responsible for the organised spread of action potentials within the heart and the resulting co-ordinated contraction of both atria and ventricles.

In pacemaker tissue, after repolarisation has occurred, the membrane potential gradually rises to the threshold level for channel opening, at which point sodium floods into the cell and initiates the next action potential (figure 3). This gradual rise is called the pacemaker (or pre-potential) and is due to a decrease in the membrane permeability to potassium ions which result in the inside of the cell becoming less negative. The rate of rise of the pacemaker potential is the main determinant of heart rate and is increased by adrenaline (epinephrine) and sympathetic stimulation and decreased by vagal stimulation and hypothermia. Pacemaker activity normally only occurs in the SA and AV nodes, but there are latent pacemakers in other parts of the conducting system which take over when firing from the SA or AV nodes is depressed. Atrial and ventricular muscle fibres do not have pacemaker activity and discharge spontaneously only when damaged or abnormal.

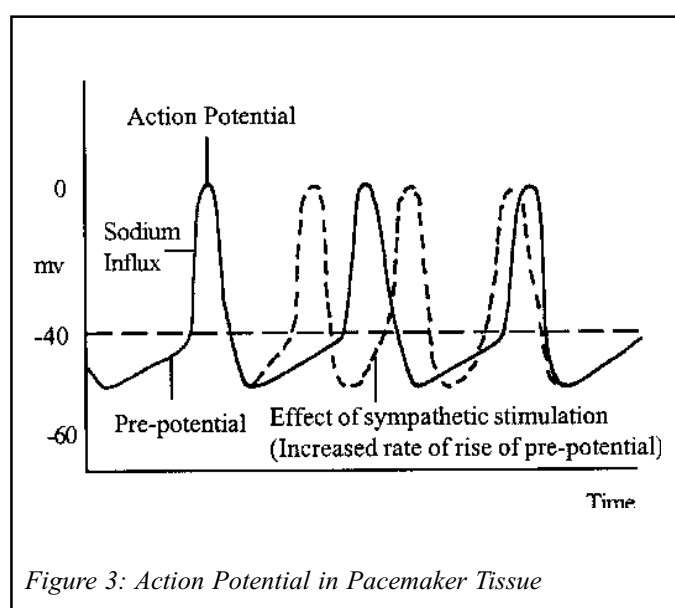


Figure 3: Action Potential in Pacemaker Tissue

GRAPHICAL RECORDING

On a paper trace the ECG is usually recorded on a time scale of 0.04 seconds/mm on the horizontal axis and a voltage sensitivity of 0.1mv/mm on the vertical axis (figure 4). Therefore, on standard ECG recording paper, 1 small square represents 0.04seconds and one large square 0.2 seconds. In the normal ECG waveform the P wave represents atrial depolarisation, the QRS complex ventricular depolarisation and the T wave ventricular repolarisation.

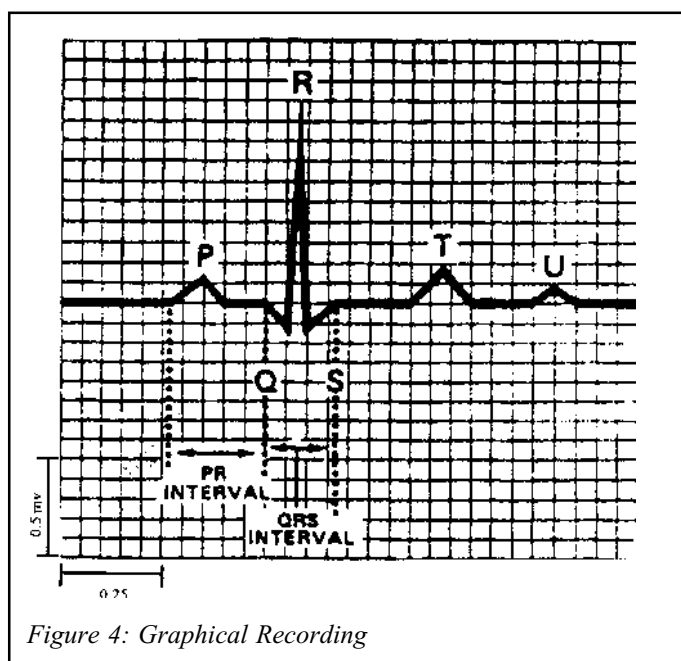


Figure 4: Graphical Recording

- The P- R interval is taken from the start of the P wave to the start of the QRS complex. It is the time taken for depolarisation to pass from the SA node via the atria, AV node and His - Purkinje system to the ventricles.
- The QRS represents the time taken for depolarisation to pass through the His - Purkinje system and ventricular muscles. It is prolonged with disease of the His - Purkinje system.

- The Q -T interval is taken from the start of the QRS complex to the end of the T wave. This represents the time taken to depolarise and repolarise the ventricles.
- The S - T segment is the period between the end of the QRS complex and the start of the T wave. All cells are normally depolarised during this phase. The ST segment is changed by pathology such as myocardial ischaemia or pericarditis.

LEAD POSITIONS

The ECG may be used in two ways. A 12 lead ECG may be performed which analyses the cardiac electrical activity from a number of electrodes positioned on the limbs and across the chest. A wide range of abnormalities may be detected including arrhythmias, myocardial ischaemia, left ventricular hypertrophy and pericarditis.

During anaesthesia, however, the ECG is monitored using only 3 (or occasionally 5) electrodes which provide a more restricted analysis of the cardiac electrical activity and cannot provide the same amount of information that may be revealed by the 12 lead ECG.

The term 'lead' when applied to the ECG does not describe the electrical cables connected to the electrodes on the patient. Instead it refers to the positioning of the 2 electrodes being used to detect the electrical activity of the heart. A third electrode acts as a neutral. During anaesthesia one of 3 possible 'leads' is generally used. These leads are called bipolar leads as they measure the potential difference (electrical difference) between two electrodes. Electrical activity travelling towards an electrode is displayed as a positive (upward) deflection on the screen, and electrical activity travelling away as a negative (downward) deflection. The leads are described by convention as follows:

ECG Normal Values

P - R interval	0.12 - 0.2 seconds (3-5 small squares of standard ECG paper)
QRS complex duration	less than or equal to 0.1 seconds (2.5 small squares)
Q - T interval	less than or equal to 0.44 seconds
corrected for heart rate (QTc)	
QTc = QT/RR interval	

- **Lead I** - measures the potential difference between the right arm electrode and the left arm electrode. The third electrode (left leg) acts as neutral.
- **Lead II** - measures the potential difference between the right arm and left leg electrode.
- **Lead III** - measures the potential difference between the left arm and left leg electrode.

Most monitors can only show one lead at a time and therefore the lead that gives as much information as possible should be chosen. The most commonly used lead is lead II (figure 5) - a bipolar lead with electrodes on the right arm and left leg as above. This is the most useful lead for detecting cardiac arrhythmias as it lies close to the cardiac axis (the overall direction of electrical movement) and allows the best view of P and R waves.

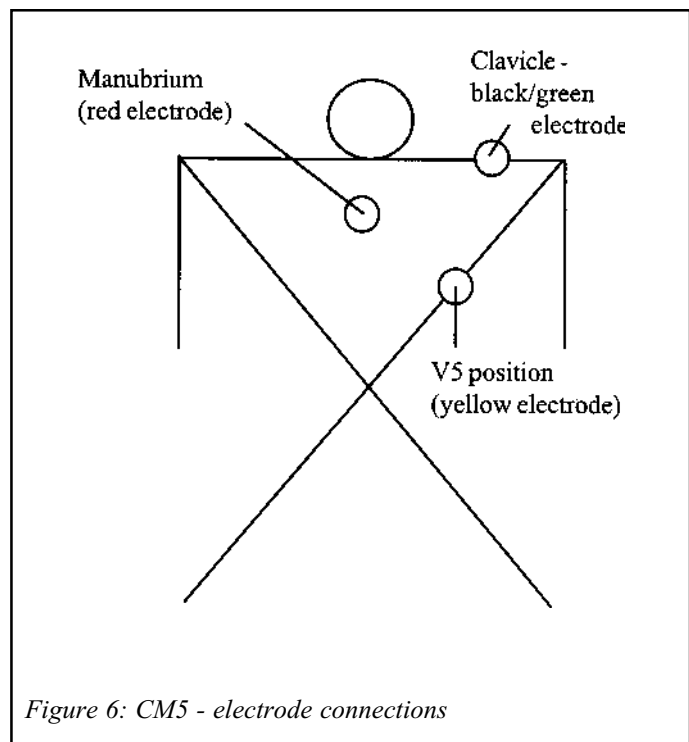
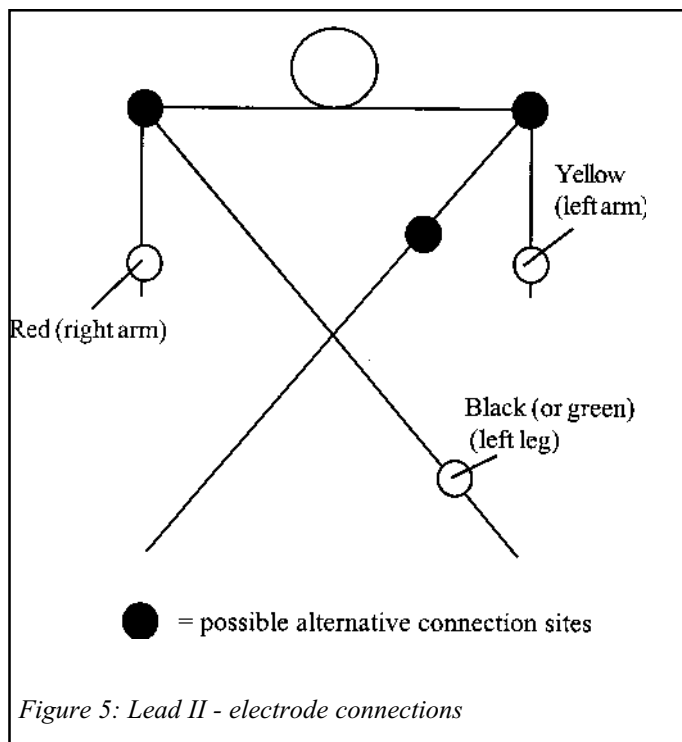
For detection of myocardial ischaemia the CM5 lead is useful (figure 6). This is a bipolar lead with the right arm electrode placed on the manubrium and left arm electrode placed at the surface marking of the V5 position (just above the 5th interspace in the anterior axillary line). The left leg lead acts as a neutral and may be placed anywhere - the C refers to 'clavicle' where it is often placed. To select the CM5 lead on the monitor, turn the selector dial to 'lead I'. This position allows detection of up to 80% of left ventricular episodes of ischaemia, and as it also displays arrhythmias it can be recommended for use

in most patients. The CB5 lead is another bipolar lead which has one electrode positioned at V5 and the other over the right scapula. This results in improved QRS and P wave voltages allowing easier detection of arrhythmias and ischaemia. Many other electrode positions have been described including some used during cardiac surgery, for example oesophageal and intracardiac ECG's.

CARDIAC ARRHYTHMIAS

The detection of cardiac arrhythmias and the determination of heart rate is the most useful function of the intraoperative ECG. Anaesthesia and surgery may cause any type of arrhythmia including:

- Transient supraventricular and ventricular tachycardias due to sympathetic stimulation during laryngoscopy and intubation.
- Bradycardias produced by surgical manipulation resulting in vagal stimulation. Severe bradycardia and asystole may result. It is more common in children because the sympathetic innervation of the heart is immature and vagal tone predominates. Bradycardias are most commonly seen in ophthalmic surgery due to the oculocardiac reflex. Generally the heart rate will improve when the surgical stimulus is removed.
- Atrial fibrillation is common during thoracic



surgery.

Drugs may also cause changes in cardiac rhythm eg;

- Halothane and nitrous oxide may cause junctional rhythms - (these will be detailed later). Halothane has a direct effect on the SA node and conducting system leading to a slowing in impulse generation and conduction and predisposes to re-entry phenomena. Catecholamines also have potent effects on impulse conduction, so the interaction of halothane and exogenous or endogenous catecholamines may cause ventricular arrhythmias. Ventricular ectopic beats are common. However rhythm disturbances such as ventricular tachycardia or rarely ventricular fibrillation may occur. The presence of cardiac disease, hypoxia, acidosis, hypercarbia (raised CO₂ level) or electrolyte disturbances will increase the likelihood of these arrhythmias.
- Arrhythmias occurring during halothane anaesthesia can often be resolved by reducing the concentration of halothane, ensuring adequate ventilation thereby preventing hypercarbia, increasing the inspired oxygen concentration and providing an adequate depth of anaesthesia for the surgical procedure. Tachyarrhythmias in the presence of halothane anaesthesia are uncommon if ventilation is adequate, and the use of adrenaline infiltration for haemostasis is limited to solutions of 1:100,000 or less and the dose in adults is not greater than 0.1mg in 10 minutes or 0.3mg per hour).
- Drugs increasing heart rate include ketamine, ether, atropine and pancuronium. Drugs decreasing heart rate include opioids, beta blockers and halothane.

Action Plan - when faced with an abnormal rhythm on the ECG monitor

Assess the vital signs - A.B.C.

- Check the airway is patent
- Check the patient is breathing adequately or is being ventilated correctly
- Listen for equal air entry into both lungs
- Circulation - check pulse, blood pressure, oxygen saturation. Is there haemodynamic compromise? Does the abnormal rhythm on the monitor match

the pulse that you can feel?

Consider the following:

- Increase the inspired oxygen concentration
- Reduce the inspired volatile agent concentration
- Ensure that ventilation is adequate to prevent CO₂ build up. Check end tidal CO₂ where this measurement is available
- Consider what the surgeon is doing - is this the cause of the problem? Eg: traction on the peritoneum or eye causing a vagal response. If so ask them to stop while you treat the arrhythmia.
- If the arrhythmia is causing haemodynamic instability, rapid recognition and treatment is required. However, many abnormal rhythms encountered in every day practice will respond to the above basic measures - sometimes even before identification of the exact rhythm abnormality is possible.

PRACTICAL INTERPRETATION AND MANAGEMENT OF ARRHYTHMIAS

When interpreting arrhythmias a paper strip is often easier to read than an ECG monitor. Where this is not possible from the theatre monitor it may be possible to obtain a paper trace by connecting a defibrillator, most of which have a facility for printing a rhythm strip. The following basic points should be considered:

Examining an ECG strip:

1. What is the ventricular rate?
2. Is the QRS complex of normal duration or widened?
3. Is the QRS regular or irregular?
4. Are P waves present and are they normally shaped?
5. How is atrial activity related to ventricular activity?

1. *What is the ventricular rate? Arrhythmias may be classified as fast or slow:*

- Tachyarrhythmias - rate greater than 100/min
- Bradyarrhythmias - rate less than 60/min
- Calculate approximate ventricular rate on a paper strip by counting the number of large squares between each QRS complex and dividing this number into 300 which will give the rate in beats/minute.

2. *Is the QRS complex of normal duration or widened? Arrhythmias may be due to abnormal impulses arising from the:*

- atria = a supraventricular rhythm
- AV node = a nodal or junctional rhythm
- or the ventricles = a ventricular arrhythmia

Supraventricular and nodal rhythms arise from a focus above the ventricles. Since the ventricles still depolarise via the normal His-Purkinje system the QRS complexes are of normal width (< 0.1sec - 2.5 small squares) - and are therefore termed 'narrow complex' rhythms. Arrhythmias arising from the ventricles will be 'broad complex' with a QRS width of >0.1sec. The QRS complexes are widened in these patients since depolarisation is via the ventricular muscle rather than the His-Purkinje system and takes longer. In a few cases where there is an abnormal conduction pathway from atria to ventricles a supraventricular rhythm may have broad complexes. This is called 'aberrant conduction'.

3. *Is the QRS regular or irregular?*

The presence of an irregular rhythm will tend to suggest ectopic beats (either atrial or ventricular), atrial fibrillation, atrial flutter with variable block or second degree heart block with variable block - see page 29.

4. *Are there P waves present and are they normally shaped?*

The presence of P waves indicates that the atria have depolarised and gives a clue to the likely origin of the rhythm. Absent P waves associated with an irregular ventricular rhythm suggest atrial fibrillation whilst a saw tooth pattern of P waves is characteristic of atrial flutter. If the P waves are upright in leads II and AVF they have originated from the sinoatrial node. However, if the P waves are inverted in these leads, it indicates that the atria are being activated in a retrograde direction ie: the rhythm is junctional or ventricular.

5. *How is atrial activity related to ventricular activity?*

Normally there will be one P wave per QRS complex. Any change in this ratio indicates a blockage to conduction at some point in the

pathway from the atria to the ventricles.

CLASSIFICATION OF ARRHYTHMIAS

Arrhythmias may be divided into narrow complex and broad complex for the purpose of rapid recognition and management.

Narrow complex arrhythmias - arise above the bifurcation of the bundle of His. The QRS duration is less than 0.1s (2.5 small squares) duration

Broad complex arrhythmias - usually arise either from the ventricles or less commonly are conducted abnormally from a site above the ventricles so that delay occurs (this is called aberrant conduction). The QRS duration is greater than 0.1s (2.5 small squares).

NARROW COMPLEX RHYTHMS:

- Sinus arrhythmia
- Sinus tachycardia
- Sinus bradycardia
- Junctional / AV nodal tachycardia
- Atrial tachycardia, atrial flutter
- Atrial fibrillation
- Atrial ectopics

BROAD COMPLEX RHYTHMS

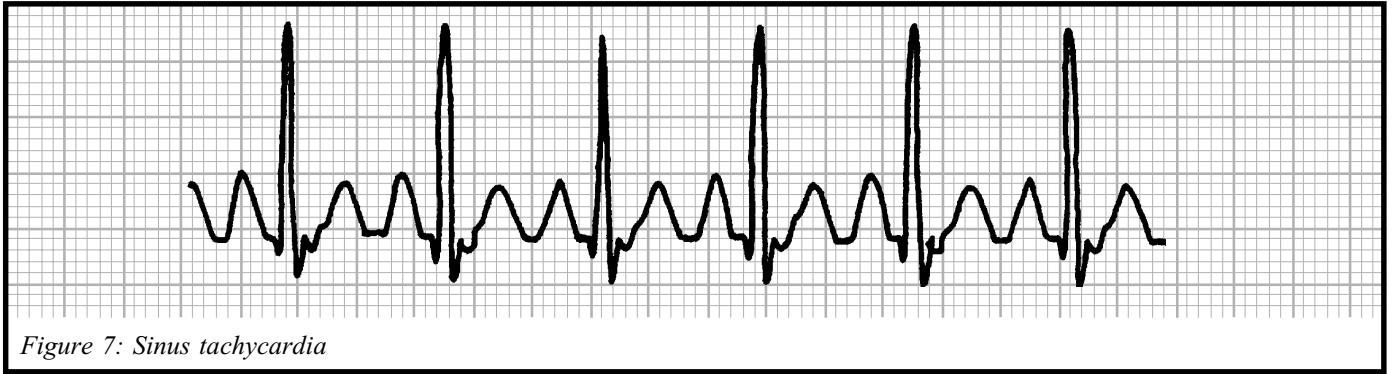
- Ventricular ectopics
- Ventricular tachycardia
- Supraventricular tachycardia with aberrant conduction
- Ventricular fibrillation

NARROW COMPLEX ARRHYTHMIAS

Sinus arrhythmia This is irregular spacing of normal complexes associated with respiration. There is a constant P-R interval with beat to beat change in the R-R interval. It is a normal finding especially in young people.

Sinus tachycardia (figure 7). There is a rate greater than 100/min in adults. Normal P-QRS-T complexes are evident. Causes include:

- Inadequate depth of anaesthesia
- Pain / surgical stimulation
- Fever / sepsis
- Hypovolaemia
- Anaemia



- Heart failure
- Thyrotoxicosis
- Drugs eg atropine, ether, ketamine, catecholamines

Management : correction of any underlying cause where possible. Beta blockers may be useful if tachycardia causes myocardial ischaemia in patients with ischaemic heart disease, but should be avoided in asthma and used with caution in patients with heart failure.

Sinus bradycardia (figure 8).

This is defined as a heart rate of less than 60 beats/minute in an adult.

It may be normal in athletic patients and may also be due to vagal stimulation during surgery - see above.

Other causes include:

- Drugs eg; beta blockers, digoxin, anticholinesterase drugs, halothane, second dose of suxamethonium (occasionally first dose in children)
- Myocardial infarction
- Sick sinus syndrome
- Raised intracranial pressure
- Hypothyroidism

- Hypothermia

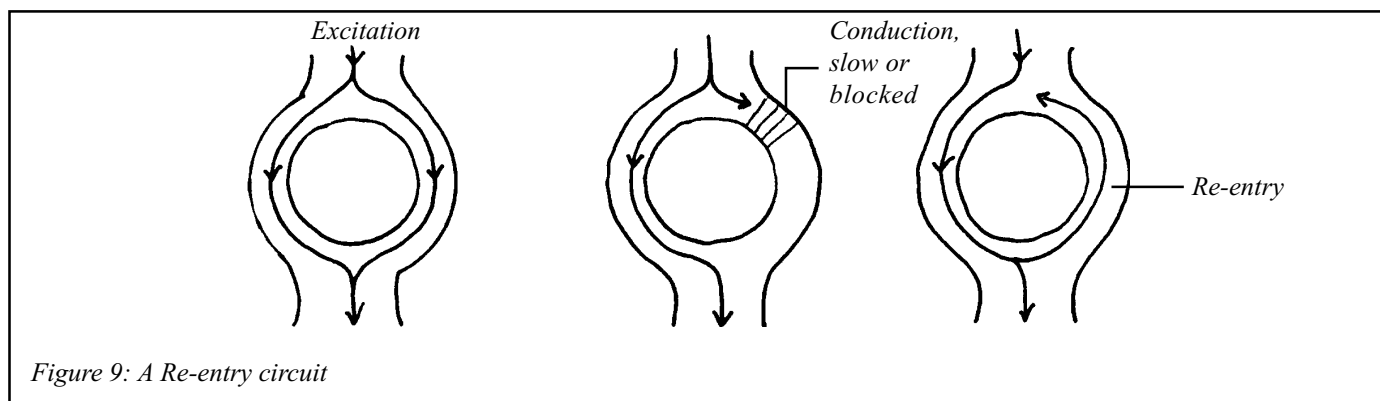
Management It is often not necessary to correct a sinus bradycardia in a fit young person, unless the rate is less than 45 - 50 beats per minute, and / or there is haemodynamic compromise. However consider:

- Correcting the underlying cause eg: stop the surgical stimulus
- Atropine up to 20 mcg/kg iv or glycopyrolate 10 mcg/kg iv. (Atropine works more rapidly and is usually given in doses of 300-400mcg and repeated if required).
- Patients on beta blockers may be resistant to atropine - occasionally an isoprenaline infusion may be required. Alternatively glucagon (2-10mg) can be used in addition to atropine.

ARRHYTHMIAS DUE TO RE-ENTRY (Circular movement of electrical impulses).

These arrhythmias occur where there is an anatomical branching and re-joining of a conduction pathway. Normally conduction would occur down both limbs equally. But if one limb is slower than the other, an impulse may pass normally down one limb but be blocked in the other. Where the pathways rejoin the impulse can then





spread backwards up the abnormal pathway. If it arrives at a time when the first pathway is no longer refractory to activation it can pass right round the circuit repeatedly activating it and resulting in a tachycardia (figure 9.) The classical example of this is the Wolf Parkinson White syndrome where there is a relatively large anatomical 'accessory' conduction pathway between the atria and the ventricles. This is called a 'macro re-entry' circuit. Other macro re-entry circuits can occur within the atrial and ventricular myocardium and are responsible for paroxysmal atrial flutter, atrial fibrillation and ventricular tachycardia. In junctional or AV nodal tachycardia there are 'micro re-entry' circuits within the AV node itself.

JUNCTIONAL / AV NODAL TACHYCARDIA (figure 10)

The term Supraventricular Tachycardia (SVT) applies to all tachyarrhythmias arising from a focus above the ventricles. However it is often used to describe junctional (AV nodal) tachycardias arising from micro re-entry circuits in or near the AV node, or as in the Wolf Parkinson White syndrome from an accessory conduction pathway between the atria and the ventricles. The ECG appearance is of a narrow complex tachycardia (QRS < 0.1s ie 2.5 small squares of standard ECG paper), with a rate of 150-200 bpm (figure 10).

The typical features seen on a 12 lead ECG taken when the patient is in sinus rhythm are:

- A short P-R interval
- A slurred upstroke on the R wave (the delta wave - best seen in V4)
- Inverted T waves in V2-5 are characteristic.

Management:

This arrhythmia may be associated with severe circulatory disturbance and needs to be managed as an emergency if it occurs during anaesthesia.

1. In the presence of hypotension, especially where the patient is anaesthetised in theatre, the first line treatment is synchronised direct current cardioversion with 200 - 360 joules.
2. Carotid Sinus Massage - this rarely converts to sinus rhythm but slows the rate and will reveal the underlying rhythm if there is any doubt. It is helpful in differentiating it from atrial flutter and fast atrial fibrillation. (The carotid sinus is a small dilatation of the proximal part of the internal carotid artery at the level of the superior border of the thyroid cartilage. It is vagally innervated and is involved in the control mechanism for causing a fall in heart rate and cardiac output in response to a rise in arterial pressure.



Gentle pressure on the internal carotid artery at this level may result in a slowing of the heart rate and occasionally termination of a re-entry supraventricular tachycardia. It should NEVER be attempted on both sides at once as this may result in asystole and occlusion of the main arterial blood supply to the brain.) It is contra-indicated in patients with a history of cerebrovascular disease.

3. Adenosine - this slows AV conduction and is especially useful for terminating re-entry SVTs of the Wolf Parkinson White type. Give 3mg iv rapidly preferably via a central or large peripheral vein - followed by a saline flush. Further doses of 6mg and then 12mg may be given at 2 min intervals if there is no response to the first dose. The effects of adenosine last only 10 -15 seconds. It should be avoided in asthma.

4. Verapamil, beta blockers or other drugs such as amiodarone or flecainide may control the rate or convert to sinus rhythm.

- Verapamil 5 -10mg iv slowly over 2 minutes. A further 5mg may be given after 10 minutes if required. Avoid giving concurrently with beta blockers as this may precipitate hypotension and asystole.
- Beta blockers eg: propranolol 1 mg over 1 minute repeated if necessary at 2 minute intervals (maximum 5mg), or sotalol 100mg over 10 minutes repeated 6 hourly if necessary. Esmolol - a relatively cardio-selective beta blocker with a very short duration of action may be given by infusion at 50 - 200 mcg/kg/minute.

Digoxin should be avoided - it facilitates conduction through the AV accessory pathway in the Wolf Parkinson White syndrome and may worsen the tachycardia. Note that atrial fibrillation in the presence of an accessory pathway may allow very rapid conduction which can degenerate to ventricular fibrillation.

ATRIAL TACHYCARDIA AND ATRIAL FLUTTER (figure 11)

This is due to an ectopic focus depolarising from anywhere within the atria. The atria contract faster than 150 bpm and P waves can be seen superimposed on the T waves of the preceding beats. The AV node conducts at a maximum rate of 200 bpm, therefore if the atrial rate is faster than this, AV block will occur. If the atrial rate is greater than 250 beats/min and there is no flat baseline between P waves, then the typical 'saw tooth' pattern of atrial flutter waves will be seen.

Atrial tachycardia and flutter may occur with any kind of block:

Eg: 2:1, 3:1, or 4:1.

Atrial tachycardia is typically a paroxysmal arrhythmia, presenting with intermittent tachycardia and palpitations, and may be precipitated by anaesthesia and surgery. It is associated in particular with rheumatic valvular disease as well as ischaemic and hypertensive heart disease and may be seen with mitral valve prolapse. It may precede the onset of permanent atrial fibrillation. Atrial tachycardia with 2:1 block is characteristic of digitalis toxicity.

Management

- This arrhythmia is very sensitive to synchronised direct current cardioversion - there is a nearly 100% success rate. Therefore in the anaesthetised patient with any degree of cardiovascular compromise this should be the first line treatment.
- Carotid sinus massage and adenosine will slow AV conduction and reveal the underlying rhythm and block where there is doubt.
- Other drug treatment is as for atrial fibrillation. (see page 25).



Figure 11: Atrial tachycardia

ATRIAL FIBRILLATION (AF - figure 12)

A common arrhythmia encountered in anaesthetic and surgical practice. There is chaotic and unco-ordinated atrial depolarisation, an absence of P waves on the ECG, with an irregular baseline and a completely irregular ventricular rate. Transmission of atrial activity to the ventricles via the AV node depends on the refractory period of the conducting tissue. In the absence of drug treatment or disease which slows conduction, the ventricular response rate will normally be rapid ie: 120 - 200 beats/min.

Common causes of AF include:

- Ischaemia
- Myocardial disease / pericardial disease / mediastinitis
- Mitral valve disease
- Sepsis
- Electrolyte disturbance (particularly hypokalaemia or hypomagnesaemia)
- Thyrotoxicosis
- Thoracic surgery

Since contraction of the atria contributes up to 30% of the normal ventricular filling, the onset of AF may result in a significant fall in cardiac output. Fast AF may precipitate cardiac failure, pulmonary oedema and myocardial ischaemia. Systemic thrombo-embolism may occur if blood clots in the fibrillating atria and subsequently embolises into the circulation. There is a 4% risk per year of an embolic cerebro-vascular episode (CVE = stroke). The treatment of AF is aimed at the restoration of sinus rhythm whenever possible. Where this is not possible, the aim is control of the ventricular rate to <100/ minute and prevention of embolic complications. The management of this arrhythmia will vary depending on how long it has been

present. In acute AF restoration of sinus rhythm is often possible, whereas in longstanding AF control of the ventricular rate is the usual aim of therapy.

Management:

1. **Acute AF** - Occuring in theatre or of recent onset (less than 48 hours):

- Correction of precipitating factors where possible, especially correction of electrolyte disturbances.
- Synchronised DC cardioversion - for recent onset AF. If AF has been present for more than several hours there is a risk of arterial embolisation unless the patient is anticoagulated. Shock at 200J then at 360J.
- Digoxin can be used acutely to slow the ventricular rate - in the presence of a normal plasma potassium concentration. An intravenous loading dose of 500mcg in 100mls of saline over 20 minutes may be given and repeated at intervals of 4 - 8 hours if necessary until a total of 1 - 1.5mg has been given. This is contraindicated if the patient is already taking digoxin when lower doses are required. There is no evidence that digoxin is useful for converting AF to sinus rhythm or maintaining sinus rhythm once established.
- Amiodarone may be used to restore sinus rhythm - it is especially useful in paroxysmal atrial fibrillation associated with critical illness, and where digoxin or beta blockers cannot be used. A loading dose of 300mg iv via a central vein is given over 1 hour and then followed by 900mg over 23 hours.
- Verapamil 5 -10 mg slowly iv over 2 minutes can be used to control the ventricular rate. Where there is no impairment of left ventricular function or coronary artery disease, the subsequent

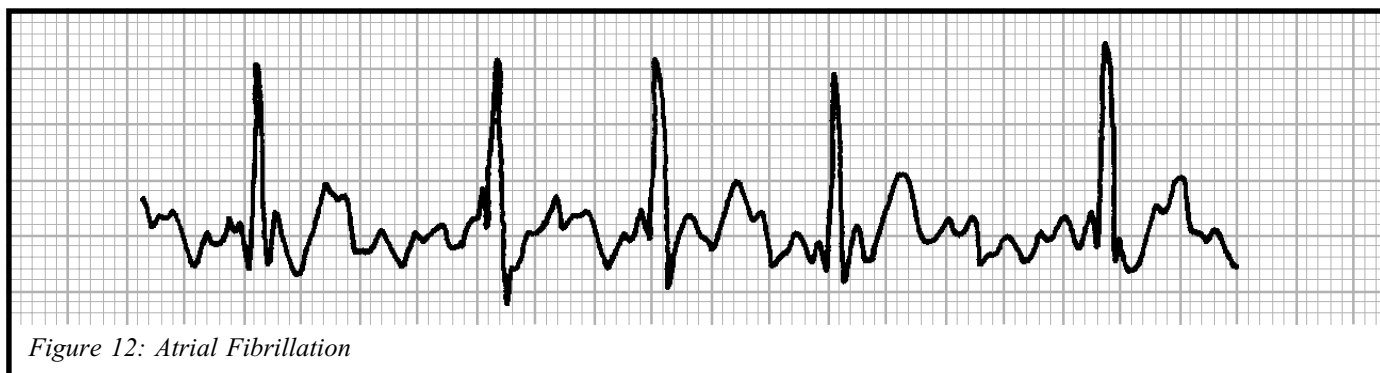


Figure 12: Atrial Fibrillation

administration of flecainide 50 - 100mg slowly iv may restore sinus rhythm. However flecainide should only be used where the arrhythmia is life threatening and no other options are open. It should be avoided if left ventricular function is poor or there is evidence of ischaemia.

- Beta blockers are sometimes used to control the ventricular rate but may precipitate heart failure in the presence of an impaired myocardium, thyrotoxicosis or calcium channel blockers, and should be used with caution.
2. **Chronic AF** with a ventricular rate of greater than 100/min. Aim to control the ventricular rate to less than 100/minute. This allows time for adequate ventricular filling and helps maintain the cardiac output.
- Digitalisation - if patient not already taking it. Consider extra digoxin if not fully loaded - beware signs of digoxin toxicity, nausea, anorexia, headache, visual disturbances etc, and arrhythmias especially ventricular ectopics and atrial tachycardia with 2:1 block.
 - Beta blockers or verapamil
 - Amiodarone

When AF has been present for more than a few hours anticoagulation is necessary before DC cardioversion to prevent the risk of embolisation. Usually patients should be warfarinised for 3 weeks prior to elective DC cardioversion, with regular monitoring of their prothrombin time. An INR of 2 or more is a satisfactory value at which to proceed with cardioversion. Warfarin should then be continued for 4 weeks afterwards. Occasionally when a patient develops AF and is compromised by it, DC cardioversion has to be considered even where anticoagulation is contraindicated (eg recent surgery).

ATRIAL ECTOPIC BEATS (figure 13)

An abnormal P wave is followed by a normal QRS complex. The P wave is not always easily visible on the ECG trace. The term 'ectopic' indicates that depolarisation originated in an abnormal place, ie not the SA node hence the abnormal shape of the P wave. If such a focus depolarises early the beat produced is called an extrasystole or premature contraction and may be followed by a compensatory pause. If the underlying SA node rate is slow, sometimes a focus in the atria takes over and the rhythm is described as an atrial escape, as it occurs after a small delay. Extrasystoles and escape beats have the same QRS appearance on the ECG, but extrasystoles occur early whereas escape beats occur late.

Causes:

- Often occur in normal hearts
- May occur with any heart disease
- Ischaemia, hypoxia
- Light anaesthesia
- Sepsis
- Shock
- Anaesthetic drugs are common causes

Management:

- Correction of any underlying cause.
- Specific treatment of atrial ectopic beats is unnecessary unless runs of atrial tachycardia occur - see above.

BROAD COMPLEX ARRHYTHMIAS

Ventricular Ectopic Beats (figure 14)

Depolarisation spreads from a focus in the ventricles by an abnormal, and therefore slow, pathway so the QRS



Figure 13: Atrial Ectopic Beats

complex is wide and abnormal. The T wave is also abnormal in shape.

In the absence of structural heart disease these are usually benign. They may be related to associated abnormalities especially hypokalaemia. They are common during dental procedures and anal stretches particularly with halothane, or whenever there is raised CO₂, light anaesthesia or no analgesia associated with halothane anaesthesia. In fit young patients under anaesthesia, they are often of little significance and respond readily to manipulation of the anaesthetic as described in 'first line management'. Small doses of intravenous beta blockers are very commonly effective in this situation.

However they may herald the onset of runs of ventricular tachycardia, and should be taken more seriously where:

- There is a bigeminal rhythm (one ectopic beat with every normal beat).
- If they occur in runs of 2 or more, or where there are more than 5/minute.
- Where they are multifocal (arising from different foci within the ventricles and hence having different shapes).
- Those where the R wave is superimposed on the T wave ('R on T' phenomenon).

The value of prophylactic treatment has been questioned as it is not known whether this influences the final outcome. However most would recommend treatment in the above four situations or where ventricular tachycardia has already occurred.

Management:

- Correction of any contributing causes identified with the anaesthetic ensuring adequate oxygenation, normocarbida and analgesia. A small dose of beta blocker is worth trying as mentioned above.

- If the underlying sinus rhythm is slow <50 bpm, then increasing this rate using intravenous atropine or glycopyrrolate may be effective as the ventricular ectopics may be a form of escape rhythm.
- Lignocaine is the drug of first choice. An initial loading dose of 50 - 100mg iv over 2 minutes is given followed by infusion of: 4mg/minute - for 30 minutes, then 2mg/minute - for 2 hours and then 1mg/minute. The dose should be reduced in the elderly, in liver disease and where there is bradycardia or hypotension.
- Alternatives include amiodarone 300mg iv (preferably via a central venous catheter) over 1 hour, followed by infusion of 900mg over 23 hours. Occasionally bretyllium or procainamide may be used.

Ventricular tachycardia (VT - figure 15)

In this rhythm a focus in the ventricular muscle depolarises at high frequency. Excitation spreads through the ventricles by an abnormal pathway and therefore the QRS complexes are wide and abnormal.

The appearance is characterised by absent P waves, wide QRS complexes which may be slightly irregular or vary in shape.

VT is a serious, potentially life threatening arrhythmia. It may be triggered intraoperatively by:

- Hypoxia
- Hypotension
- Fluid overload
- Electrolyte imbalance (low K⁺, Mg²⁺ etc)
- Myocardial ischaemia
- Injection of adrenaline

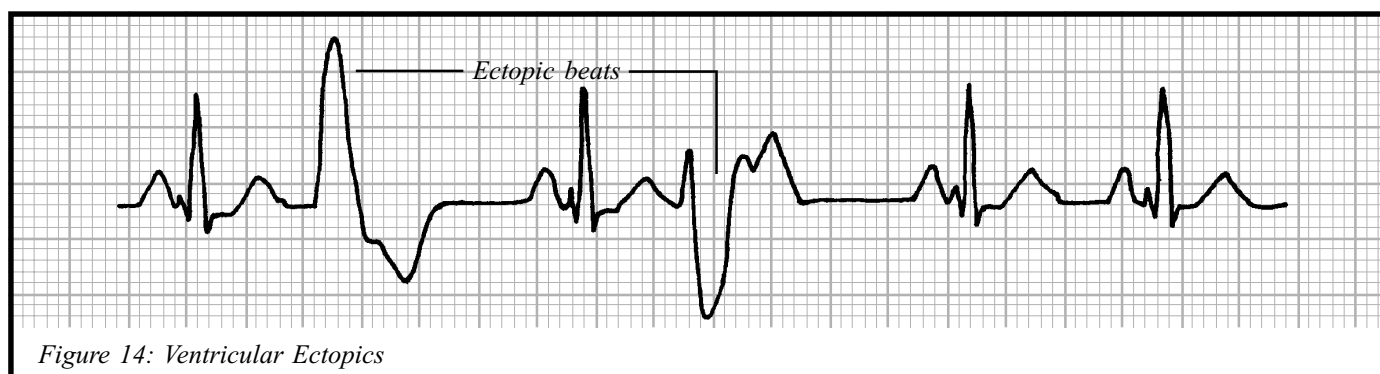


Figure 14: Ventricular Ectopics

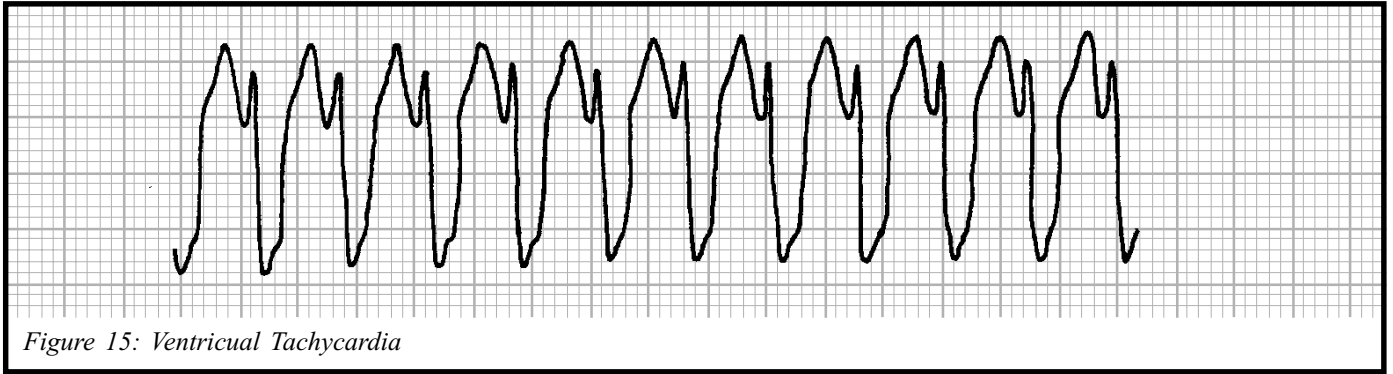


Figure 15: Ventricular Tachycardia

Management:

- Synchronised direct current cardioversion is the first line treatment if the patient is haemodynamically unstable. This is safe and effective and will restore sinus rhythm in virtually 100% of cases. If the VT is pulseless or very rapid, synchronisation is unnecessary. But otherwise synchronisation is used to avoid a 'shock on T' phenomenon which may initiate VF. If the patient lapses back into VT, drugs such as lignocaine or amiodarone may be given to sustain sinus rhythm.
- Lignocaine given as a 100mg bolus restores sinus rhythm in up to 60% and may be followed by a maintenance infusion as above.
- Verapamil is ineffective in ventricular tachycardia and may worsen hypotension and precipitate cardiac failure.

Other drugs which may be used if lignocaine fails:

- **Amiodarone** 300mg iv - via a central venous catheter over 1 hour followed by infusion of 900mg over 23 hours.
- **Procainamide** 100mg iv over 5 minutes followed by one or two further boluses before commencing infusion at 3mg/min.
- **Mexiletine** 100 - 250mg iv at 25mg/min followed by infusion 250mg over 1 hour, 125mg/hour for 2 hours, then 500mcg/min.
- **Bretylium tosylate** 400 - 500 mg diluted in 5% dextrose over 10 minutes
- **Propranolol** 0.5 - 1.0mg iv and repeated if necessary particularly if the underlying pathology is myocardial ischaemia or infarction.

- **Sotalol** 100mg iv over 5 minutes. This was shown to be better than lignocaine for acute termination of ventricular tachycardia.
- Overdrive pacing can be used to suppress VT by increasing the heart rate.

Supraventricular tachycardia with aberrant conduction

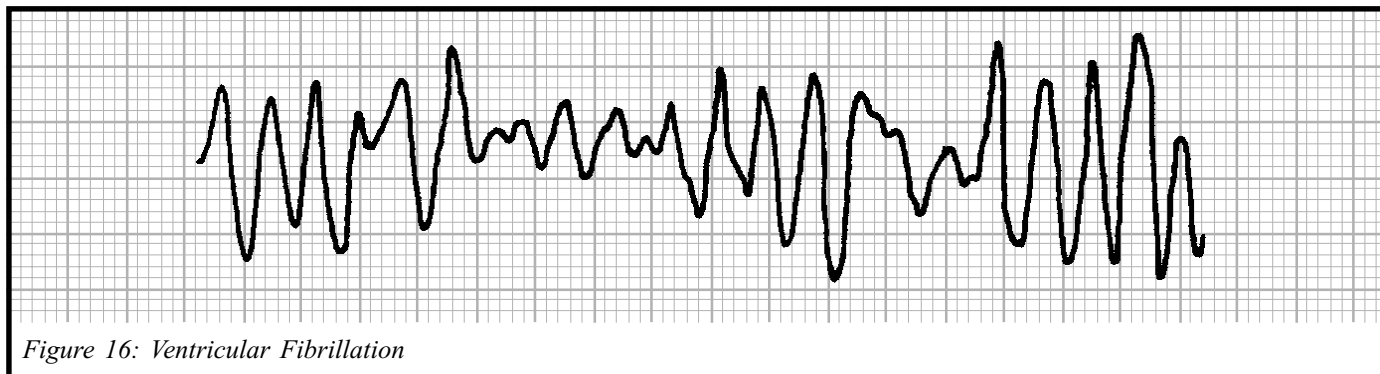
When there is abnormal conduction from the atria to the ventricles, a supraventricular tachycardia (SVT) may be broad complex as discussed above. This may occur for example if there is a bundle branch block. Sometimes the bundle branch block may be due to ischaemia and may only appear at high heart rates. SVTs may be due to an abnormal or accessory pathway (as in the Wolf Parkinson White syndrome), but during the tachycardia the complex is of normal width as conduction in the accessory pathway is retrograde, ie; it is the normal pathway that initiates the QRS complex. Adenosine may be used diagnostically to slow AV conduction and will often reveal the underlying rhythm if it arises from above the ventricles. In the case of SVT it may also result in conversion to sinus rhythm. In practice however the differentiation of the two is not important, and all such tachycardias should be treated as ventricular tachycardia if there is any doubt.

Ventricular Fibrillation (figure 16)

This results in cardiac arrest. There is chaotic and disorganised contraction of ventricular muscle and no QRS complexes can be identified on the ECG.

Management

Immediate direct current cardioversion as per established resuscitation protocol. (See Update 10).



DISTURBANCES OF CONDUCTION

The wave of cardiac excitation which spreads from the sinoatrial node to the ventricles via the conduction pathways may be delayed or blocked at any point.

First Degree Block (figure 17)

There is a delay in the conduction from the sinoatrial node to the ventricles, and this appears as a prolongation of the PR interval i.e. greater than 0.2 seconds. It is normally benign but may progress to second degree block - usually of the Mobitz type I. First degree heart block is not usually a problem during anaesthesia.

Second Degree Block - Mobitz Type I (Wenkebach) (figure 18)

There is progressive lengthening of the PR interval and then failure of conduction of an atrial beat. This is followed

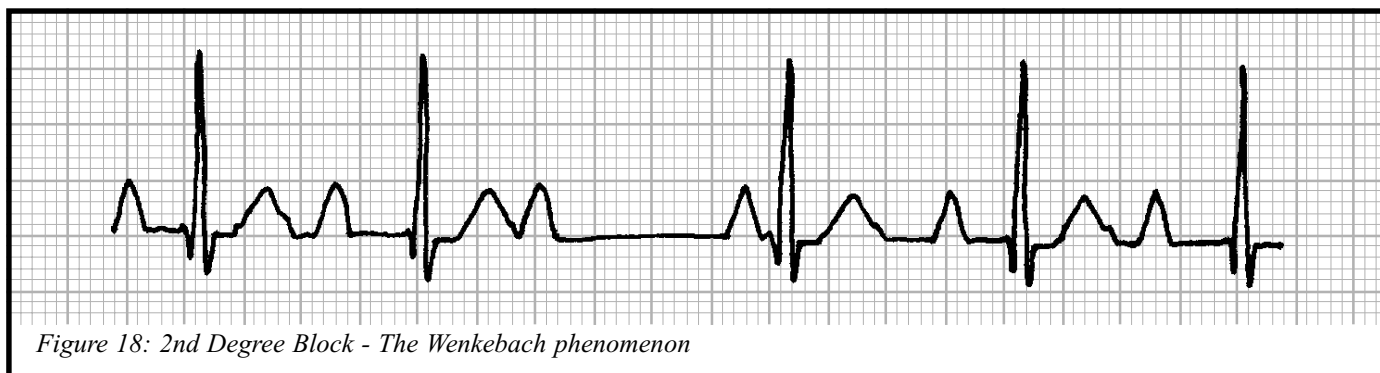
by a conducted beat with a short PR interval and then the cycle repeats itself. This occurs commonly after an inferior myocardial infarction, and tends to be self limiting. It does not normally require treatment although a 2:1 type block may develop with haemodynamic instability.

Second Degree Block - Mobitz Type II (figure 19)

If excitation intermittently fails to pass through the AV node or the bundle of HIS, this is the Mobitz type II phenomenon. Most beats are conducted normally but occasionally there is an atrial contraction without a subsequent ventricular contraction. This often progresses to complete heart block and if recognised preoperatively will need expert assessment.

Second Degree Block - 2:1 Type

There may be alternate conducted and non-conducted



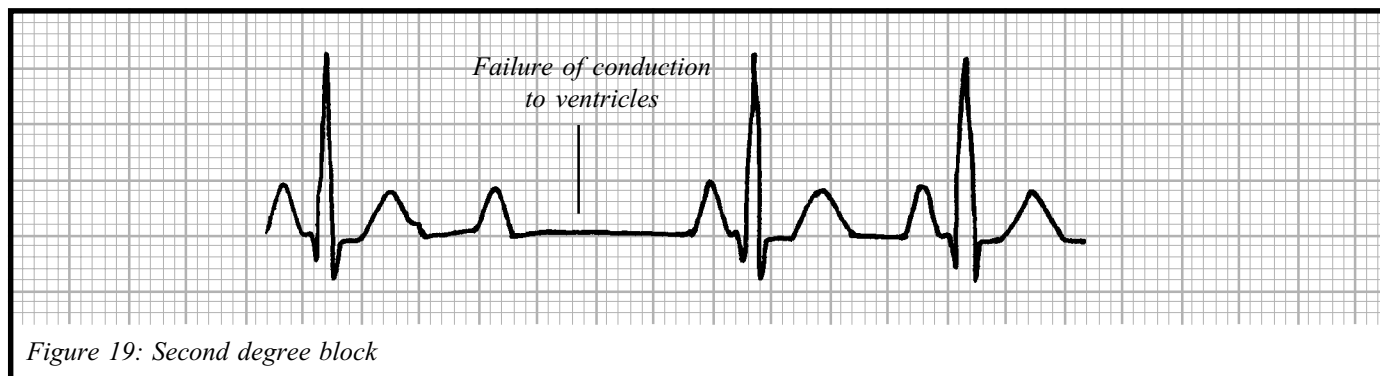


Figure 19: Second degree block

beats, resulting in 2 P waves for every QRS complex - this is 2:1 block. A 3:1 block may also occur, with one conducted beat and two non-conducted beats. This may also herald complete heart block, and in some situations the placing of a temporary transvenous pacing wire pre-operatively would be recommended.

Complete Heart Block (figure 20)

There is complete failure of conduction between the atria and the ventricles. The ventricles are therefore excited by a slow escape mechanism from a focus within the ventricles. There is no relationship between the P waves and the QRS complexes, and the QRS complexes are abnormally shaped. This may occur occasionally as a transient phenomenon in theatre as a result of vagal stimulation, in which case it often responds to stopping surgery and intravenous atropine. When it occurs in association with acute inferior myocardial infarction, it is due to AV nodal ischaemia and is often transient. Very rarely it may be congenital! However if it occurs with anterior myocardial infarction it indicates more extensive damage including to the HIS - Purkinje system. It may also occur as a chronic state usually due to fibrosis around the bundle of HIS.

Management

- Isoprenaline given by intravenous infusion can be used to increase the ventricular rate

- In the acute situation a temporary transvenous pacing wire may be required. A permanent pacemaker will be required in the longer term if the block is chronic and before contemplating elective surgery.

Bundle Branch Block (figure 21)

If the electrical impulse from the SA and AV nodes reaches the interventricular septum normally the PR interval will be normal. However if there is a subsequent delay in depolarisation of the right or left bundle branches, there will be a delay in depolarisation of part of the ventricular muscle and the QRS complex will be wide and abnormal.

A wide complex rhythm which is present at the start of surgery on initial attachment of the ECG monitor is usually due to bundle branch block (BBB), and is not an indication for cancelling the operation. However this does indicate the importance of attaching the ECG monitor before induction of anaesthesia, particularly where a pre-operative ECG is not available. Any changes on the ECG during anaesthesia and surgery can then easily be compared to the patients 'normal' ie pre-anaesthetic ECG tracing. The definition of which bundle is blocked can only be achieved by analysing a full 12 lead ECG. Two types of BBB are recognised.



Figure 20: Complete heart block

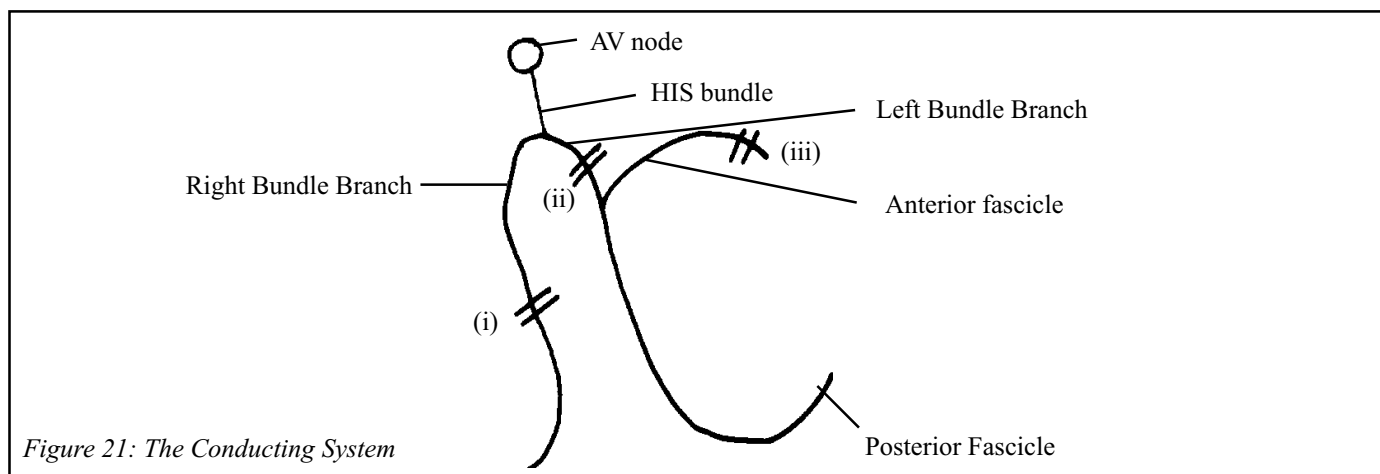


Figure 21: The Conducting System

- **Right Bundle Branch Block (i).** This may indicate problems with the right side of the heart, but a right bundle branch block type pattern with a normal axis and QRS duration is not uncommon in normal individuals.
- **Left Bundle Branch Block (ii).** This often indicates heart disease and makes further interpretation of the ECG other than rate and rhythm impossible.
- **2nd degree AV block of any type** if there is symptomatic bradycardia.
- **Asymptomatic 2nd degree heart block or first degree block** with symptoms suggestive of sick sinus syndrome (intermittent tachycardia and bradycardia) plus documented relief of symptoms with a temporary pacing wire. In both of these cases the weight of current opinion favours pacing.
- **Any type of bundle branch block with intermittent second or third degree block, or syncope** should be paced.
- **Bifascicular block** is relatively common in the elderly and does not require pacing.

Other forms of BBB

Bi-Fascicular Block (i and iii). This is a diagnosis which can only be made on a formal 12 lead ECG, and is included for completeness. It is the combination of right bundle branch block and block of the left anterior or posterior fascicle and appears on the ECG as a RBBB pattern with axis deviation. This progresses to complete heart block in a few patients.

Tri-Fascicular Block This is the term sometimes used to indicate the presence of a prolonged PR interval together with a bi-fascicular block.

PRE - OPERATIVE PROPHYLACTIC PACEMAKER INSERTION

Where facilities allow, pacemakers are sometimes inserted prior to surgery in patients who are at risk of developing complete heart block perioperatively. Those at risk of this complication have recently been described by the American college of cardiology and the American heart association. A pacemaker should be considered for:

- 3rd degree AV block which is symptomatic or has a ventricular escape rate of less than 40 beats per minute. Where the rate is greater than 40, there is conflicting evidence of benefit but the weight of opinion is in favour of pacing.

DETECTION OF MYOCARDIAL ISCHAEMIA (figure 22).

Cardiac events are the main cause of death following anaesthesia and surgery. Perioperative myocardial ischaemia is predictive of intra and post operative myocardial infarction. The likelihood of detection of ischaemia intraoperatively on the ECG is increased by the use of the CM5 lead as discussed above. This lead has the highest probability of detecting ischaemia, particularly in the lateral wall of the left ventricle which is the zone at greatest risk. Lead II is more likely to detect infero-posterior ischaemia and is therefore useful in those patients whose pre-operative ECG shows evidence of inferior or posterior ischaemia or infarction.

The ECG should always be recorded from before the start of the anaesthetic so that any subsequent changes can be observed. ST segment depression of 1mm or more below the isoelectric line with or without T wave changes indicates myocardial ischaemia. The magnitude of ST depression correlates with the severity but not the extent of the

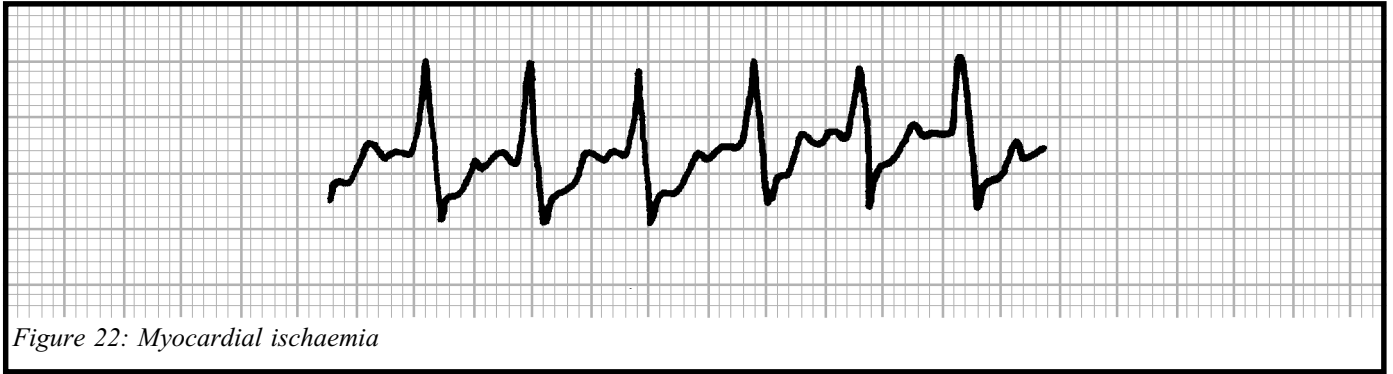


Figure 22: Myocardial ischaemia

ischaemia. The ST segment depression moves progressively from up-sloping to horizontal to down-sloping as ischaemia worsens. Down-sloping ST segment depression may indicate transmural ischaemia (through the full wall thickness).

On a 12 lead ECG full thickness myocardial infarction results in ST segment elevation often with the subsequent development of pathological Q waves (greater than 1 mm thick and 2mm deep). In subendocardial infarction - typically there is deep symmetrical T wave inversion. In subepicardial infarction - there is loss of R wave amplitude without development of Q waves.

Management

- If ST segment depression develops during anaesthesia, 100% oxygen should be given, the volatile agent decreased and the blood pressure and heart rate normalised as far as possible. It is important to maintain diastolic blood pressure and systemic vascular resistance, in order to maintain coronary artery perfusion. In this situation methoxamine (if available) in 2mg iv increments titrated to effect may be useful.
- Postoperative management in a high care environment should be considered where possible, with oxygen therapy, adequate analgesia and correction of fluid and electrolyte balance being of great importance. Monitoring should be continued into the postoperative period as this is the time when further (often silent) ischaemia and infarction may occur. Oxygen should be given to all high risk patients post operatively, ideally for at least 48 hours.

OTHER ECG CHANGES SEEN IN THEATRE

Occasionally the ECG changes shape slightly with a change in position of the patient or during different phases of mechanical ventilation. This usually causes a slight change in the position of the heart and results in the ECG being

recorded from a different angle. It is not usually of importance.

ECG APPEARANCE OF ABNORMAL POTASSIUM CONCENTRATIONS.

The ECG trace may develop characteristic changes with alterations in the concentration of various electrolytes. It is rarely possible to diagnose these from the ECG alone but the reading may give rise to a suspicion which should be confirmed by the laboratory.

Hyperkalaemia

- Tall peaked T waves
- Reduced P waves with widened QRS complexes
- Ultimately a sine wave pattern - pre-cardiac arrest
- Cardiac arrest in diastole

Hypokalaemia

- Increased myocardial excitability - any arrhythmia may occur
- Prolonged PR interval
- Prominent U waves
- Enhancement of digitalis toxicity

FURTHER READING AND REFERENCES

1. Ganong WF. A Review of Medical Physiology. Stamford: Appleton and Lange, 1997.
2. Hutton P, Prys-Roberts C. Monitoring in Anaesthesia and Intensive Care. London: WB Saunders Company Ltd, 1994.
3. Hinds CJ, Watson D. Intensive Care - A Concise Textbook. London: W.B Saunders Company Ltd, 1996.
4. The 1998 ERC Guidelines for Adult Advanced Life Support. Resuscitation 1998;37:81-90
5. Hampton J. The ECG made easy. London: Churchill Livingstone, 1986.
6. Mangano DT. Perioperative Cardiac Morbidity. Anaesthesiology 1990; 72:153-84.
7. Nathanson MH, Gajraj NM. The Perioperative Management of Atrial Fibrillation. Anaesthesia 1998; 53: 665-76.
8. Gregoratos G et al. ACC/AHA guidelines for implantation of cardiac pacemakers and antiarrhythmia devices. Executive summary. Circulation 1998; 97: 1325-35.