

# UPDATE IN ANAESTHESIA



*A journal for anaesthetists in developing countries*

## WORLD ANAESTHESIA

### EDITORIAL

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### The World Federation of Anaesthesiologists

The World Federation of Anaesthesiologists (WFA), founded in 1955, is a Federation of about 100 National and Regional Societies of Anaesthesia whose main aim is to improve the standards of anaesthesia around the world. It is run by the Officers and an Executive Committee supported by 4 standing committees including Finance, Statutes and Bylaws, and the very active Education and Publications Committees. There are also 6 specialist committees - Quality of practice, Safety, Equipment and Technology, Paediatrics, Obstetrics, Pain, Resuscitation and Intensive care.

One of the objectives of the Federation is to promote education and the dissemination of scientific information. A World congress is held every 4 years. The provision of continuing education for anaesthetists has been a major thrust of the Federation the past 15 – 20 years. Much of this work has been aimed at parts of the world where difficult circumstances have hindered the development of training in anaesthesia.

Between 1984 and 1988 John Zorab and Jack Moyers edited several volumes of WFA Lectures, which contained a wide range of topics written by leading anaesthesiologists. These were widely distributed. The first proactive course to be supported by the WFA in the recent era was a single day one on Paediatric anaesthesia held at the end of the East African meeting in Arusha in December 1985. Until this time the WFA's role in educational activities had mainly been supporting requests from national societies for lecturers, and sponsoring visiting educational teams to different parts of the world.

At the World Congress in Washington in 1988 the Education Committee was asked to develop post-graduate refresher courses in anaesthesia starting with English and French speaking Africa. During the next 4 years, 50 countries were visited and even more benefited, because anaesthetists from small countries, such as the Pacific

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Islands, could attend regional courses. Lecturers came from 25 countries representing a truly international effort. These were exciting times. For many anaesthetists, WFA courses provided the first educational activity they had encountered since training. Lecturers and students alike reported enthusiastically at the content and atmosphere of the courses.

In the huge area of the Pacific there are many scattered islands with small populations and only a few doctors. During the mid 1980's Anthea Hatfield from New Zealand visited several of these island hospitals, sometimes accompanied by a technician who was able to repair faulty equipment. Subsequently the Australian Society became involved, initially allocating \$3 and then \$5 of each membership subscription to help fund anaesthesia courses in Fiji with WFA support. After 4 years the Australian Government granted financial support, first for the course

and then for a resident lecturer who helped the first trainees through to a Diploma examination held in 1996 - the first post graduate medical qualification in the Pacific!

The pattern of anaesthesia training courses continues and has been added to recently by setting up Regional Training Centres. These are run in partnerships between WFSA and some of the better resourced national societies. One centre in Bangkok trains anaesthesiologists from countries such as Laos and Cambodia. After graduating they are able to return to their own country to train their own people. Training centres formerly existed in Copenhagen, Caracas and Manila supported by WHO, WFSA and the local societies.

It is well known that there is a shortage of suitable anaesthesia publications in many countries. The group, "World Anaesthesia", started "Update in Anaesthesia", which is distributed free of charge to many anaesthetists working in relatively inaccessible parts of the world. In 1992 the WFSA recognised that printed material is important and set up the Publications Committee. Since then the two groups have combined efforts in producing a joint Newsletter, "World Anaesthesia" and continuing to publish "Update". The latter is now produced in English, Spanish, Arabic, Russian and Mandarin and hopefully soon in French. It is circulated to all national societies with a total circulation of

around 13,000. Both publications are on the Web site:

<http://www.nda.ox.ac.uk/wfsa/>

The number of visitors to the Web site has doubled in 12 months reaching 11,283 from 91 countries in the past year. This makes the efforts of those producing the publications more worthwhile and must encourage anaesthetists who in the past have felt isolated. There is more to come. Distant learning on the Internet will hopefully soon come to fruition.

Much has been done to improve access to continuing education through courses and training centres in the late eighties and early nineties, while printed and electronic material is becoming increasingly available, even in relatively remote parts of the world, in the more recent years. We are grateful to those in WFSA and World Anaesthesia who have helped make this happen. There are many who should be acknowledged, and deserve our special thanks for the tremendous time, effort and enthusiasm they have put into these projects. Many of these people have first hand experience of working in less affluent countries.

**Kester Brown**

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## CARDIOVASCULAR PHYSIOLOGY

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### INTRODUCTION

The cardiovascular system consists of the heart and two vascular systems, the systemic and pulmonary circulations. The heart pumps blood through two vascular systems - the low pressure pulmonary circulation in which gas exchange occurs, and then the systemic circulation, which delivers blood to individual organs, matching supply to metabolic demand. Blood pressure and flow is largely controlled by the autonomic nervous system (**The Autonomic Nervous System**, Update in Anaesthesia 1995;5:3-6), and is also influenced by surgery and anaesthetic drugs. A good working knowledge of cardiovascular physiology is necessary to practice safe anaesthesia.

### THE HEART

The heart comprises four chambers, and is divided into a right and left side, each with an atrium and a ventricle. The

atria act as reservoirs for venous blood, with a small pumping action to assist ventricular filling. In contrast, the ventricles are the major pumping chambers for delivering blood to the pulmonary (right ventricle) and systemic (left ventricle) circulations. The left ventricle is conical in shape and has to generate greater pressures than the right ventricle, and so has a much thicker and more muscular wall. Four valves ensure that blood flows only one way, from atria to ventricle (tricuspid and mitral valves), and then to the arterial circulations (pulmonary and aortic valves). The myocardium consists of muscle cells which can contract spontaneously, also pacemaker and conducting cells, which have a specialised function.

### ELECTROPHYSIOLOGY OF THE HEART

Myocardial contraction results from a change in voltage across the cell membrane (depolarisation), which leads to

an action potential. Although contraction may happen spontaneously, it is normally in response to an electrical impulse. This impulse starts in the sinoatrial (SA) node, a collection of pacemaker cells located at the junction of the right atrium and superior vena cava. These specialised cells depolarise spontaneously, and cause a wave of contraction to pass across the atria. Following atrial contraction, the impulse is delayed at the atrioventricular (AV) node, located in the septal wall of the right atrium. From here His-Purkinje fibres allow rapid conduction of the electrical impulse via right and left branches, causing almost simultaneous depolarisation of both ventricles, approximately 0.2 seconds after the initial impulse has arisen in the sinoatrial node. Depolarisation of the myocardial cell membrane causes a large increase in the concentration of calcium within the cell, which in turn causes contraction by a temporary binding between two proteins, actin and myosin. The cardiac action potential is much longer than that of skeletal muscle, and during this time the myocardial cell is unresponsive to further excitation.

### The cardiac cycle

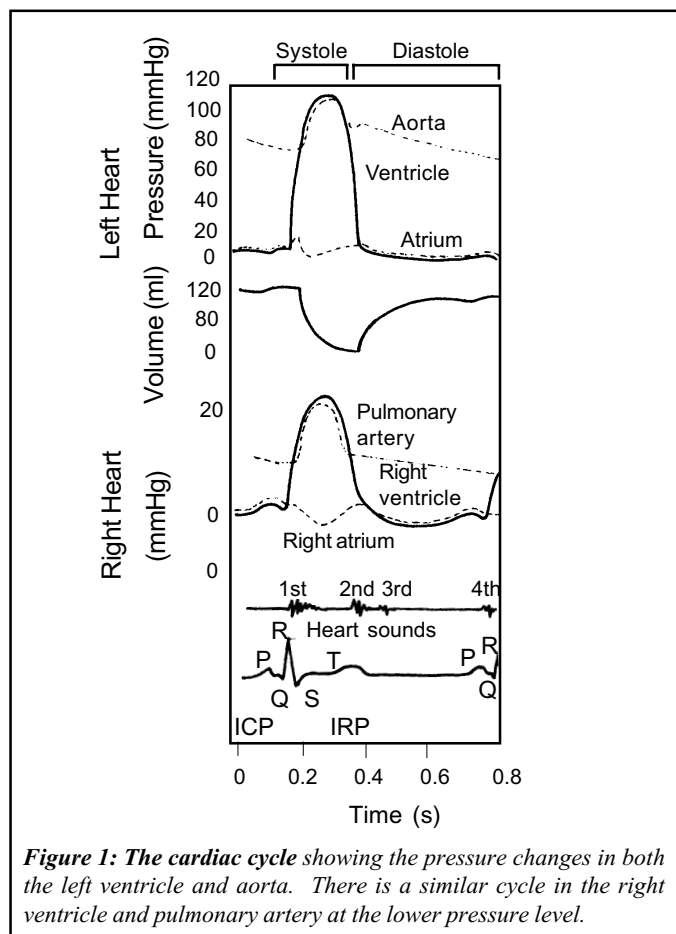
The relationship between electrical and mechanical events in the cardiac cycle is summarised in Figure 1. *Systole* refers to contraction, while *diastole* refers to relaxation.

#### Teaching point

The electrocardiogram (ECG) measures changes in skin electrical voltage/potential caused by electrical currents generated by the myocardium. The P wave reflects atrial depolarisation, the QRS complex ventricular depolarisation, and the T wave ventricular repolarisation (Figure 1). *Repolarisation* is a process that occurs in many cells where the electrical potential across the cell membrane returns from the value during the *action potential* to that of the resting state, the *resting potential*. Although the ECG shows heart rate and rhythm and can indicate myocardial damage, it gives no information on the adequacy of contraction. Normal electrical complexes can exist in the absence of cardiac output, a state known as *pulseless electrical activity* or *electromechanical dissociation*.

Both contraction and relaxation can be *isometric*, when changes in intraventricular pressure occur without a change in length of the muscle fibres. The cycle starts with depolarisation at the sinoatrial node leading to atrial contraction. Until this time blood flow into the ventricles has been passive, but the atrial contraction increases filling by 20-30%. Ventricular systole causes closure of the

atrioventricular valves (1st heart sound), and contraction is isometric until intraventricular pressures are sufficient to open the pulmonary and aortic valves, when the ejection phase begins. The volume of blood ejected is known as the stroke volume. At the end of this phase ventricular relaxation occurs, and the pulmonary and aortic valves close (2nd heart sound). After isometric relaxation ventricular pressures fall to less than atrial pressures. This leads to opening of the atrioventricular valves and the start of ventricular diastolic filling. The whole cycle then repeats following another impulse from the sinoatrial node.



**Figure 1:** The cardiac cycle showing the pressure changes in both the left ventricle and aorta. There is a similar cycle in the right ventricle and pulmonary artery at the lower pressure level.

### The coronary circulation

Myocardial blood supply is from the right and left coronary arteries, which run over the surface of the heart giving branches to the endocardium (the inner layer of the myocardium). Venous drainage is mostly via the coronary sinus into the right atrium, but a small proportion of blood flows directly into the ventricles through the Thebesian veins, delivering unoxygenated blood to the systemic circulation. Oxygen extraction by the tissues is dependent on consumption and delivery. Myocardial oxygen consumption is higher than in skeletal muscle (65% of arterial oxygen is extracted as compared to 25%). Therefore any increased myocardial metabolic demand must be matched by increased coronary blood flow. This

is a local response, mediated by changes in coronary arterial tone, with only a small input from the autonomic nervous system.

### Teaching point

Coronary blood flow occurs mostly during diastole, because during systole the blood vessels within the myocardium are compressed. Increased heart rates, which reduce the time for diastolic filling, can reduce myocardial blood supply and cause ischaemia. In heart failure, the ventricle is less able to empty and therefore the intraventricular volume and pressure is higher than normal. During diastole, this pressure is transmitted to the ventricular wall and opposes and reduces coronary flow, especially in the endocardial vessels.

### Cardiac Output

Cardiac output (CO) is the product of heart rate (HR) and stroke volume (SV):  $CO = HR \times SV$

For a 70kg man normal values are HR=70/min and SV=70ml, giving a cardiac output of about 5litre/min. The cardiac index is the cardiac output per square metre of body surface area - normal values range from 2.5-4.0 litre/min/metre.square.

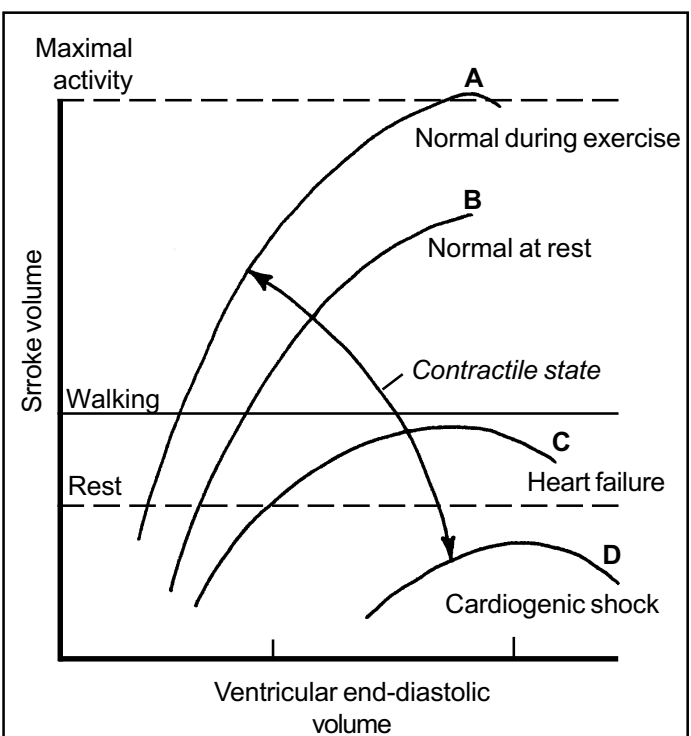
**Heart rate** is determined by the rate of spontaneous depolarisation at the sinoatrial node (see above), but can be modified by the autonomic nervous system. The vagus nerve acts on muscarinic receptors to slow the heart, whereas the cardiac sympathetic fibres stimulate beta-adrenergic receptors and increase heart rate.

**Stroke volume** is determined by three main factors: *preload*, *afterload* and *contractility*. These will be considered in turn:

*Preload* is the ventricular volume at the end of diastole. An increased preload leads to an increased stroke volume. Preload is mainly dependent on the return of venous blood from the body. Venous return is influenced by changes in position, intra-thoracic pressure, blood volume and the balance of constriction and dilatation (tone) in the venous system. The relationship between ventricular end-diastolic volume and stroke volume is known as **Starling's law of the heart**, which states that the energy of contraction of the muscle is related/proportional to the initial length of the muscle fibre. This is graphically illustrated in Figure 2 by a series of "Starling curves". As volume at the end of diastole (*end-diastolic volume*) increases and stretches

the muscle fibre, so the energy of contraction and stroke volume increase, until a point of over-stretching when stroke volume may actually decrease, as in the failing heart. Cardiac output will also increase or decrease in parallel with stroke volume **if there is no change in heart rate**. The curves show how the heart performs at different states of *contractility*, ranging from the normal heart to one in cardiogenic shock. This is a condition where the heart is so damaged by disease that cardiac output is unable to maintain tissue perfusion. Also shown are increasing levels of physical activity which require a corresponding increase in cardiac output.

*Afterload* is the resistance to ventricular ejection. This is caused by the resistance to flow in the systemic circulation and is the **systemic vascular resistance**. The resistance is determined by the diameter of the arterioles and pre-capillary sphincters; the narrower or more constricted, the higher the resistance. The level of systemic vascular resistance is controlled by the sympathetic system which, in turn, controls the tone of the muscle in the wall of the

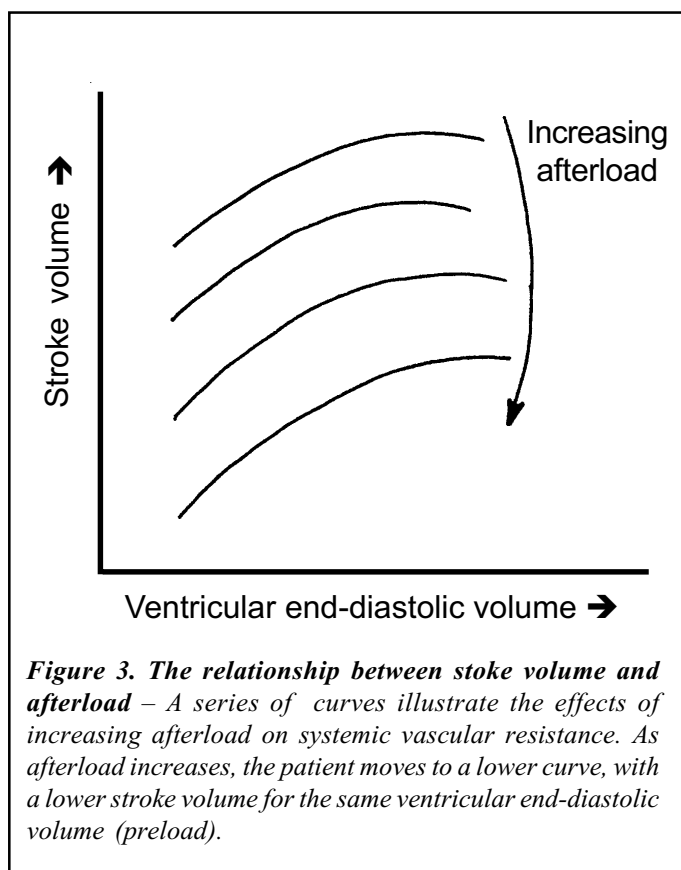


**Figure 2. Starling's Law of the Heart** - Curves A&B illustrate the rise in cardiac output with increases in ventricular end-diastolic volume (*pre-load*) in the normal heart. Note that with an increase in contractility there is a greater cardiac output for the same ventricular end-diastolic volume. In the diseased heart (C & D) cardiac output is less and falls if ventricular end diastolic volume rises to high levels, as in heart failure or overload.

arteriole, and hence the diameter. The resistance is measured in units of  $\text{dyne}\cdot\text{sec}/\text{cm}^5$ . A series of Starling curves with differing afterloads is shown in Figure 3, demonstrating a fall in stroke volume as afterload increases. The relationship between systemic vascular resistance and the control of arterial pressure is discussed below.

*Contractility* describes the ability of the myocardium to contract in the absence of any changes in preload or afterload. In other words, it is the “power” of the cardiac muscle. The most important influence on contractility is the sympathetic nervous system. Beta-adrenergic receptors are stimulated by noradrenaline released from nerve endings, and contractility increases. A similar effect is seen with circulating adrenaline and drugs such as ephedrine, digoxin and calcium. Contractility is reduced by acidosis, myocardial ischaemia, and the use of beta-blocking and anti-arrhythmic agents.

Cardiac output will change to match changing metabolic demands of the body. The outputs of both ventricles must be identical, and also equal the venous return of blood from the body. The balancing of cardiac output and venous return is illustrated during the response to exercise. Blood vessels dilate in exercising muscle groups because of increased metabolism, and blood flow increases. This



**Figure 3. The relationship between stroke volume and afterload** – A series of curves illustrate the effects of increasing afterload on systemic vascular resistance. As afterload increases, the patient moves to a lower curve, with a lower stroke volume for the same ventricular end-diastolic volume (preload).

increases venous return and right ventricular preload. Consequently more blood is delivered to the left ventricle and cardiac output increases. There will also be increased contractility and heart rate from the sympathetic activity associated with exercise, further increasing cardiac output to meet tissue requirements.

### Teaching point

A pulmonary artery catheter can measure pressures in the right heart as it is *float*ed into position. The catheter includes a small balloon which is transiently inflated to *wedge* it into a small pulmonary artery, occluding pulmonary arterial flow. This will give a characteristic waveform, and it is assumed that the measured pressure equals that in the left atrium. The catheter can also be used to measure cardiac output. However, in the absence of such monitoring, clinical examination gives a good indication of cardiac function. Skin temperature, capillary refill\*, pulse rate and volume, urine output and level of consciousness are reliable markers of cardiac output, and are easily assessed.

\**Capillary refill*- when pressure is applied to skin or a finger nail, it goes white. When the pressure is released, the colour rapidly returns within 2-3 seconds. This is capillary refill, and is prolonged when the peripheral circulation is poor due to hypovolaemia or a poor cardiac output.

### THE SYSTEMIC CIRCULATION

The systemic blood vessels are divided into arteries, arterioles, capillaries and veins. Arteries supply blood to the organs at high pressure, whereas arterioles are smaller vessels with muscular walls which allow direct control of flow through each capillary bed. Capillaries consist of a single layer of endothelial cells, and the thin walls allow exchange of nutrients between blood and tissue. Veins return blood from the capillary beds to the heart, and contain 70% of the circulating blood volume, in contrast to the 15% in the arterial system. Veins act a reservoir, and venous tone is important in maintaining the return of blood to the heart, for example in severe haemorrhage, when sympathetic stimulation causes venoconstriction.

## Blood flow

The relationship between flow and driving pressure is given by the **Hagen-Poiseuille** formula. This states that flow rate in a tube is proportional to:

$$\frac{\text{Driving pressure} \times \text{Radius}^4}{\text{Length} \times \text{Viscosity}}$$

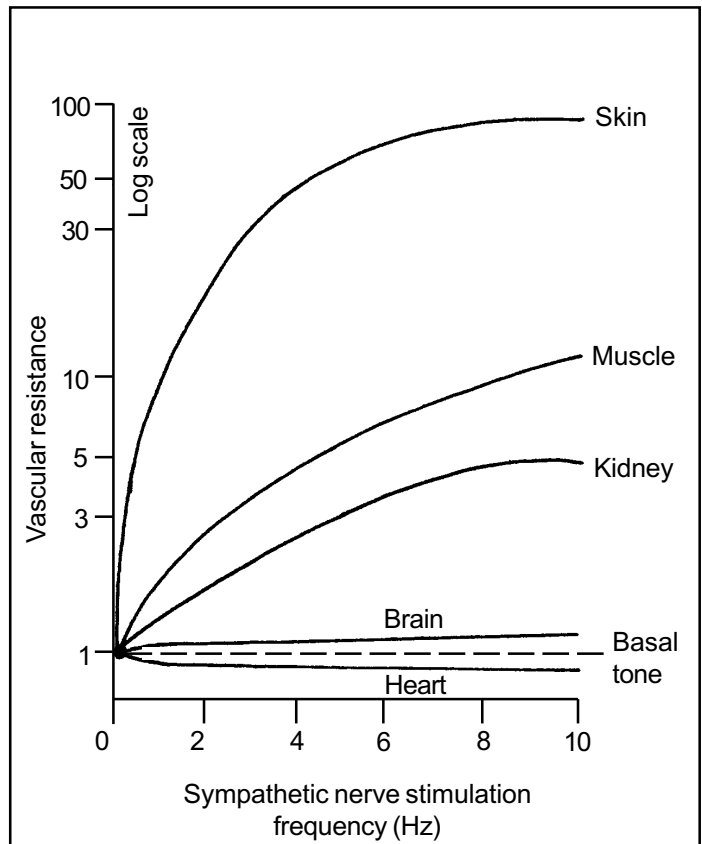
In blood vessels flow is pulsatile rather than continuous, and viscosity varies with flow rate, so the formula is not strictly applicable, but it illustrates an important point; small changes in radius result in large changes in flow rate. In both arterioles and capillaries changes in flow rate are brought about by changes in tone and therefore vessel radius.

Viscosity describes the tendency of a fluid to resist flow. At low flow rates the red blood cells stick together, increasing viscosity, and remain in the centre of the vessel. The blood closest to the vessel wall (which supplies side branches) therefore has a lower haematocrit. This process is known as *plasma skimming*. Viscosity is reduced in the presence of anaemia, and the resulting increased flow rate helps maintain oxygen delivery to the tissues.

## Control of the systemic circulation

Arteriolar tone determines blood flow to the capillary beds. A number of factors influence arteriolar tone, including *autonomic control*, *circulating hormones*, *endothelium derived factors* and the local concentration of *metabolites*.

*Autonomic control* is largely by the sympathetic nervous system, which supplies all vessels except capillaries. Sympathetic fibres arise from the thoracic and lumbar segments of the spinal cord. These are under the control of the **vasomotor centre** in the medulla, which has distinct vasoconstrictor and vasodilator areas. Although there is a baseline sympathetic discharge to maintain vascular tone, increased stimulation affects some organs more than others (Figure 4). This tends to redistribute blood from skin, muscle and gut to brain, heart and kidney. Increased sympathetic discharge is one of the responses to hypovolaemia, for example in severe blood loss, with the effect of protecting blood supply to the vital organs. The predominant sympathetic influence is vasoconstriction via alpha-adrenergic receptors. However, the sympathetic system also causes vasodilation via beta adrenergic and cholinergic receptor stimulation, but only in skeletal muscle.



**Figure 4.** The effects of sympathetic nervous stimulation on vascular resistance in different organs – Note that for the same sympathetic stimulation, the resistance is higher in skin than in the kidney, thus helping to preserve the flow to the kidney.

This increased blood flow to muscle is an important part of the “fight or flight” reaction, when exercise is anticipated.

*Circulating hormones* such as adrenaline and angiotensin II are potent vasoconstrictors, but they probably have little effect on acute cardiovascular control. In contrast, *endothelium derived factors* play an important role in controlling local blood flow. These substances are either produced or modified in the vascular endothelium, and include prostacyclin and nitric oxide, both potent vasodilators. An accumulation of *metabolites* such as CO<sub>2</sub>, K<sup>+</sup>, H<sup>+</sup>, adenosine and lactate causes vasodilation. This response is probably an important mechanism of *autoregulation*, the process whereby blood flow through an organ is controlled locally, and remains constant over a wide range of perfusion pressure. Autoregulation is a particular feature of the cerebral and renal circulations.

## Control of arterial pressure

Systemic arterial pressure is controlled closely in order to maintain tissue perfusion. The **mean arterial pressure (MAP)** takes account of pulsatile blood flow in the arteries,

and is the best measure of perfusion pressure to an organ. MAP is defined:

$$\text{MAP} = \text{Diastolic arterial pressure} + \frac{\text{pulse pressure}}{3}$$

where pulse pressure is the difference between systolic and diastolic arterial pressure.

MAP is the product of cardiac output (CO) and systemic vascular resistance (SVR):

$$\text{MAP} = \text{CO} \times \text{SVR}$$

If cardiac output falls, for example when venous return decreases in hypovolaemia, MAP will also fall unless there is a compensatory rise in SVR by vasoconstriction of the arterioles. This response is mediated by **baroreceptors**, which are specialised sensors of pressure located in the carotid sinus and aortic arch, and connected to the vasomotor centre. A fall in blood pressure causes reduced stimulation of the baroreceptors, and consequent reduced discharge from the baroreceptors to the vasomotor centre. This causes an increase in sympathetic discharge leading to vasoconstriction, increased heart rate and contractility, and secretion of adrenaline. Conversely, rises in blood pressure stimulate the baroreceptors, which leads to increased parasympathetic outflow to the heart via branches of the vagus nerve, causing slowing of the heart. There is also reduced sympathetic stimulation to the peripheral vessels causing vasodilation. Baroreceptor responses provide immediate control of blood pressure; if hypotension is prolonged, other mechanisms start to operate, such as the release of angiotensin II and aldosterone from the kidneys and adrenal glands, which leads to salt and water being retained in the circulation.

### Cardiovascular responses to anaesthesia

All anaesthetic agents have a direct depressant effect on the myocardium. Therefore they reduce myocardial contractility, and many also reduce sympathetic stimulation of the vascular system. The result is a decreased cardiac output accompanied by vasodilation, causing hypotension. This fall in blood pressure can compromise perfusion of vital organs, especially at induction of anaesthesia in the hypovolaemic patient. In **contrast**, agents such as **ketamine** and **ether** increase sympathetic activity, which opposes the direct depressant

### Teaching point

The *Valsalva manoeuvre* is a simple test of the baroreceptor reflex. The patient tries to breathe out forcefully against a closed larynx - "straining" - resulting in an increased intrathoracic pressure. This causes decreased venous return, cardiac output and a fall in blood pressure leading to reduced baroreceptor discharge to the vasomotor centre. This then causes peripheral vasoconstriction, and an increase in heart rate which is the normal response. This has the effect of maintaining *systolic* pressure, although the *pulse* pressure is reduced due to vasoconstriction.

effect. Thus cardiac output and blood pressure are maintained despite the direct myocardial depressant action.

Volatile anaesthetic agents reduce discharge from the sinoatrial node. This can lead to *junctional* rhythms, when the atrioventricular node takes over as pacemaker, associated with an absent P wave on the ECG. Local anaesthetic agents depress conduction of the cardiac impulse. This effect can be therapeutic, for example in the treatment of ventricular arrhythmias with lignocaine. However, at higher concentrations local anaesthetics can cause cardiac arrest - it is vital to avoid accidental intravenous injection when using these agents.

Controlled ventilation in a paralysed patient has many effects. Firstly it increases intrathoracic pressure which reduces venous return and preload, causing a fall in cardiac output. Secondly, changes in the partial pressure of carbon dioxide (PaCO<sub>2</sub>) resulting from changes in ventilation will also have cardiovascular effects. A low PaCO<sub>2</sub>, which commonly occurs during controlled ventilation, causes peripheral vasoconstriction by a direct effect. This increases systemic vascular resistance, increases afterload and can result in a fall in cardiac output. It also causes cerebral vasoconstriction, reducing cerebral blood volume (**Neurophysiology**, Update in Anaesthesia 1998). A high PaCO<sub>2</sub> usually occurs in the anaesthetised patient during spontaneous breathing, and causes vasodilation and increased sympathetic activity, leading to increased cardiac output. However, the heart will be more likely to develop arrhythmias, particularly when using volatile agents.

Spinal and epidural anaesthesia blocks sympathetic nerves as well as sensory and motor fibres. This can lead to marked hypotension due to arteriolar and venous dilation because the sympathetic nerves to the lower extremities are blocked. Cardiac sympathetic nerve fibres, which arise

from the high thoracic spinal cord, may also be blocked, allowing an unopposed vagal action on the heart. In this case there will not be an appropriate increase in cardiac output, and blood pressure will fall further with a bradycardia.

### Teaching point

For patients with coronary artery disease, it is important to use an anaesthetic technique which does not cause further myocardial ischaemia. The important principle is to ensure that *myocardial oxygen supply* is greater than *myocardial oxygen demand*. The balance between these two variables is influenced by the following factors:

#### Myocardial oxygen supply

Heart rate

Diastolic time

Coronary perfusion pressure

Aortic diastolic blood pressure

Ventricular end-diastolic blood pressure

Arterial oxygen content

Arterial oxygen partial pressure

Haemoglobin concentration

Coronary artery diameter

#### Myocardial oxygen demand

Heart rate

Ventricular wall tension

Preload

Afterload

Contractility

## THE PHYSIOLOGY OF OXYGEN DELIVERY

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In order to survive humans have to be able to extract oxygen from the atmosphere and transport it to their cells where it is utilised for essential metabolic processes. Some cells can produce energy without oxygen (anaerobic metabolism) for a short time, although it is inefficient. Other organs (e.g. brain) are made up of cells that can only make the energy necessary for survival in the presence of a continual supply of oxygen (aerobic metabolism). Tissues differ in their ability to withstand anoxia (lack of oxygen). The brain and the heart are the most sensitive. Initially a lack of oxygen affects organ function but with time irreversible damage is done (within minutes in the case of the brain) and revival is impossible.

### OXYGEN TRANSPORT FROM AIR TO TISSUES

Oxygen is transported from the air that we breathe to each cell in the body. In general, gases move from an area of high concentration (pressure) to areas of low concentration (pressure). If there are a mixture of gases in a container, the pressure of each gas (partial pressure) is equal to the pressure that each gas would produce if it occupied the container alone.

### Atmosphere to alveolus

The air (atmosphere) around us has a total pressure of 760 mmHg (1 atmosphere of pressure = 760mmHg = 101kPa = 15lbs/sq. in). Air is made up of 21% oxygen, 78% nitrogen and small quantities of CO<sub>2</sub>, argon and helium. The pressure exerted by the main two gases individually, when added together, equals the total surrounding pressure or atmospheric pressure. The pressure of oxygen (PO<sub>2</sub>) of dry air at sea level is therefore 159 mmHg (21/100 x 760=159). However by the time the inspired air reaches the trachea it has been warmed and humidified by the upper respiratory tract. The humidity is formed by water vapour which as a gas exerts a pressure. At 37°C the water vapour pressure in the trachea is 47 mmHg. Taking the water vapour pressure into account, the PO<sub>2</sub> in the trachea when breathing air is (760-47) x 21/100 = 150 mmHg. By the time the oxygen has reached the alveoli the PO<sub>2</sub> has fallen to about 100 mmHg. This is because the PO<sub>2</sub> of the gas in the alveoli (P<sub>A</sub>O<sub>2</sub>) is a balance between two processes: the removal of oxygen by the pulmonary capillaries and its continual supply by alveolar ventilation (breathing).



### Alveolus to blood

Blood returning to the heart from the tissues has a low  $PO_2$  (40 mmHg) and travels to the lungs via the pulmonary arteries. The pulmonary arteries form pulmonary capillaries, which surround alveoli. Oxygen diffuses (moves through the membrane separating the air and the blood) from the high pressure in the alveoli (100 mmHg) to the area of lower pressure of the blood in the pulmonary capillaries (40 mmHg). After oxygenation blood moves into the pulmonary veins which return to the left side of the heart to be pumped to the systemic tissues. In a 'perfect lung' the  $PO_2$  of pulmonary venous blood would be equal to the  $PO_2$  in the alveolus. Three factors may cause the  $PO_2$  in the pulmonary veins to be less than the  $PAO_2$ : ventilation/perfusion mismatch, shunt and slow diffusion.

### Ventilation/perfusion mismatch

In a 'perfect lung' all alveoli would receive an equal share of alveolar ventilation and the pulmonary capillaries that surround different alveoli would receive an equal share of cardiac output i.e. ventilation and perfusion would be perfectly matched.

Diseased lungs may have marked mismatch between ventilation and perfusion. Some alveoli are relatively overventilated while others are relatively overperfused (the most extreme form of this is shunt where blood flows past alveoli with no gas exchange taking place (figure 1). Well ventilated alveoli (high  $PO_2$  in capillary blood) cannot make up for the oxygen not transferred in the underventilated alveoli with a low  $PO_2$  in the capillary blood. This is because there is a maximum amount of oxygen which can combine with haemoglobin (see haemoglobin-oxygen dissociation curve figure 2a). The pulmonary venous blood (mixture of pulmonary capillary blood from all alveoli) will therefore have a lower  $PO_2$  than the  $PO_2$  in the alveoli ( $PAO_2$ ). Even normal lungs have some degree of ventilation/perfusion mismatch; the upper zones are relatively overventilated while the lower zones are relatively overperfused and underventilated.

**Shunt** occurs when deoxygenated venous blood from the body passes unventilated alveoli to enter the pulmonary veins and the systemic arterial system with an unchanged  $PO_2$  (40 mmHg). (Figure 1.) Atelectasis (collapsed alveoli), consolidation of the lung, pulmonary oedema or small airway closure (see later) will cause shunt.

### Diffusion

Oxygen diffuses from the alveolus to the capillary until the  $PO_2$  in the capillary is equal to that in the alveolus. This process is normally complete by the time the blood has

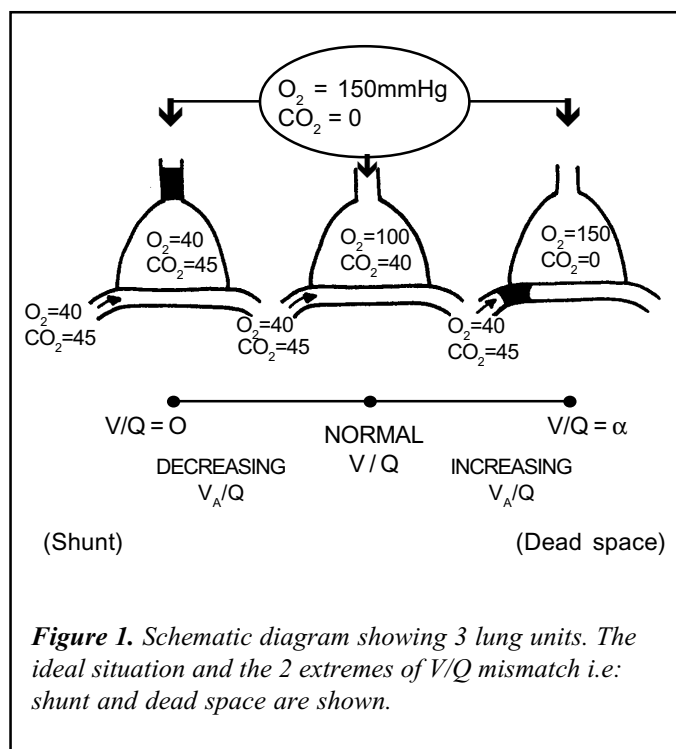
passed about one third of the way along the pulmonary capillary.

In the normal lung, the diffusion of oxygen into the blood is very rapid and is complete, even if the cardiac output is increased (exercise) and the blood spends less time in contact with the alveolus. This may not happen when the alveolar capillary network is abnormal (pulmonary disease). However, the ability of the lung to compensate is great and problems caused by poor gas diffusion are a rare cause for hypoxia, except with diseases such as alveolar fibrosis.

In order to decrease the detrimental effect that shunt and ventilation/perfusion mismatch have on oxygenation, the blood vessels in the lung are adapted to vasoconstrict and therefore reduce blood flow to areas which are underventilated. This is termed hypoxic pulmonary vasoconstriction and reduces the effect of shunt.

### Oxygen carriage by the blood

Oxygen is carried in the blood in two forms. Most is carried combined with haemoglobin (figure 2b) but there is a very small amount dissolved in the plasma. Each gram of haemoglobin can carry 1.31 ml of oxygen when it is fully saturated. Therefore every litre of blood with a Hb concentration of 15g/dl can carry about 200 mls of oxygen when fully saturated (occupied) with oxygen ( $PO_2 > 100$  mmHg). At this  $PO_2$  only 3 ml of oxygen will dissolve in every litre of plasma.



**Figure 1.** Schematic diagram showing 3 lung units. The ideal situation and the 2 extremes of  $V/Q$  mismatch i.e. shunt and dead space are shown.

If the  $PO_2$  of oxygen in arterial blood ( $P_{A}O_2$ ) is increased significantly (by breathing 100% oxygen) then a small amount of extra oxygen will dissolve in the plasma (at a rate of 0.003 ml  $O_2$ /100ml of blood /mmHg  $PO_2$ ) but there will normally be no significant increase in the amount carried by haemoglobin, which is already >95% saturated with oxygen. When considering the adequacy of oxygen delivery to the tissues, three factors need to be taken into account, haemoglobin concentration, cardiac output and oxygenation.

### Oxygen cascade

Oxygen moves down the pressure or concentration gradient from a relatively high level in air, to the levels in the respiratory tract and then alveolar gas, the arterial blood, capillaries and finally the cell. The  $PO_2$  reaches the lowest level (4-20 mmHg) in the mitochondria (structures in cells responsible for energy production). This decrease in  $PO_2$  from air to the mitochondrion is known as the oxygen cascade and the size of any one step in the cascade may be increased under pathological circumstances and may result in hypoxia (figure 3).

### Oxygen delivery

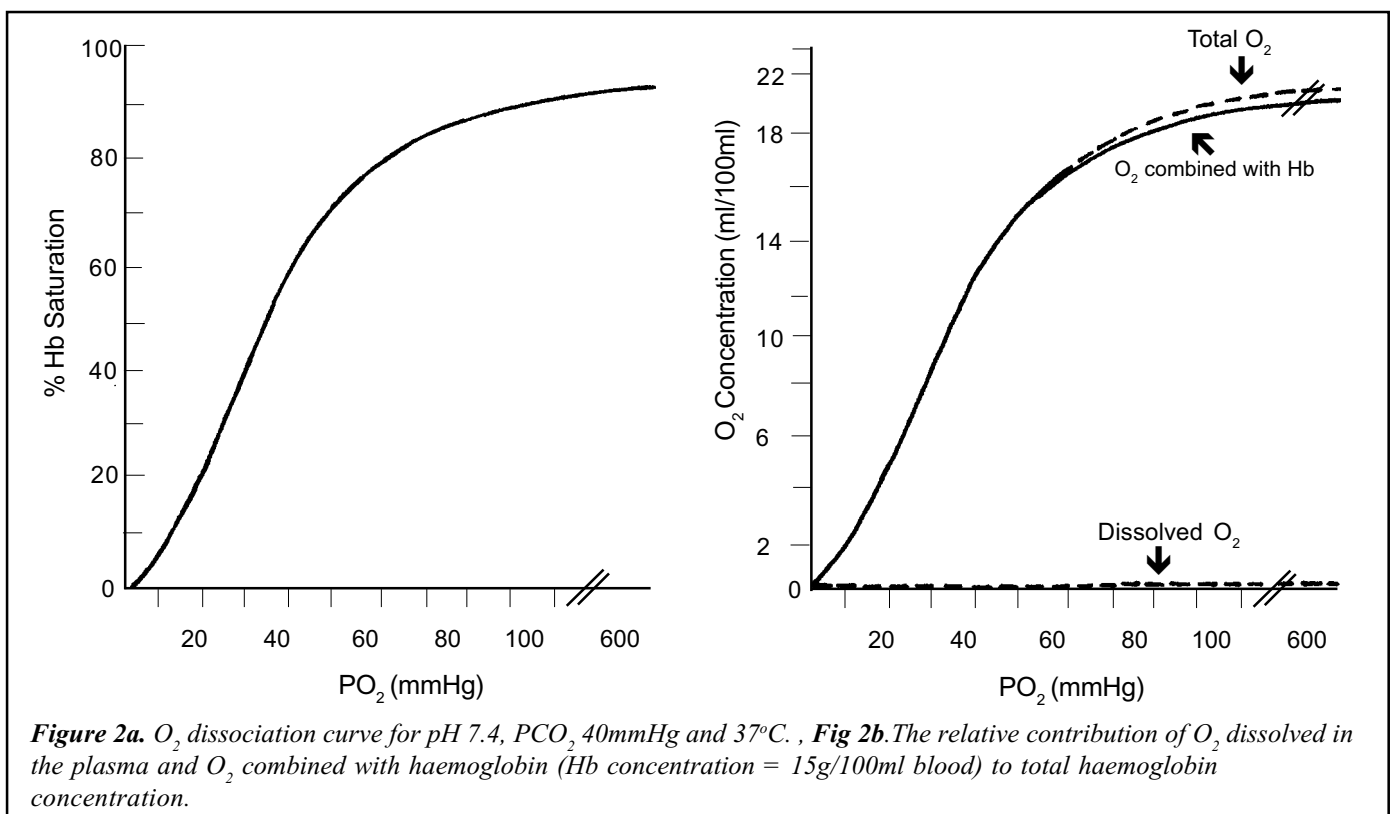
The quantity of oxygen made available to the body in one minute is known as the oxygen delivery and is equal to the cardiac output x the arterial oxygen content (see previously) ie. 5000ml blood/min x 200 ml $O_2$ /1000 ml blood = 1000ml  $O_2$ /min.

Oxygen delivery (mls  $O_2$ /min) = Cardiac output (litres/min) x Hb concentration (g/litre) x 1.31 (mls  $O_2$ /g Hb) x % saturation

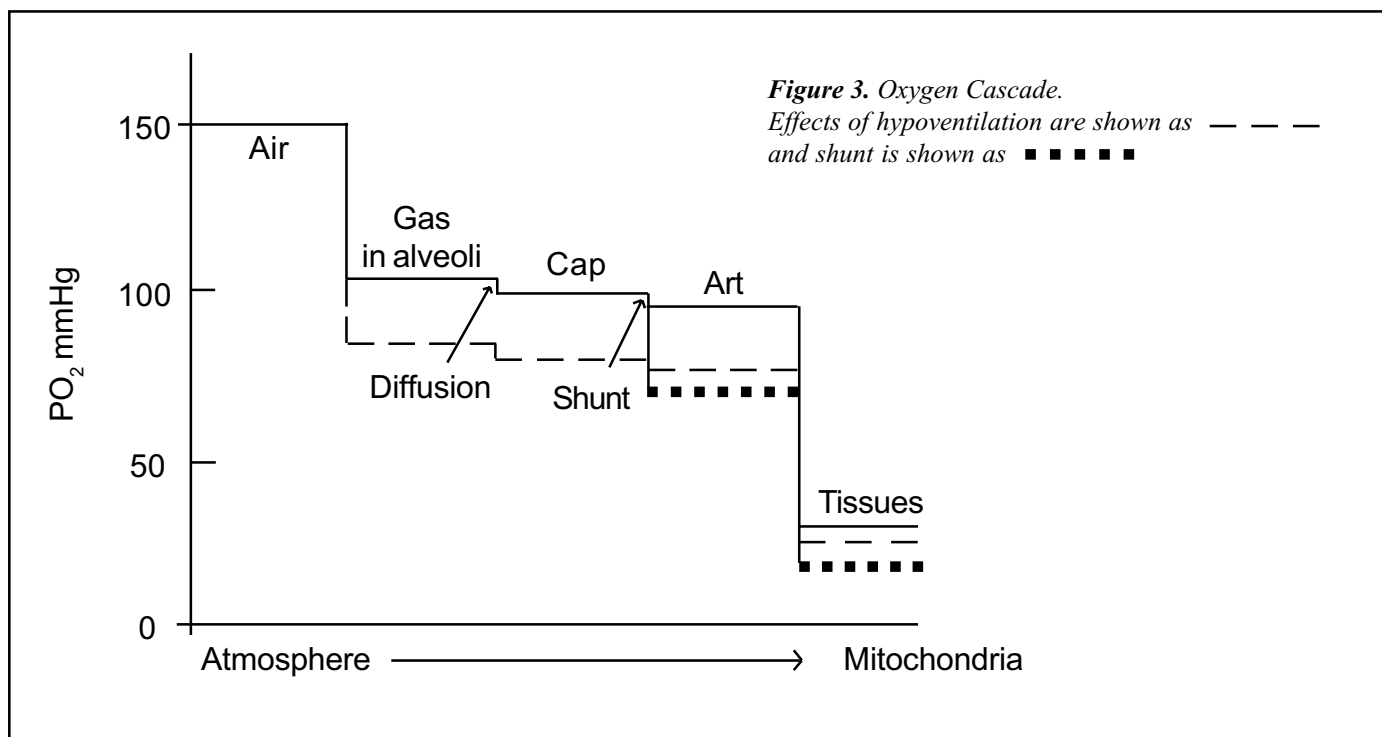
### Oxygen consumption

Approximately 250 ml of oxygen are used every minute by a conscious resting person (oxygen consumption) and therefore about 25% of the arterial oxygen is used every minute. The haemoglobin in mixed venous blood is about 70% saturated (95% less 25%).

In general there is more oxygen delivered to the cells of the body than they actually use. When oxygen consumption is high (eg. during exercise) the increased oxygen requirement is usually provided by an increased cardiac output – see formula above for how this works. However, a low cardiac output, a low haemoglobin concentration (anaemia) or a low haemoglobin  $O_2$  saturation will result in an inadequate delivery of oxygen, unless a compensatory change occurs in one of the other factors. Alternatively, if oxygen delivery falls relative to oxygen consumption the tissues extract more oxygen from the haemoglobin (the saturation of mixed venous blood falls below 70%)(a-b in figure 4). A reduction below point ‘c’ in figure 4 cannot be compensated for by an increased oxygen extraction and results in anaerobic metabolism and lactic acidosis.



**Figure 2a.**  $O_2$  dissociation curve for pH 7.4,  $PCO_2$  40mmHg and 37°C. , **Fig 2b.** The relative contribution of  $O_2$  dissolved in the plasma and  $O_2$  combined with haemoglobin (Hb concentration = 15g/100ml blood) to total haemoglobin concentration.



## OXYGEN STORES

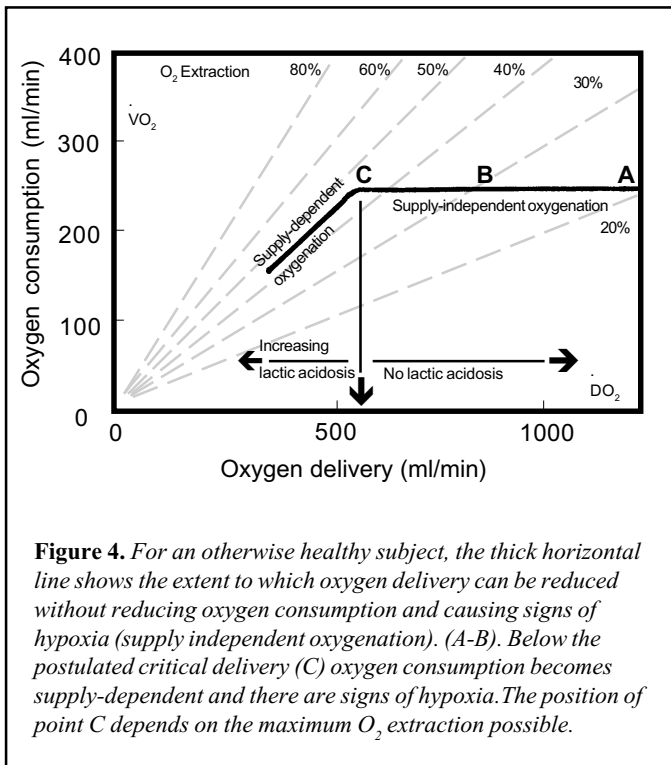
In spite of the great importance of oxygen, the stores of oxygen in the body are small and would be unable to sustain life for more than a few minutes. If breathing ceases, oxygen stores are limited to the oxygen in the lung and in the blood. The amount of oxygen in the blood depends on the blood volume and haemoglobin concentration. The amount of oxygen in the lung is dependent on the lung volume at functional residual capacity (FRC) and the alveolar concentration of oxygen. The FRC is the volume of air (about 3 litres in an adult) that is present in the lungs at the end of a normal expiration ie when the elastic recoil of the lung is balanced by the relaxed chest wall and diaphragm. While breathing air the total stores (oxygen in blood and lung) are small and because the major component of this store is the oxygen bound to haemoglobin (see figure 5) only a small part of these stores can be released without an unacceptable reduction in  $P_{aO_2}$  (when haemoglobin is 50% saturated with oxygen the  $P_{aO_2}$  will have fallen to 26 mmHg). Breathing 100% oxygen causes a large increase in the total stores as the FRC fills with oxygen. The major component of the store is now in the lung and 80% of this oxygen can be used without any reduction in haemoglobin saturation ( $P_{aO_2}$  still about 100mmHg). This is the reason why pre-oxygenation is so effective. (see later)

## OXYGEN TRANSPORT - THE EFFECTS OF ANAESTHESIA

Hypoventilation may occur during anaesthesia due to airway obstruction, the effects of volatile anaesthetic agents, opioids and other sedatives. In contrast, ketamine and ether anaesthesia (less than 1 MAC) cause less respiratory depression than other anaesthetic agents. Alveolar  $PO_2$  is a balance between the oxygen supplied by breathing and that used by metabolic processes in the body. Hypoventilation and a decreased inspired oxygen concentration will therefore cause a reduction in alveolar  $PO_2$  ( $P_{AO_2}$ ). The increased utilisation of oxygen when metabolic rate is raised such as with postoperative shivering or malignant hyperpyrexia also causes a reduction in alveolar  $PO_2$ .

If the  $P_{aO_2}$  falls to less than 60mmHg the aortic and carotid body chemoreceptors respond by causing hyperventilation and increasing cardiac output through sympathetic nervous system stimulation. This normal protective response to hypoxia is reduced by anaesthetic drugs and this effect extends into the post-operative period.

Following induction of anaesthesia there is a rapid reduction in FRC that results in airway closure - small airways, particularly in dependant parts of the lung, collapse and remain closed throughout the respiratory cycle. This results in some alveoli not being ventilated at all (true shunt). Ventilation/perfusion ( $V/Q$ ) mismatch is also increased.



Anaesthesia causes a 15% reduction in metabolic rate and therefore a reduction in oxygen requirements. Artificial ventilation causes a further 6% reduction in oxygen requirements as the work of breathing is removed. Anaesthetic agents do not affect the carriage of oxygen by haemoglobin.

**THE PRACTICAL USE OF OXYGEN**

*Inspired oxygen concentration*

The efficiency of oxygenation during anaesthesia is reduced due to hypoventilation and venous admixture. An inspired oxygen in the range of 25%-30% is usually effective in restoring the PaO<sub>2</sub> to normal when hypoxaemia is due to hypoventilation. (figure 6) When hypoxaemia is due to venous admixture it is only possible to restore the PaO<sub>2</sub> by increasing the inspired oxygen concentration if the venous admixture does not exceed the equivalent of a 30% shunt. (figure 7) The inspired oxygen concentration during maintenance of anaesthesia should routinely be increased to 30% whenever possible to compensate for hypoventilation and shunt which normally accompany anaesthesia. Additional oxygen may need to be administered to patients at risk of decreased oxygen delivery (anaemia or decreased cardiac output) or increased oxygen consumption (fever).

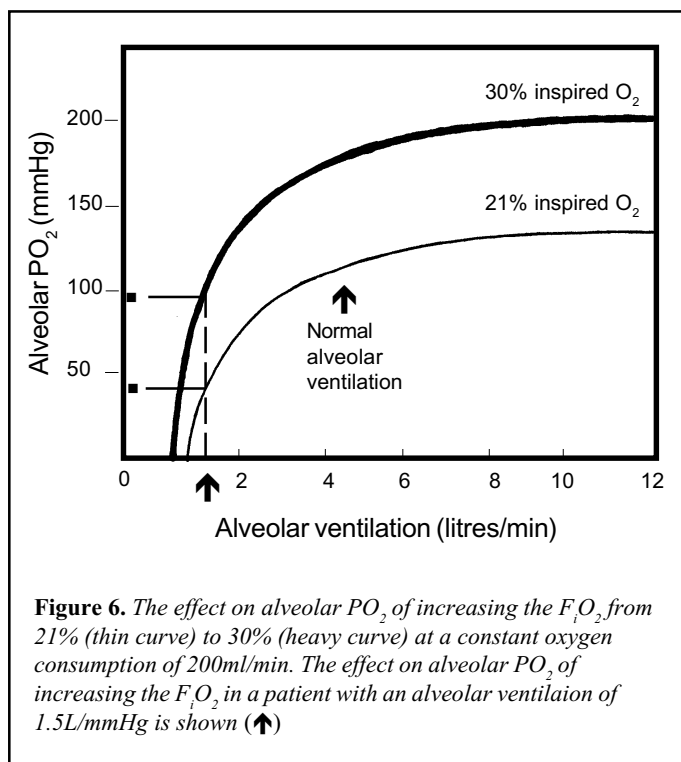
*Pre-oxygenation*

The small volume of the oxygen stores in the FRC of a patient breathing air means that there will be a rapid fall in oxygen saturation during apnoea (e.g. following induction of anaesthesia, during laryngospasm or during upper airway obstruction). Pre-oxygenation involves the breathing of 100% oxygen for three minutes through an anaesthetic circuit with a face mask firmly applied to the face. This is the time taken to replace the nitrogen in the FRC with oxygen using normal tidal ventilation. Although FRC falls on induction of anaesthesia the extra oxygen contained within the FRC provides an essential store of oxygen for periods of apnoea, such as may occur during rapid

Atelectasis (collapsed alveoli) also develops rapidly in the dependant lung regions and results in true shunt. As explained earlier, airway closure, V/Q mismatch and shunt will cause the oxygen saturation in the pulmonary veins to be less than in the pulmonary capillaries of ventilated alveoli. This 'venous admixture' increases from 1% to around 10% following induction of anaesthesia. With the possible exception of patients spontaneously breathing while anaesthetised with ketamine, this increase in venous admixture occurs irrespective of the anaesthetic agent used and whether muscle relaxants are used or not. It should be viewed as an unavoidable adverse effect of anaesthesia. Volatile anaesthetic agents suppress hypoxic pulmonary vasoconstriction, and blood flow to underventilated or collapsed alveoli is not reduced. Many anaesthetic agents depress cardiac output and therefore decrease oxygen delivery.

**Fig 5. Principal stores of oxygen in the body**

	While breathing AIR	While breathing 100% O <sub>2</sub>
In the lungs (FRC)	450ml	<b>3000ml</b>
In the blood	850ml	950ml
Dissolved or bound in tissues	250ml	300ml
<b>Total</b>	<b>1550ml</b>	<b>4250ml</b>



sequence induction or difficult intubation. Patients with a small FRC (infants, pregnancy, obesity) or a low haemoglobin concentration and therefore smaller oxygen stores desaturate more rapidly and pre-oxygenation is especially indicated in these patients.

#### Anoxic gas mixtures

If, during the course of an anaesthetic 100% nitrous oxide is given to the patient in error, the fall in alveolar  $PO_2$  will be much more rapid than during apnoea. The alveolar  $PO_2$  can fall to dangerously low levels in as little as 10 seconds. This is because the oxygen in the patient's lungs and blood (oxygen stores) is being actively washed out with each breath that contains no oxygen. The fall in  $PO_2$  is therefore more rapid than would occur if it was only being used up by the metabolic needs of the body (250ml/min).

#### Crisis management

When managing emergencies during anaesthesia consideration should always be given to the immediate administration of 100% oxygen while the cause is found and rectified. It is the most appropriate treatment for acute deterioration in cardiorespiratory function.

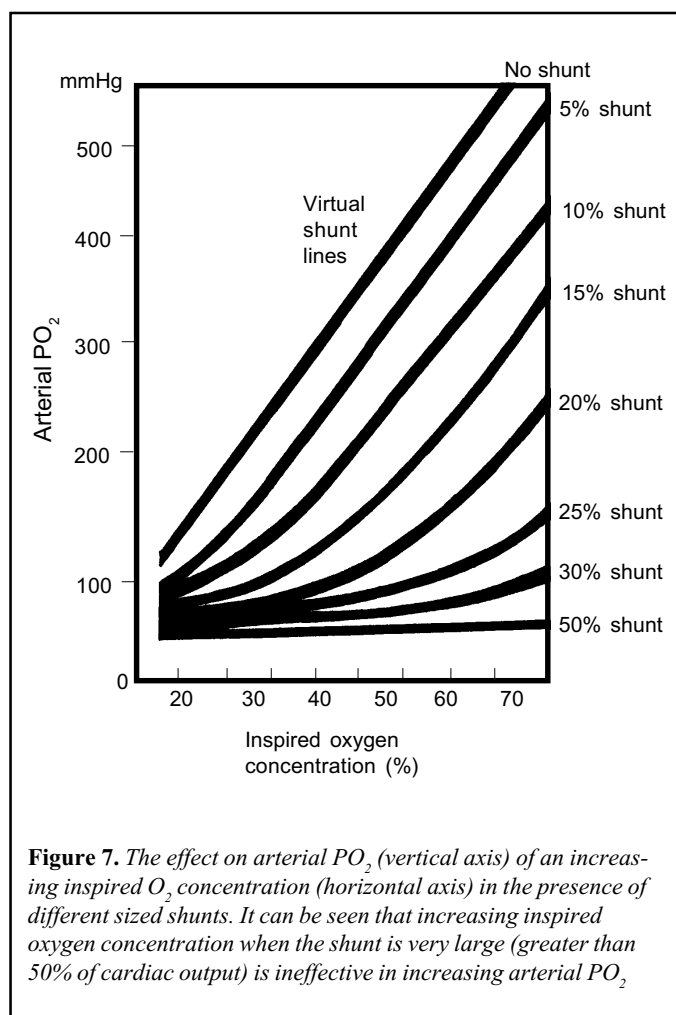
#### Diffusion hypoxia

Nitrous oxide is forty times more soluble in blood than nitrogen. When nitrous oxide is discontinued at the end of anaesthesia, nitrous oxide diffuses out of the blood into the alveoli in large volumes during the next 2 - 3 minutes.

If the patient is allowed to breathe air at this time the combination of nitrous oxide and nitrogen in the alveoli reduces the alveolar  $PO_2$ . This is called diffusion hypoxia and is avoided by increasing the inspired concentration of oxygen by the administration of 100% oxygen for 2 - 3 minutes after discontinuing nitrous oxide.

#### Postoperative oxygen

The causes of increased venous admixture (ventilation/perfusion mismatch, shunt and airway closure) and the abnormal response to hypoxia continue into the postoperative period for a number of days following major surgery. Postoperative hypoventilation is common and may be due to the residual effect of anaesthesia, the use of opioid analgesia, pain or airway obstruction. Shivering in the immediate postoperative period causes an increase in oxygen consumption. Additional oxygen should therefore be given to all unconscious patients in recovery and to those awake patients who either shiver, hypoventilate, desaturate or who are considered to be at special risk (eg. ischaemic heart disease).



Postoperatively, on the ward, episodes of airway obstruction during sleep are common and may aggravate borderline oxygenation due to the above factors. This is due to the use of opioid analgesia and a change in sleep pattern that occurs on the second and third postoperative nights. It is clear that after major surgery the risk of hypoxaemia extends well into the postoperative period. Small degrees of cyanosis are not easy to detect clinically, especially in anaemic patients, and therefore oxygen should be given to these patients wherever possible. It is especially important to give it overnight to patients at special risk (ischaemic heart disease). Postoperative pain should be effectively treated (see Update 7) as patients in pain following abdominal or thoracic surgery will be reluctant to breathe deeply. If opioid analgesics are indicated, hypoventilation should be anticipated, and oxygen given.

### PROBLEMS ASSOCIATED WITH OXYGEN ADMINISTRATION

It has been suggested that high concentrations of oxygen (90-100%) administered to patients for a prolonged period (several days) may cause pulmonary damage. There is little evidence to support this and should never prevent its use in treating severe hypoxia.

High concentrations of oxygen will encourage collapse of alveoli with low ventilation/perfusion ratios. Oxygen is rapidly and completely absorbed from these alveoli, and when it is the only gas being given, these underventilated alveoli collapse. When air and oxygen is used, the nitrogen present is absorbed more slowly and prevents the alveolus from collapsing.

Oxygen therapy may rarely depress ventilation in patients suffering from severe chronic obstructive airways disease. Some of these patients lose their sensitivity to carbon dioxide and rely on hypoxia to stimulate breathing. In these patients, when high concentrations of oxygen are given, serious hypoventilation and hypercapnia can result due to the fact that their hypoxia is reversed. This is extremely rare.

In the second part of this article we plan to discuss the more practical aspects of oxygen production and storage and the equipment needed to safely administer oxygen to patients.

#### References:

1. Nunn JF. Applied Respiratory Physiology (3<sup>rd</sup> Edition). Butterworths 1987
2. West JB. Respiratory Physiology (4<sup>th</sup> Edition). Williams and Wilkins 1990

## PHARMACOLOGY OF VASOPRESSORS AND INOTROPES

*Dr Karen Gilmore, Frenchay Hospital, Bristol, UK & Christine Nyanzi, Gihundwe Hospital, Rwanda.*

A “vasopressor” causes vasoconstriction and an “inotrope” increases the force of cardiac contraction. Vasopressors and inotropes work via the Autonomic Nervous System.

**Neurotransmission at postganglionic receptors.** The postganglionic receptors of the Parasympathetic Nervous System PNS are termed muscarinic, and acetylcholine (ACh) is the neurotransmitter. The equivalent receptors in the Sympathetic Nervous System (SNS) are noradrenergic receptors and noradrenaline (Norad) is the endogenous (naturally occurring) neurotransmitter (table 1).

These noradrenergic receptors are further subdivided, the subdivisions relevant to this article are Alpha1 ( $\alpha_1$ ), Beta1 ( $\beta_1$ ), Beta2 ( $\beta_2$ ) and Dopamine (D). The main actions of each receptor subtype are as shown in table 2.

### VASOPRESSORS AND INOTROPES

This group of drugs is useful for resuscitation of seriously ill patients, and for the treatment of hypotension in theatre. All of these drugs act directly or indirectly on the SNS, but the effect of each varies according to which sympathetic receptor the drug has greatest affinity for. The duration of action also varies. **Direct acting drugs** act by stimulating the SNS receptor whereas **indirect acting drugs** cause the release of noradrenaline from the receptor which produces the effect. Some drugs have a mixed effect.

#### ADRENALINE (EPINEPHRINE)

Adrenaline acts on  $\alpha_1$ ,  $\beta_1$  and  $\beta_2$  receptors. It is said to prepare the body for a “fight or flight” response.

Table 1

	<b>Preganglionic receptor type (and neurotransmitter)</b>	<b>Post ganglionic receptor type (and neurotransmitter)</b>
PNS	Nicotinic (Ach)	Muscarinic (Ach)
SNS	Nicotinic (Ach)	Noradrenergic (Norad)

**Actions**

*CVS:* Increased heart rate and force of contraction produce an increase in cardiac output. Systolic blood pressure (SBP) rises, but with low doses diastolic blood pressure (DBP) may fall due to vasodilation and increased blood flow through skeletal muscle beds ( $\beta_2$ ). At higher doses the vasoconstrictor effects of  $\alpha_1$  stimulation become more apparent, causing the cool pale extremities of a frightened person.

*RS:* Bronchial smooth muscle is relaxed resulting in bronchodilation ( $\beta_2$ ).

*Other:* Adrenaline mobilises glucose from glycogen and raises blood sugar. Pupillary dilation (mydriasis) occurs.

**Side effects** Ventricular arrhythmias, hypertension. Care with halothane anaesthesia as arrhythmias may occur.

**Preparation** 1:1000 i.e. 1mg in 1 ml.  
1:10,000 i.e. 1mg in 10ml

**Indications and doses**

*Cardiac Arrest* - see page 21

*Anaphylactic shock* - 1:10,000 adrenaline given iv in 1 ml doses until effective. If no iv access available then 0.5ml of 1:1,000 im.

*Additive to local anaesthetic* - add adrenaline to local anaesthetic to make a concentration of 1:200,000 - see page 50

*Acute severe asthma attack unresponsive to normal treatment* may require infusions of adrenaline, though 0.5ml of 1:1000 s/c may be used.

*Septic shock* - require infusions of adrenaline

**Length of action** Short, few minutes only with intravenous bolus.

**EPHEDRINE**

Ephedrine acts directly on  $\beta_1$  and  $\beta_2$  receptors, and indirectly on  $\alpha_1$  receptors by causing noradrenaline release.

**Action** It causes a rise in blood pressure and heart rate, and some bronchodilation.

**Side effects** May cause tachycardia and hypertension. Possible arrhythmias if used with halothane.

**Preparation** 3% or 5% solution: 1 ml ampoules.

**Indications** Low blood pressure due to vasodilation e.g. following spinal or epidural anaesthesia and drug overdoses. Best vasopressor to use in pregnancy as it does not reduce placental blood flow.

**Dose** 3-10 mg boluses iv, repeat until effective. Maximum dose is 60mg.

**Length of action** 5-15 minutes, repeated doses less effective (i.e. it demonstrates tachyphylaxis).

**METHOXAMINE**

Methoxamine acts on  $\alpha_1$  receptors.

**Actions** Increases blood pressure. There may be a reflex decrease in heart rate, and therefore it is good for hypotension with tachycardia. Useful during spinal anaesthesia.

Table 2

$\alpha_1$	Peripheral arteriolar vasoconstriction
$\beta_1$	Cardiac increased heart rate and force of contraction.
$\beta_2$	Bronchial smooth muscle dilation. Vasodilation in skeletal muscle. Also some cardiac effects.
D	Increased renal blood flow

For a full explanation of receptors and their actions refer to *Update in Anaesthesia 1995:5*

**Side effects** May produce bradycardia

**Dose** 2-4mg boluses IV, repeated as necessary.

### METARAMINOL

Acts directly on  $\alpha$ 1 receptors and also causes noradrenaline and adrenaline release.

**Actions** Increases blood pressure and cardiac output. Less likely to cause a reflex bradycardia than methoxamine or phenylephrine.

**Dose** - 1mg boluses iv, 2-10mg s/c or im, by infusion at 1-20mg/hr.

### PHENYLEPHRINE

Acts directly on  $\alpha$ 1 receptors,

**Action** Hypertension and a reflex decrease in heart rate.

**Dose** 2-5mg im or sc, 0.1-0.5mg iv, by infusion 20-50mcg/min.

### INOTROPES GIVEN BY INFUSION

Adrenaline is the most commonly available inotrope, and in many cases the most appropriate drug to maintain blood pressure. When other inotropes are available, some may offer advantages in certain situations. The inotropes listed below are only given by infusion unless a bolus dose is stated. They are mostly very short acting, their effects lasting from a few seconds to one or two minutes and should be given via a central line (except for aminophylline and salbutamol) via an infusion controller. The patient must be closely monitored, particularly the ECG and blood pressure. Tachycardia, arrhythmias, and hypertension or hypotension are side effects of these drugs. Although called inotropes some of these drugs also have vasoconstrictor properties.

### NORADRENALINE

Acts mainly on  $\alpha$ 1 receptors with few effects on  $\beta$  receptors.

**Actions** Increases blood pressure by vasoconstriction. Less likely to cause tachycardia than adrenaline.

**Indications** Septic shock where peripheral vasodilation may be causing hypotension.

**Cautions** Acts by increasing afterload and therefore not appropriate for use in patients in cardiogenic shock. Blood supply to kidneys and peripheries may be reduced.

**Dose** - 1-30mcg/min

- Add 4mg to 250ml 0.9% NaCl or 5% dextrose to give 16mcg/ml.  
Run at 0-112ml/hr

### DOPAMINE

Acts on D,  $\beta$ 1,  $\beta$ 2 and  $\alpha$ 1 receptors, depending on the dose administered.

**Actions** Dose dependent. It used to be popular to increase urine output via its effect on the D receptors in the kidney. However, less commonly used for this purpose as it does not prevent renal failure.

**Indications** Hypotension.

**Dose** 1-2mcg/kg/min - acts on D receptors usually increasing urine output

2-10mcg/kg/min - also acts on  $\beta$  receptors to increase cardiac output

>10mcg/kg/min - additionally has effects on  $\alpha$ 1 receptors to vasoconstrict.

- Add 3mg/kg (body weight) to 50mls 0.9%NaCl or 5% glucose
- 1ml/hr = 1mcg/kg/min

### DOBUTAMINE

Acts on  $\beta$ 1 and  $\beta$ 2, with minimal action on  $\alpha$ 1 receptors.

**Actions** It increases cardiac output and reduces afterload ( $\beta$ 2 effects on skeletal muscle).

**Indications** Cardiogenic shock.

**Dose** 2-30mcg/kg/min

- Add 3mg/kg to 50mls 0.9%NaCl or 5% glucose
- 1ml/hr = 1mcg/kg/min

### DOPEXAMINE

Acts on  $\beta$ 2 and D receptors.

**Actions** It increases cardiac output and reduces afterload. Increases blood supply to the kidneys and possibly also the gastrointestinal tract.

**Dose** 0.5-6mcg/kg/min



**SALBUTAMOL**

Acts on  $\beta_2$  receptors

**Actions** Relaxes bronchial smooth muscle i.e. bronchodilation, may increase heart rate

**Indications** Severe acute asthma.

**Dose** By infusion 5-20mcg/min. Can also be given in bolus form iv in the initial treatment of an attack at a dose of 5mcg/kg over several minutes.

**ISOPRENALINE**

Acts on  $\beta_1$  and  $\beta_2$  receptors

**Actions** Main action is increased heart rate. Also increased force of contraction, and bronchodilation.

**Indications** Complete heart block, overdose of beta blocker or severe bradycardia unresponsive to atropine. Can be used to treat asthma, but less suitable than drugs that act only on  $\beta_2$  receptors e.g. salbutamol

**Dose** 0.02-0.2mcg/kg/min by infusion  
5-20mcg bolus iv

**PHOSPHODIESTERASE INHIBITORS**

(e.g. AMINOPHYLLINE, ENOXIMONE)

Prevent breakdown of cAMP by enzyme phosphodiesterase: this produces effects at  $\beta_1$  and  $\beta_2$  receptors.

**Actions** Inodilation i.e. increased rate and force of contraction, vasodilation in skeletal muscle. Also bronchodilation.

**Indications** Aminophylline: asthma, cardiac failure.  
Enoximone: cardiac failure in patients failing to respond to dobutamine

**CLINICAL CASE STUDY – USE OF VASOPRESSORS****Lower segment Caesarean section (LSCS) under spinal anaesthesia**

A patient is scheduled for LSCS under spinal

anaesthesia. An iv infusion is set up and 1000 mls of Hartmanns run in whilst the spinal is performed. The patient is placed supine with a 15-degree left-lateral tilt to minimise aortocaval compression (i.e. pressure from the uterus on the inferior vena cava reducing venous return to the heart).

Despite good positioning and iv fluids, hypotension is very likely at this stage because of vasodilation due to the spinal. The patient should be given ephedrine in boluses of 6-9mg, which may need to be repeated several times. Alternatively, 30-60mg of ephedrine can be added to the intravenous infusion, and the rate titrated according to the BP. The SBP should be maintained above 100mmHg. (A hazard of adding ephedrine to the infusion is that the anaesthetist may forget to reduce the rate of infusion when the BP has returned to normal, and the patient may become dangerously hypertensive.)

Once the baby has been delivered aortocaval compression is no longer a problem, and further ephedrine is not usually required. If hypotension persists, ensure that hypovolaemia is not the cause. Intravenous fluids should be given to restore blood volume, rather than vasopressors. Ephedrine is the best vasopressor for LSCS because it has fewest effects on placental blood supply. If ephedrine is not available another vasopressor should be used. Alternatively small doses of adrenaline (20-50mcg) can be given, in a dilute preparation.

**Summary**

The common causes of hypotension during LSCS under spinal anaesthesia are:

- Vasodilation - treat with fluids and ephedrine
- Aortocaval compression – tilt patient 15 degrees to left
- Bleeding – replace blood loss with intravenous fluids

## ANAESTHESIA AND THE LIVER

*Dr Matthew Roberts, Royal Army Medical Corps and Dr Ray Towey, Mwanza Hospital, Tanzania.*

Anaesthesia and surgery in patients with problems related to the liver cause concern because of the central role of the liver in many of the body's metabolic and synthetic functions. The process of anaesthesia may adversely effect these functions and equally the patient's response to anaesthetic drugs and surgery may be influenced by hepatic dysfunction. It is therefore necessary for those practicing anaesthesia anywhere in the world to have an understanding of liver function in normal physiology.

### Liver Functions

The liver conjugates bilirubin, produced from the degradation of the haemoglobin of red cells that are at the end of their normal life span. This now water-soluble form of bilirubin is then excreted into the bile ducts and thence into the small intestine. Also passed to the gut are the bile salts produced by the liver and necessary for the absorption of the fat-soluble vitamins A, D, E and K. Vitamin K is essential for the production of prothrombin and some other protein factors that are essential for the normal clotting of blood.

Synthesis of many proteins takes place in the liver including most clotting factors and many carrier proteins, such as albumin, which to a varying degree bind drugs used during anaesthesia. The liver is also central in lipid metabolism with cholesterol and triglycerides synthesised here. The synthesis and breakdown of glycogen in the liver is pivotal in carbohydrate metabolism. It stores glycogen and releases glucose into the blood when the blood glucose falls for any reason.

The liver is responsible for the biotransformation of drugs either by oxidation or conjugation in order to render them water-soluble and therefore more easily excreted in the urine or bile.

### The Effect of Anaesthesia and Surgery on Liver Function

Inhalational anaesthetics effect carbohydrate metabolism in several ways. Ether, unlike the newer agents, enhances the breakdown of glycogen in the liver. Halothane has been shown, experimentally, to decrease the rate of glycogenesis, inhibit insulin release and inhibit the effect of insulin on the tissues. The catecholamine mediated stress response to surgery and trauma also increases glycogenolysis, so the overall effect of both surgery and inhalational anaesthesia is to elevate blood glucose.

Protein synthesis is reduced by halothane but this is of questionable clinical significance.

Halothane and ether both inhibit the Cytochrome P450 enzyme system, slowing the oxidative metabolism of drugs; gluconuride conjugations are not effected. The following drugs, as a result, have a prolonged half-life in the presence of halothane, fentanyl, ketamine, lignocaine, pancuronium and propranolol.

Hepatic blood flow is decreased by halothane in parallel with an overall decrease in cardiac output. Intermittent positive pressure ventilation and decreases in carbon dioxide potentiate this effect while hypoventilation and increased carbon dioxide results in an increase in hepatic blood flow. These effects are unlikely in isolation to lead to liver hypoxia or damage.

Opioids such as morphine, pethidine and fentanyl are known to be able to cause spasm of the Sphincter of Oddi and increase biliary pressure, the effect lasting about two hours in the case of morphine. This should not preclude their use to provide adequate analgesia in biliary surgery.

### Halothane and Jaundice

It was discovered some years ago that some adult patients can, very rarely, become jaundiced from severe hepatic damage after a second halothane anaesthetic. The incidence of this *halothane hepatitis* in adults is thought to be 1:7000-30,000 halothane anaesthetics. It is even rarer in paediatric patients and with the newer volatile agents. The risk is thought to be higher in women, the middle aged and the obese.

The cause of so-called halothane hepatitis is not fully established and may be multifactorial. The effect seems to be related to the degree of metabolism of the volatile agent, so toxic metabolites may be involved. The onset time of the jaundice is shorter with increasing numbers of exposures to halothane and there have been suggestions of a possible immunological cause. It has also been suggested that reduced hepatic blood flow and hypoxia are to blame. In most cases of post operative jaundice halothane is unlikely to be the cause so given the rarity of the condition, and the limited choice of agents in the developing world, anaesthetists under these circumstances should not hesitate to use halothane whenever it is appropriate.

Interestingly there is no evidence that halothane will precipitate hepatic deterioration in patients with jaundice of a different origin. Ether may cause a transient depression of liver function but does not cause significant damage.

**Anaesthesia and Surgery in Patients with Liver or Biliary Dysfunction** Before the anaesthetist can assess the implications of a patient's hepatic disease for the conduct of an anaesthetic, it is necessary to understand the various causes of jaundice - the cardinal sign of liver disease.

**Jaundice** can be prehepatic (haemolytic), hepatic (hepatocellular) or posthepatic (obstructive) in origin. An example of prehepatic jaundice is in the haemolysis that accompanies the breakdown of a large haematoma, or the jaundice that can occur when there is a massive intravascular haemolysis - as in some forms of malaria or in sickle cell anaemia. In these situations the hepatocellular function is normal but overwhelmed and so the increased bilirubin is for the most part unconjugated. Protein and carbohydrate metabolism is intact and there is no reduction in the absorption of Vitamin K or production of clotting factors.

Where there is actual hepatocellular dysfunction, as in hepatitis or cirrhosis, there may be evidence of decreased protein synthesis, with oedema and ascites, signs of delayed clotting only partly reversed by vitamin K administration, and even encephalopathy. Hepatic encephalopathy is a condition of progressive deterioration of cerebral function from drowsiness to coma, in patients with liver disease, probably caused by toxic metabolites of proteins in the large intestine not adequately detoxified by the liver. These patients may show other signs of chronic liver disease as listed in Table 1.

Patients with active liver disease, such as hepatitis, are at high risk of deterioration during surgery and this should be avoided or delayed where possible. The poorest outcome is predicted by the combination of deranged clotting, oedema and encephalopathy.

**Obstructive Jaundice** Biliary obstruction is the most likely cause of jaundice to be encountered by the anaesthetist in the developing world. It can result from a stone in the common bile duct, pancreatic tumour or ascending cholangitis where the bile and biliary tree are infected. Hepatocellular function is normal (although it may deteriorate in prolonged obstruction) so the excess plasma bilirubin is chiefly conjugated. As conjugated bilirubin is water-soluble it will be excreted in the urine which becomes dark. Stools are pale as a result of poor lipid absorption. Although protein synthesis is normal, the production of vitamin K dependant clotting factors will be reduced, as the absorption of vitamin K is dependent on the excretion of bile salts into the small intestine. The clotting time can, therefore, be prolonged but this can be readily reversed by parenteral administration of vitamin K. Surgery in these cases is to remove or bypass the obstruction or to drain infected obstructed bile.

#### Liver Function Tests

Where laboratory investigations are available these should be part of any preoperative assessment of patients with probable liver or biliary dysfunction. As well as providing an indication of severity, they may help differentiate between prehepatic, hepatocellular and obstructive jaundice.

Jaundice is the outward sign of an elevation of serum bilirubin. As already discussed a predominantly conjugated bilirubin suggests an obstructive cause, while unconjugated bilirubin points to a prehepatic problem. Hepatic disease may result in a predominantly unconjugated or a mixed pattern. Dark urine containing bilirubin suggests biliary obstruction.

Protein and albumin levels are usually normal in prehepatic or obstructive jaundice, whereas low values may indicate impaired synthetic activity in the liver resulting from hepatocellular damage.

**Table 1** Some Signs of Liver Disease

Jaundice	Hepatomegaly	Spider Naevi	Splenomegaly
Scratch Marks	Ascites	Palmer Erythema	Dilated Abdominal Veins
Peripheral Oedema	Finger Clubbing	Testicular Atrophy	Bruising
Gynaecomastia	Confusion/Coma		

A measure of clotting impairment is the Prothrombin Time, given as an absolute value or compared to a control. The WHO recommends the use of the International Normalized Ratio (INR) which is the Prothrombin Time ratio obtained when using a WHO International Reference Thromboplastin. An elevated INR may indicate impaired synthesis of clotting factors due to hepatocellular damage or malabsorption of vitamin K due to biliary obstruction. The INR is also used to monitor therapy with the anticoagulant warfarin.

Plasma glucose should be measured because of the pivotal position the liver plays in carbohydrate metabolism discussed earlier.

Alanine Transaminase (ALT) and Aspartate Transaminase (AST) are enzymes that are released into the circulation by damaged hepatocytes. Raised levels of these enzymes tend, therefore, to indicate hepatocellular damage. AST can also be elevated in other circumstances such as myocardial infarction so other indicators of liver disease must be sought.

Alkaline Phosphatase (ALP) is an enzyme localized near the bile canaliculi and is elevated in biliary obstruction. Again this enzyme is not specific to hepatobiliary disease, it is, for example, raised in malignant bone disease. An accompanying rise in Gamma-glutamyl Transferase (Gamma GT) suggests that the ALP is from the liver.

Other laboratory investigations of use in the preoperative assessment of these patients are the haemoglobin, a blood film (for evidence of haemolysis), plasma urea and creatinine.

### **Hepatorenal failure**

One of the main concerns in surgery for biliary obstruction is the development of renal failure. This serious condition has been recognized since the 1950's although the cause is not completely understood. It may be related to pre and perioperative dehydration and hypovolaemia, falls in renal blood flow during surgery, a direct effect of the excess conjugated bilirubin on the renal tubules or possibly an increased absorption of endotoxin from the gut.

The key to managing this condition is to avoid it developing by ensuring adequate hydration and a urine flow of at least 50mls/hr in the average adult patient. In most patients with moderately elevated bilirubin this can be achieved with simple fluid loading for 12 hours before surgery using 0.9% NaCl and during the operation. If the urine output is not

maintained in this way mannitol 10% should be administered until an adequate diuresis is achieved.

Where the bilirubin is greatly elevated (>140 micromols/litre), patients should be given intravenous fluids during the 24 hours before surgery and for 36 hours postoperatively. In these cases mannitol 10% 0.5-1g/kg should be administered prior to surgery, although the patient must not be allowed to become dehydrated as a result of an over-zealous diuresis.

Hepatorenal failure does not appear to be a major risk in patients with prehepatic jaundice.

### **Drug Elimination**

In biliary obstruction there is no significant alteration in drug handling and normal doses of thiopentone, opiates, benzodiazepines and muscle relaxants are given. Although the nondepolarising muscle relaxant vecuronium is partly cleared through the bile, the normal rapid uptake by the liver cells is unchanged and there is no effect on the half-life.

In contrast, where there is significant hepatocellular dysfunction as in advanced cirrhosis or acute hepatitis, drug handling can be disturbed. Decreased synthesis leads to lowered levels of carrier proteins in the blood. This means that for the same dose of a highly protein bound drug, such as thiopentone, there will be a greater level of unbound and therefore active drug. Smaller doses are required. The liver produces serum cholinesterase, responsible for the breakdown of suxamethonium, but a reduction of 50% is required for any clinically significant prolongation of the effect of this drug, which is uncommon.

Drugs that are metabolised in the liver may have prolonged half-lives when hepatocellular function is poor. This may lead to accumulation of drugs given by infusion and where drugs given in repeat or top up doses, such as muscle relaxants, the interval between doses should be prolonged. Ideally drugs such as induction agents should be titrated to effect and neuromuscular blockade should be monitored with a peripheral nerve stimulator.

### **Regional Anaesthesia**

Spinal and epidural anaesthesia carries the risk of epidural haematoma and paralysis if there is abnormal clotting but there are otherwise no special precautions. The half-life of lignocaine is prolonged in liver failure but this is not significant when used in regional anaesthesia.

## Conclusions

It is important to assess each patient with disease of the liver or bile ducts for signs of liver dysfunction, bearing in mind that conditions involving the liver do not necessarily disturb normal hepatic physiology – eg amoeboma and schistosomiasis. Patients with severe liver failure are unlikely to present for surgery in the developing world unless they suffer unrelated trauma. These patients would be at high risk of deterioration or death during surgery; the

combination of clotting abnormality, oedema and encephalopathy predicts a poor outcome. If these patients must have surgery vitamin K should be administered preoperatively and care taken with drug dosage. The more likely indication for surgery is biliary obstruction. Here drug dosing is less problematic, the more important issues being correction of impaired clotting and avoidance of the hepatorenal syndrome.

## RESUSCITATION FROM CARDIAC ARREST

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In recent years, organisations such as the European Resuscitation Council, the American Heart Association and the International Liaison Committee on Resuscitation have produced guidelines in an attempt to improve the quality of cardiopulmonary resuscitation (CPR). They are based on international consensus views and the most recent of them, relating to Advanced Life Support, were published in 1998. The techniques of CPR, based on such guidelines, have now become a standard part of health professional training in many parts of the world.

The aim of this article is to provide an overview of resuscitation based on these guidelines and will be confined to the management of cardiac arrest including some comment on the more specialised areas of electrocution, drowning and arrests related to anaesthesia.

### BACKGROUND PHYSIOLOGY AND PATHOPHYSIOLOGY

The maintenance of normal tissue metabolism relies principally on an adequate delivery of oxygen, in a functioning circulation. Failure of delivery rapidly results in the following changes:

**Hypoxia** After a brief period of cardiac arrest,  $P_{aO_2}$  falls dramatically as oxygen continues to be consumed. In addition, progressive accumulation of carbon dioxide shifts the oxygen-haemoglobin dissociation curve to the right. This initially improves oxygen transfer to the tissues but

without further delivery tissue hypoxia ensues. In the brain, the  $P_{aO_2}$  falls from 13kPa to 2.5 kPa within 15 seconds and consciousness is lost. After a minute, the  $P_{aO_2}$  will have fallen to zero.

**Acidosis** The brain and heart have a relatively high rate of oxygen consumption (4mls/min and 23mls/min respectively) and  $O_2$  delivery to them will fall below critical levels during cardiac arrest. In the case of ventricular fibrillation, myocardial metabolism continues at an approximately normal rate, exhausting oxygen and high energy phosphate supplies. Acidosis then arises as the result of increased anaerobic metabolism and the accumulation of carbon dioxide in the tissues.

The degree of acidosis developing in the brain, even with basic life support, will threaten tissue survival within 5 - 6 minutes. Also, in the heart, even with the restoration of a perfusing rhythm, acidosis depresses contractility and there is a higher risk of further arrhythmias.

Cardiovascular collapse prompts a massive stress response. Catecholamines are released in large amounts, together with adrenal corticosteroids, anti-diuretic hormone and other hormonal responses. The possible detrimental effects of these changes include hyperglycaemia, hypokalaemia, increased lactate levels and a tendency towards further arrhythmias.

## CAUSES OF CARDIAC ARREST

There are many causes of cardiac arrest. In the developed world most are related to ischaemic heart disease. Table 1 lists other common causes.

**Table 1. Causes of Cardiac Arrest**

### Cardiac disease

- Ischaemic heart disease
- Acute circulatory obstruction
- Fixed output states
- Cardiomyopathies
- Myocarditis
- Trauma and tamponade
- Direct myocardial stimulation

### Circulatory causes

- Hypovolaemia
- Tension pneumothorax
- Air or pulmonary embolism
- Vagal reflex mechanisms

### Respiratory causes

- Hypoxia (usually causes asystole)
- Hypercapnia

### Metabolic changes

- Potassium disturbances
- Acute hypercalcaemia
- Circulating catecholamines
- Hypothermia

### Drug effects

- Direct pharmacological actions
- Secondary effects

### Miscellaneous causes

- Electrocution
- Drowning

## PREVENTION OF CARDIAC ARREST

Patients who develop a cardiac arrest may have been severely ill for some hours prior to the event. Warning signs such as: *hypotension, tachycardia, chest pain, dyspnoea, fever, restlessness or confusion* indicate that a patient is seriously ill. *Hypoxaemia, hypovolaemia and sepsis* may progress to cardiac arrest unless rapidly diagnosed and corrected. CPR for patients who are septic or hypovolaemic usually fails.

## RESUSCITATION

**The primary aim of resuscitation is to restore a beating heart and a functioning circulation.** This article considers basic and advanced life support.

## BASIC LIFE SUPPORT

Basic Life Support (BLS) establishes a clear airway followed by assisted ventilation and support of the circulation, all without the aid of specialised equipment. The recommended sequence for BLS is shown in figure 1.

When approaching a patient who appears to have suffered a cardiac arrest the rescuer should check that there are no hazards to himself before proceeding to treat the patient. Although this rarely arises in hospital, patients may suffer a cardiac arrest due to electric shocks or toxic substances. In these situations the rescuer may be in considerable danger, and must ensure that any hazard is taken account of and eliminated as a risk.

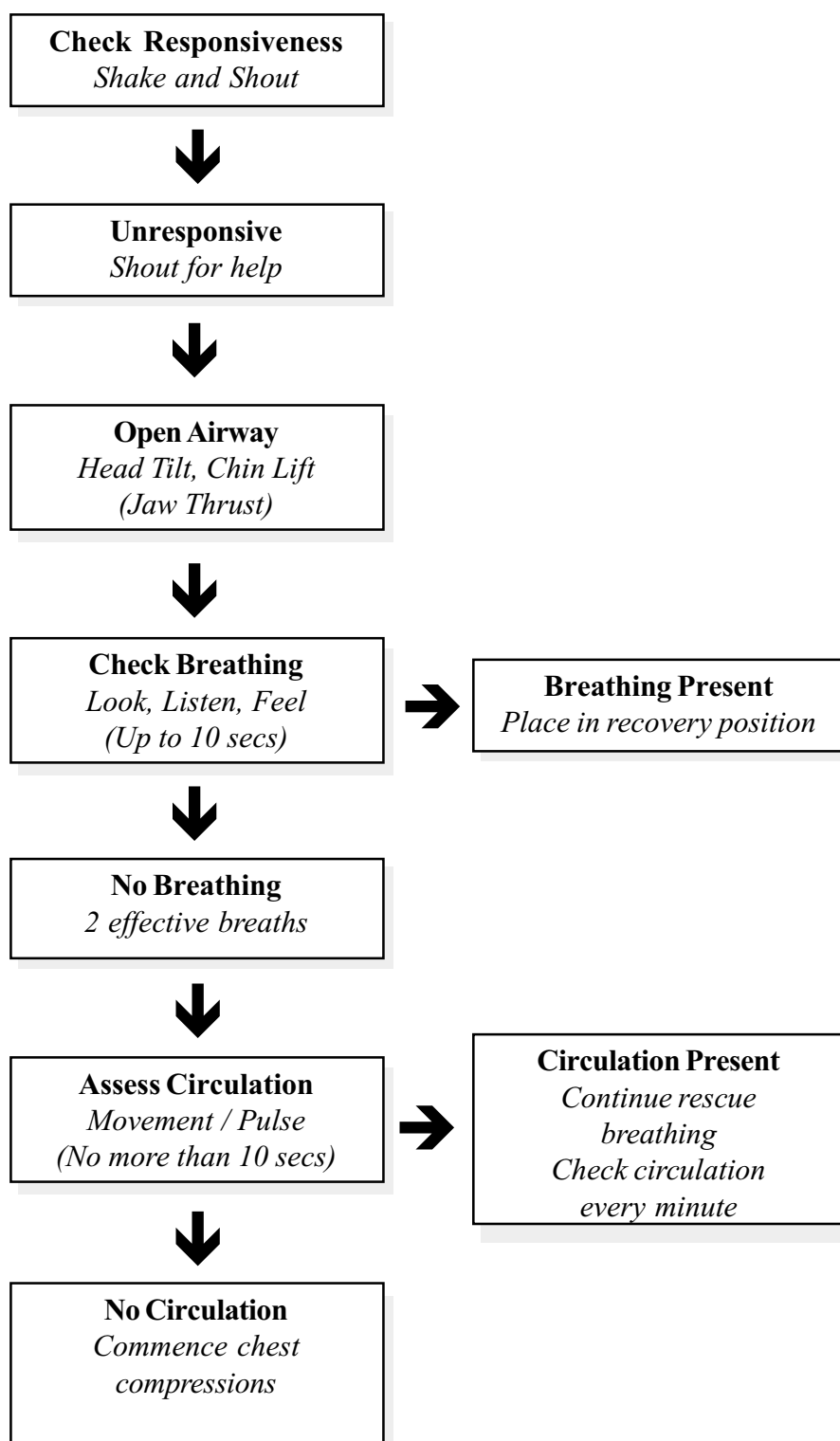
**Checking responsiveness** is best done by speaking loudly to the casualty, and trying to rouse them by shaking a shoulder. If there is no response send for help as the patient is being treated.

**Opening the airway** This can normally be done by simply extending the head and performing a chin lift. In some patients a jaw thrust will be required along with the insertion of an oropharyngeal airway. False teeth that are loose or other debris within the airway should be removed.

**Assisted ventilation** should be provided if the patient is not breathing. It may be provided using expired air ventilation (mouth to mouth, mouth to nose, using a Laerdal pocket mask) or by using a self inflating bags, usually with supplemental oxygen. Oxygen should be added to self inflating bag, using a reservoir on the inlet side of the bag. Adequacy of ventilation is judged by each breath producing adequate movement of the chest on inspiration. In general tidal volumes of 400 – 500mls are optimal.

**Chest compressions** (previously known as **cardiac massage**) are used whenever a central pulse (carotid) is absent. The technique creates positive pressure within the chest and forces blood out of the chest during the compression phase. Due to the valves within the venous system and the heart, most of the blood flows forward through the arteries. When the chest recoils to its normal position blood returns to the chest from the venous side of the circulation. A small amount of flow is produced by direct compression of the heart between the sternum and the spine. During chest compressions approximately 25% of the normal cardiac output is produced.

Current guidelines advise that 5 chest compressions are carried out for each ventilation when two rescuers are available. In the event of only one rescuer, 15 compressions

**ADULT BASIC LIFE SUPPORT**

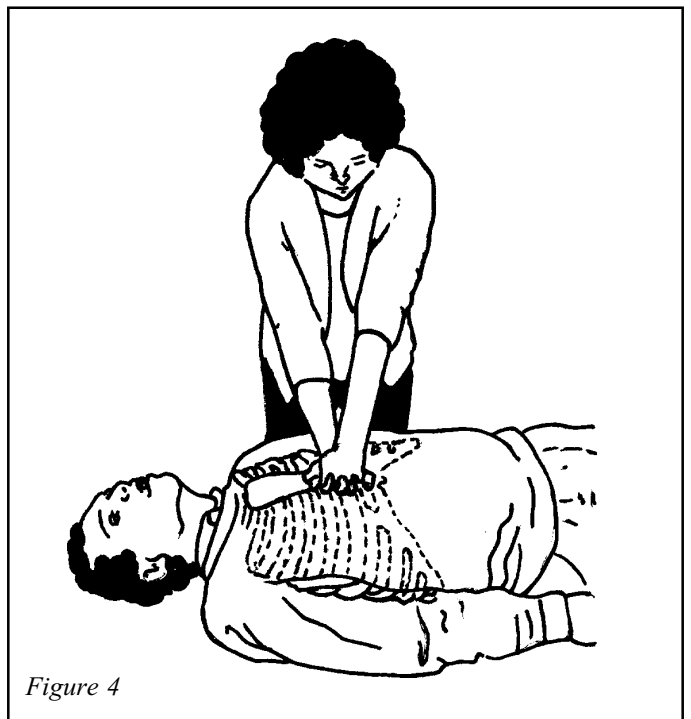
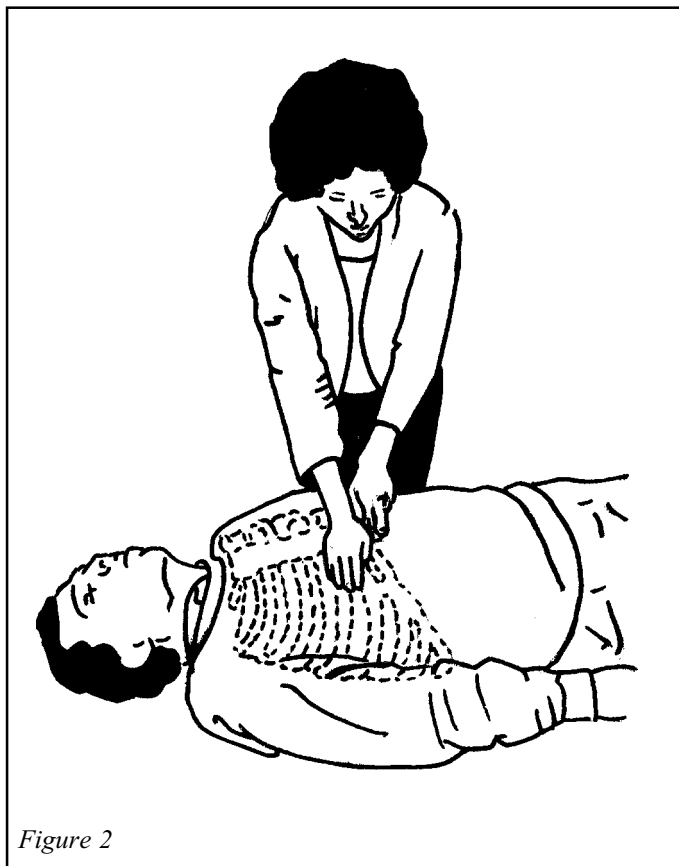
*Send or go for assistance as soon as possible according to guidelines*

Figure 1: Recommended sequence for BLS

should be followed by 2 ventilations. The overall rate of chest compressions should be 100/minute.

**When starting chest compressions:**

- Get the patient on a firm surface
- Feel the xiphisternum, and measure 2 finger breadths up on the sternum (figure 2). Without moving your fingers, place the heel of the second hand on the sternum. Put both hands together as shown in figure 3 and depress the sternum 4-5cm in an adult.
- Keep your elbows (figure 4) straight, and ensure that all the pressure is directed through the sternum and not through the ribs. To perform chest compressions adequately, it is necessary to be above the patient. Stand on a platform if necessary.
- During a cardiac arrest change the person performing chest compressions regularly, as it is tiring when performed properly.
- The rescuer performing chest compressions should count out loud "1,2,3,4,5", and the rescuer ventilating the patient should count the number of cycles completed.



Early BLS has been shown to improve outcome, particularly when access to advanced airway management and defibrillation is likely to be delayed. Although the barely adequate level of oxygen delivery achieved during BLS may be regarded as a holding measure, it is of great importance and will occasionally reverse the primary cause of the cardiac arrest and restore some circulation preventing the rhythm degenerating into asystole.



**Table 2. General Management Principles for Cardiac Arrest**

1. Establish the safety of the victim and potential rescuer.
2. Confirm the diagnosis of an arrest
3. Send for help
4. Establish Basic Life Support
5. Aim for early and frequent defibrillation if indicated, with regular doses of adrenaline and CPR.
6. If there is doubt about the rhythm, or no ECG monitor is available, treat adults as being in VF.
7. Except for defibrillation, chest compressions should not be interrupted for more than 10 seconds to allow invasive procedures or advanced airway management.
8. Administer drugs intravenously whenever possible. Use a 20-50ml 0.9% saline flush with the peripheral route.
9. Consider and treat any underlying causes
10. Consider antiarrhythmic drugs and sodium bicarbonate as described below.

### ADVANCED LIFE SUPPORT

Advanced Life Support refers to the use of specialised techniques, in an attempt to rapidly restore an effective rhythm to the heart. The most important components of the advanced life support techniques are direct current defibrillation and efficient BLS. The general principles involved with resuscitation from a cardiac arrest are shown in Table 2, and each technique involved with ALS is described below.

### SPECIALISED TECHNIQUES IN ADVANCED LIFE SUPPORT

#### Advanced Airway Management

Advanced airway management requires specialised equipment and skills and should be used in an apnoeic patient receiving basic life support.

**Oral and nasopharyngeal airways** are easy to insert with minimal experience. The commonest forms are the Guedel oropharyngeal airway and the more easily tolerated nasopharyngeal airway.

An oropharyngeal airway is sized by matching the distance between the corner of the mouth and the angle of the jaw. The nasopharyngeal airway is matched approximately to the diameter of the patient's little finger and should be well lubricated before insertion. Do not use a nasopharyngeal airway if there is any suspicion of a basal skull fracture.

**Tracheal intubation** is the best way of providing a secure and reliable airway. However, the technique requires special skills and equipment and attempts at intubation may cause further complications and delay if performed

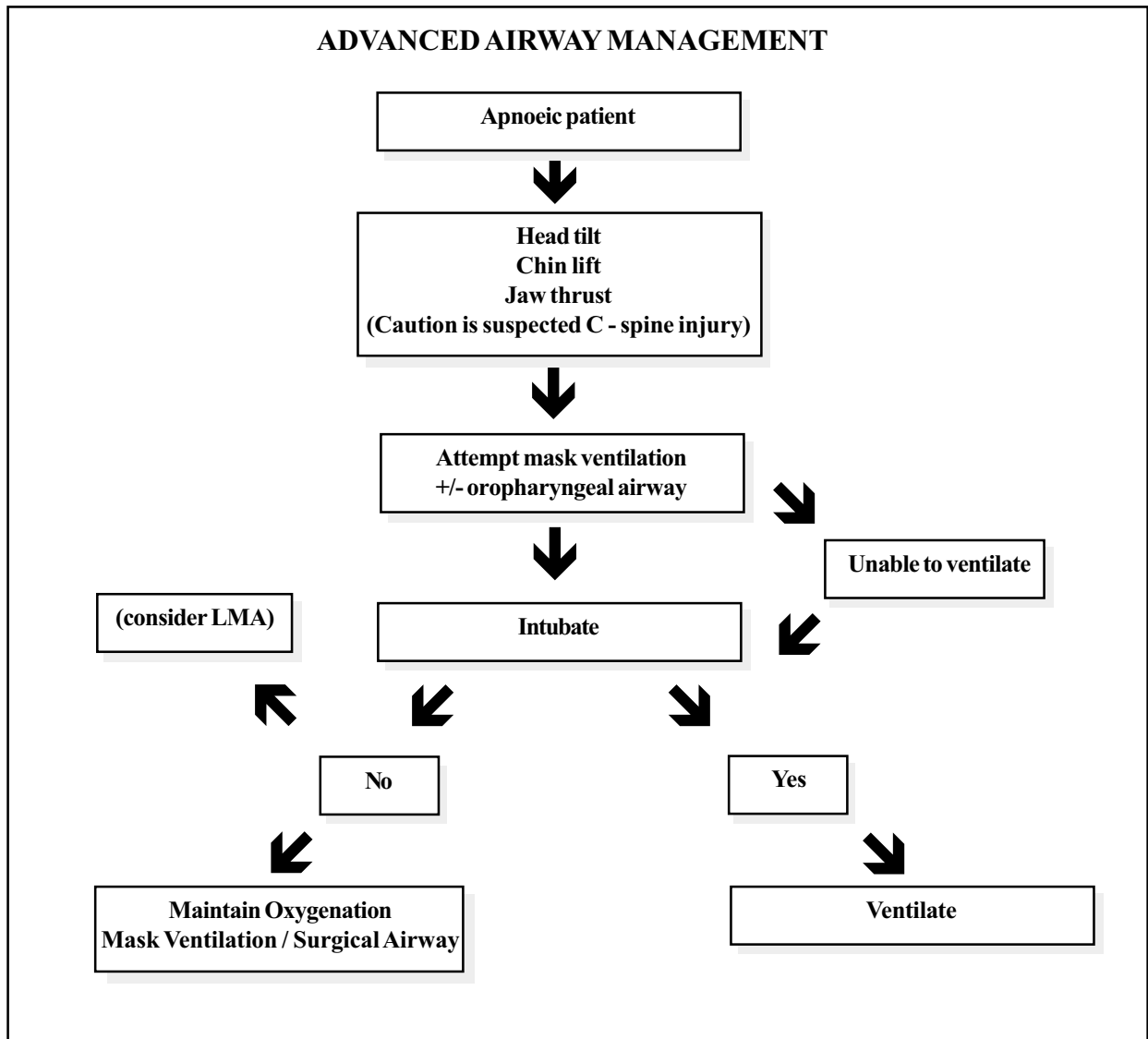
incorrectly. Confirmation of the tube's position is most reliably achieved by seeing it pass between the cords during intubation, auscultation of the chest and, if available, end-tidal carbon dioxide measurement. Various simple oesophageal detector devices are also available (see Update 1997;7:30).

Application of cricoid pressure should be considered if there is a major risk of gastric contents contaminating the airway. It should be applied until the airway is secured with a cuffed endotracheal tube. However it may make intubation more difficult for the inexperienced operator, particularly if it is not done completely correctly (see Update 1994;4:1-5).

#### Other oropharyngeal airway devices

Although it has been part of routine anaesthetic practice for around ten years in the UK, the laryngeal mask airway (LMA) has also been used for failed intubation and, more recently in resuscitation.

The insertion technique is easily taught and it provides a similarly efficiency of ventilation as a bag and mask technique. However in a few cases LMA's are difficult to position correctly, ventilation of poorly compliant lungs is uncertain, and they do not reliably protect the airway from gastric contents. The double lumen Combitube® has also been used during resuscitation. It is inserted blindly into the oesophagus and then used to inflate the lungs via the second lumen. See Update 1998;9:37-45 for more details of the LMA and the Combitube.



Flow chart for the advanced management of the airway and ventilation

**Surgical airway** techniques are required when life-threatening airway obstruction is present and other means of establishing an airway have failed. Emergency access to the airway is via the relatively avascular cricothyroid membrane. This membrane is identified by locating the midline depression between the easily identifiable cricoid cartilage and the lower edge of the thyroid cartilage.

**Cricothyroid emergency airway.** A 12 or 14 gauge cannula with a syringe attached is introduced through the cricothyroid membrane until air can be aspirated. The cannula is then advanced off the needle down the trachea. The hub of the cannula is connected to an oxygen source at 15 litres/minute and the patient ventilated for one second and then allowed to exhale for 4 seconds. In the absence of an oxygen supply, short-term improvised connections can be made by:

- The cricothyroid cannula is connected to a 10ml syringe with the plunger removed. An 8.0mm endotracheal tube is inserted into the barrel, the cuff inflated and a self-inflating bag connected and ventilation attempted.
- A 3.5mm endotracheal tube connector will usually fit directly into the cricothyroid cannula and allow connection to a self inflating bag.

Although the patient may be oxygenated in this way, ventilation to remove CO<sub>2</sub> cannot be achieved and respiratory acidosis will ensue. Spontaneous respiration is impossible through a needle cricothyrotomy and careful observation is required to prevent barotrauma. A clear expiratory pathway is required to allow the oxygen to escape as an intravenous cannula is not adequate by itself.

A needle cricothyrotomy will ensure a supply of oxygen for a maximum of 10-20 minutes and it should be converted to a surgical cricothyrotomy to allow adequate ventilation. A horizontal incision is made through the membrane and a small (size 5.0-6.5) endotracheal or tracheostomy tube is inserted and connected to a self inflating bag, providing highly efficient ventilation and airway security. Although a simple concept, the equipment may take time to assemble and there is a significant complication and failure rate. Therefore in theatre, or accident/emergency, the equipment should be prepared and ready for use.

### Blind, single-stage cricothyrotomy techniques

Several kits are commercially available (Portex, Cook Critical Care, Rusch) which are designed to pass a tube through the cricothyroid membrane in a single manoeuvre. They use either a guidewire, introducer or dilational technique and all provide a 22mm connection to standard ventilation equipment.

### Defibrillation

The majority of adult cardiac arrests involve ventricular fibrillation that may be reversed by electrical defibrillation. The likelihood of successful defibrillation decreases with the duration of cardiac arrest (an estimated 2 - 7% for every minute of the arrest) and, although BLS measures will slow the deterioration, asystole will inevitably ensue.

Defibrillation delivers an electrical current through the heart simultaneously depolarising a critical mass of the myocardium and introducing a co-ordinated absolute refractory period. This results in a period during which another action potential cannot be triggered by a stimulus of any magnitude and, if successful will stop the chaotic electrical activity of ventricular fibrillation momentarily. The

pacemaker cells of the sino-atrial (SA) node have the opportunity to re-establish sinus rhythm as they are the earliest myocardial cells to depolarise spontaneously.

All defibrillators consist of a power source, an energy selector, an AC/DC converter, a capacitor and a set of electrode paddles (Figure 5). Modern machines allow

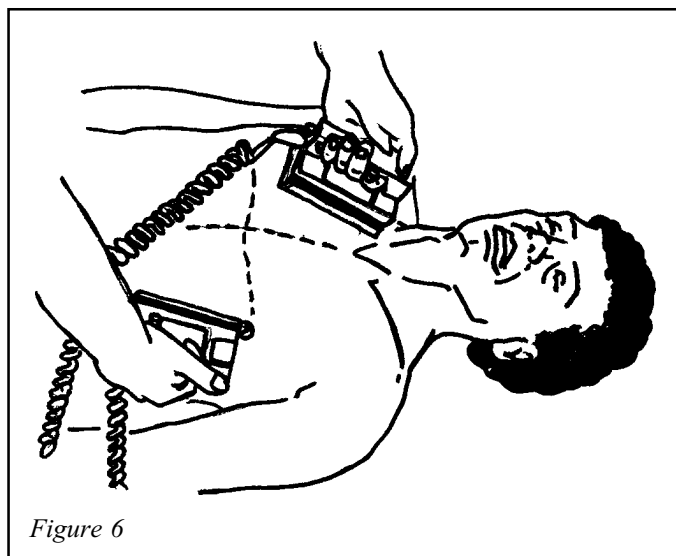


Figure 6

ECG monitoring via the paddles or via leads attached to the machine. The power output is expressed in terms of delivered energy (in Joules), which is the energy delivered through the paddles to the chest wall.

Only a relatively small proportion of the energy is delivered to the heart and variations in transthoracic impedance (resistance to current flow caused by chest tissues) will occur. The energy requirement for defibrillation (defibrillation threshold) will tend to increase with the duration of the arrest. Empirical energy levels of 200 Joules (J) for the first two shocks and 360J subsequently have been decided upon for adult resuscitation. DC shocks should be delivered with the correct paddle position and good contact using conductive pads or a coupling medium. Although the polarity of the paddles is not crucial, the cardiac complexes are upright on the screen if the paddles labelled "sternum" and "apex" are placed correctly. The sternal paddle is placed high on the right side of the anterior chest wall, lateral to the upper part of the sternum and below the clavicle; the apex paddle is placed just lateral to the position of the normal apex beat (figure 6), avoiding breast tissue in females. Other positions, such as apex-posterior may be tried if the conventional paddle position is not successful.

In recent years, semi and fully automatic defibrillators have been developed. When connected to the patient these are able to interpret cardiac rhythms and deliver shocks when



Figure 5: A defibrillator

## DEFIBRILLATION SEQUENCE

Action	Announcements
1. Switch on.	
2. Place coupling pads/gel in correct position	
3. Apply paddles	
4. Check ECG rhythm and confirm no pulse	
5. Select non-synchronised (VF) setting	
6. Charge to required energy level	<i>“Charging”</i>
7. Ensure no-one is in contact with anything touching the patient	<i>“Stand clear”</i>
8. Press paddle buttons simultaneously	<i>“Shocking now”</i>
9. Check ECG rhythm	
10. Check for output if rhythm change	<i>“Check pulse”</i>
11. Return to ALS algorithm for further steps	

appropriate. Some are also able to measure the transthoracic impedance of the patient and attempt to match the energy delivery to the required current flow. The very latest generation of machines use bi- and tri-phasic energy wave forms to achieve successful defibrillation at lower energy levels.

Regardless of the type of defibrillator available, it is essential that the staff using it are familiar with its operation, and are trained regularly in its use.

### Cardiac Arrest: Defibrillation Technique

It is assumed that the rhythm has been confirmed as suitable for defibrillation. The first three shocks of the ALS algorithm should be completed within 90 seconds. Unless the rhythm changes on the ECG trace, there is no need to check the pulse between cycles of defibrillation.

### Drug Therapy

**Adrenaline (epinephrine)** is the main drug used during resuscitation from cardiac arrest. A 1mg dose should be given at least every three minutes during the arrest. Intravenous adrenaline enhances cerebral and myocardial blood flow by increasing peripheral vascular resistance and raising aortic diastolic pressure. These peripheral vascular actions are primarily alpha1 ( $\alpha_1$ ), and alpha2 ( $\alpha_2$ ), receptor-mediated. Beta1 ( $\beta_1$ ) and Beta2 ( $\beta_2$ ) receptor actions also occur though a beta effect has not been shown to be beneficial in restoring spontaneous circulation in VF, asystole or EMD. Indeed,  $\beta_1$  effects may increase myocardial oxygen demand and increase the risk of arrhythmias in a beating heart. Recently, high dose

adrenaline (5mg) has been tried during resuscitation in an attempt to improve the survival of cardiac arrest but there was no improvement in outcome.

The ALS algorithm suggests the use of antiarrhythmics, buffers, atropine and pacing. Antiarrhythmic drugs are considered on page 30.

**Atropine** as a single dose of 3mg is sufficient to block vagal tone completely and should be used once in cases of asystole. It is also indicated for symptomatic bradycardia in a dose of 0.5mg – 1mg.

**Sodium bicarbonate** In prolonged arrests, the effects of acidosis become significant. The use of sodium bicarbonate as a buffer has been controversial; it is associated with hyperosmolarity and carbon dioxide production, and may worsen intra-cellular acidosis. Carbon dioxide-consuming buffers, such as Carbicarb and THAM have been developed, but no buffer has been shown to improve outcome. Nevertheless, sodium bicarbonate continues to be recommended (50mls of 8.4% solution) after 15 minutes of cardiac arrest or when the arterial pH is less than 7.1, or the base deficit is more negative than –10. It should be used early in arrests caused by acidosis, hyperkalaemia or tricyclic overdose, but must not be given by the tracheal route or mixed with calcium or adrenaline solutions.

### Drug Delivery

The optimal route of administration for these drugs is via a central venous cannula. However, they are usually given through a peripheral cannula and in this situation, drug

administration should be followed by a 20-50ml 0.9% saline flush and elevation of the limb to assist entry to the central circulation.

CPR should not be interrupted for more than 10 seconds to permit intravenous cannulation and consideration should be given to the tracheal route if no intravenous access exists. Although a second-line choice, endotracheal tube placement will often precede intravenous access and adrenaline, atropine and lignocaine can all be given intratracheal in doses 2 times the normal intravenous dose, diluted up to 10mls in 0.9% saline. When gaining iv access during a cardiac arrest, choose the most proximal large vein that can be easily cannulated: the external jugular vein is often suitable. ***Central venous cannulation should only be attempted by those experienced in the technique.***

The unpredictable drug delivery and risk of damage to the left anterior descending coronary artery make direct intracardiac injection impractical and unsafe.

### **Advanced Life Support Algorithm**

The algorithm (figure 7) guides the response to cardiac arrest. If the arrest is witnessed, a precordial thump should be considered. This is delivered with a heel of a clenched fist from a height of around 8 inches from the chest. This generates a few joules of electrical current within the heart which, in the early phase of a cardiac arrest, may be enough to return sinus rhythm. A precordial thump should not be administered by people who have not been trained in the technique, or if the arrest has not been witnessed. Perform a pulse check after delivering a precordial thump.

The priority in advanced life support is to determine the underlying rhythm causing the cardiac arrest and whether any underlying treatable cause can be found. The algorithm details the management according to whether the underlying rhythm falls into the category of Ventricular Fibrillation (VF)/ Pulseless Ventricular Tachycardia (VT) or, Asystole / Pulseless Electrical Activity (in the figure = Non VF /VT).

### **Ventricular Fibrillation or Pulseless**

#### **Ventricular Tachycardia**

When VF or VT are diagnosed the patient should be defibrillated as quickly as possible using three shocks of 200J, 200J then 360J. Unless the rhythm changes on the ECG trace, there is no point in checking the pulse between shocks as this will delay the next defibrillation attempt. Palpation of a major artery is carried out if the ECG appearances are compatible with an output, or if purposeful

movements are made. If these shocks are not successful, CPR should be resumed for one minute while the airway is secured and iv access is achieved. A dose of iv adrenaline (1mg) is injected and consideration is given to any specifically treatable causes of VF such as hypothermia and toxins. After another 10 cycles of CPR, the ECG trace is re-examined. Persistent VF is treated with a further three shocks of 360J as required. These take priority over any continuing attempts at securing the airway or establishing iv access. It is recommended that this sequence is followed for at least 9-12 shocks before consideration is given to the use of antiarrhythmic drugs. Adrenaline should be administered every 2–3 minutes during resuscitation.

**If there is no cardiac monitor but a defibrillator is available, it is better to treat the rhythm as VF, as this rhythm has the best prognosis.**

### **Asystole or Pulseless Electrical Activity (PEA)**

Asystole occurs when there is no detectable electrical activity in the heart and is associated with a very poor prognosis. Pulseless Electrical Activity (or Electro-Mechanical Dissociation - EMD) is present when the ECG shows a rhythm normally associated with an output but with no detectable central pulse. In either case, the defibrillation-based treatment loop is not appropriate.

In asystole or PEA treatment options are more limited. The right-hand loop of the algorithm is followed. The airway is secured and iv access obtained as soon as possible and CPR is continued with doses of adrenaline administered every three minutes. Atropine (3mg) is given once in asystole. The chance of surviving asystole or EMD is improved if a reversible cause can be identified which can be treated. The most likely ones are listed in the algorithm. Acute hypovolaemia is the most commonly treatable cause, and always results from extremely severe haemorrhage (>50% blood volume). These patients usually need immediate surgery to control haemorrhage and rapid fluid replacement. Any change of the ECG consistent with VF should prompt an immediate transfer to the other treatment loop.

### **Stopping Resuscitation**

The decision to stop resuscitation attempts is usually made by the team treating the arrest. It is usually the responsibility of the most experienced doctor present and should involve the whole team. Patients in asystole or PEA, who have no underlying cause diagnosed, and who do not respond to BLS and adrenaline, have a very poor prognosis and in our experience resuscitation attempts are normally stopped after 10–15 minutes.

### CARDIAC ARREST

**The ALS Algorithm for the management of Cardiac Arrest in Adults**  
 Note that each successive step is based on the assumption that the one before has been successful.

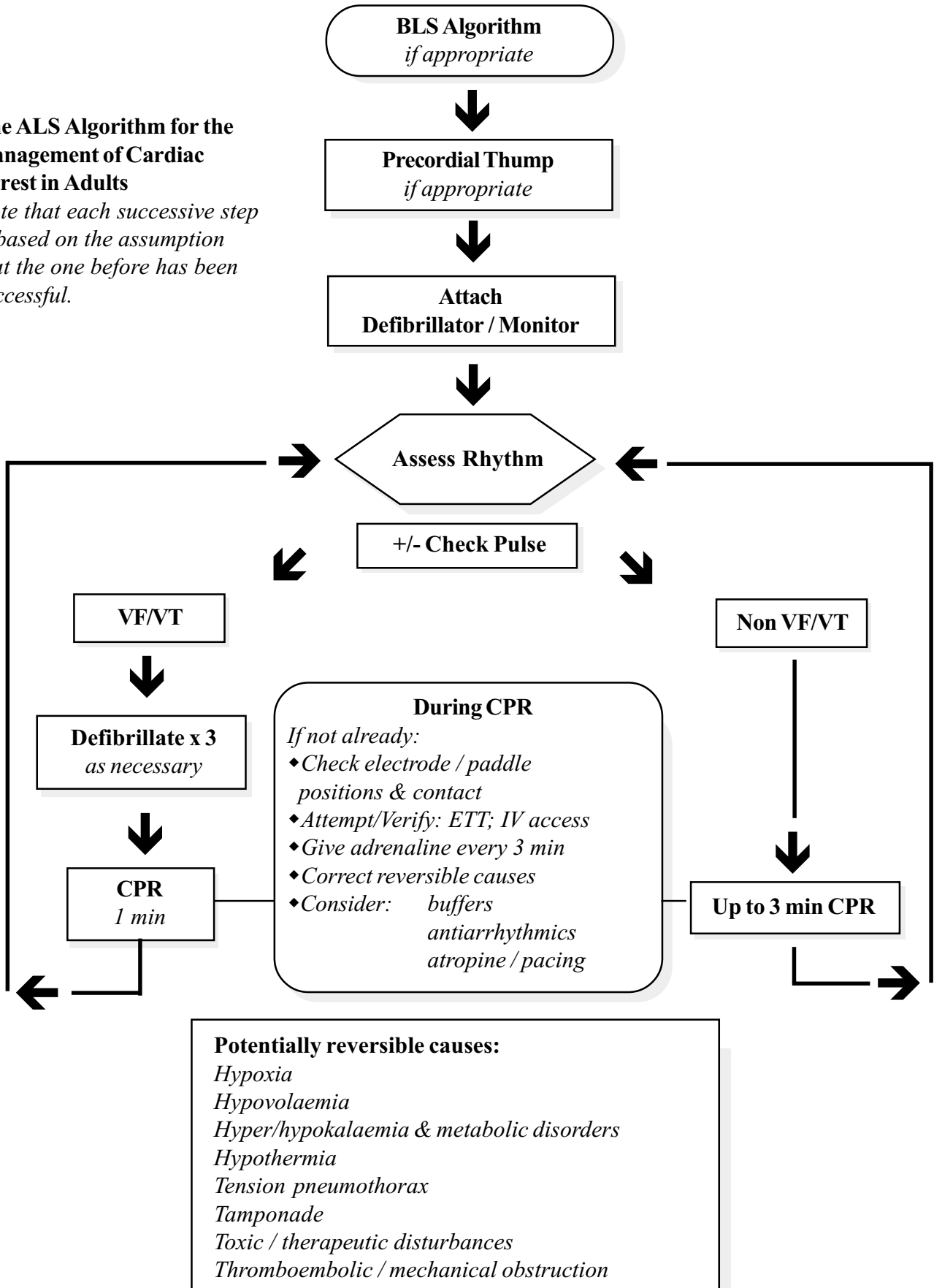


Figure 7: ALS flowchart

Treatment would normally be continued while the ECG trace indicates the presence of VF. However successful resuscitation becomes unlikely after 12 shocks, and rare after 15–20 minutes of attempted resuscitation. The highest survival rates occur in witnessed VF arrests, when BLS has been started immediately and defibrillation is very rapid. Outcome studies from VF carried out in hospitals in the developed world in the 1990's indicated an initial resuscitation success rate of up to 50% but a survival to discharge rate of up to only 20% in this population.

Patients who have severe underlying illnesses or terminal conditions usually have a cardiac arrest as a terminal event and resuscitation in these patients is usually unsuccessful, and often inappropriate. In many hospitals patients in this category may be designated "Not for Resuscitation" after discussion with the relatives and or the patient, and the medical and nursing teams caring for them. The legal position of such decisions, and the methods for making them varies from country to country.

Patients who suffer unwitnessed cardiac arrests and have delayed BLS / defibrillation as a result have a dismal outlook and resuscitation attempts will be unsuccessful in most cases.

### **Managing Cardiac Arrests without a Defibrillator.**

Clearly without the aid of a defibrillator, cardiac arrest management is more limited and the diagnosis and treatment of the likely underlying problem provides the best chance of survival. Basic life support should be initiated, adrenaline given and resuscitation attempted while any reversible factors (such as hypovolaemia) are diagnosed and treated.

### **Other Antiarrhythmic Therapy**

Although the defibrillator remains the main technique, a number of antiarrhythmic drugs may prove useful. They may be used to treat a persistent, life-threatening arrhythmia, to lower the threshold for successful defibrillation or as prophylaxis against further rhythm disturbances.

Each agent has specific indications but most are negatively inotropic - clearly undesirable in resuscitation. Lignocaine, bretylium, amiodarone and magnesium are the most commonly used agents. There is a lack of human-based evidence for their effectiveness, reflecting the difficulty in performing meaningful clinical studies in resuscitation.

**Lignocaine** (lidocaine) has antiarrhythmic properties derived from sodium channel blockade, resulting in membrane stabilisation. The pacemaker action of the SA node is suppressed and conduction within the ventricular

muscle is inhibited. There is little effect on the atrio-ventricular (AV) node and its myocardial depressant and pro-arrhythmic effects are minimal.

Lignocaine is established for the treatment of ventricular tachycardia. The ability of lignocaine to improve the chances of successful defibrillation of persistent VF is less certain, but it is often tried when repeated unsuccessful attempts at defibrillation have been made. Lignocaine is also used to treat haemodynamically stable VT.

The dose of lignocaine for refractory ventricular fibrillation is 100mg iv and for haemodynamically stable ventricular tachycardia is 1mg/kg iv – repeated once if necessary - and followed by an intravenous infusion of 4mg/min for 30 minutes, 2 mg/min for 2 hours and then 1mg/minute.

**Amiodarone** produces potassium channel blockade with some inhibition of sodium channel mediated depolarisation, a lengthening of the myocardial action potential and a degree of  $\beta$ -blockade. This gives it an antifibrillatory action and lowers the defibrillation threshold with a minimal effect on myocardial contractility.

Its routine use during cardiac arrest is yet to be proven and it is generally reserved for the second-line treatment of peri-arrest tachyarrhythmias. Amiodarone is preferably administered centrally and slowly. Usually a 300mg loading dose is given over one hour followed by an infusion of 900mg in 1000ml of 5% glucose over the following 24hrs. In more urgent situations, the first 300mg dose can be given peripherally over 5-15 minutes and followed by a further 300mg over one hour.

**Bretylium tosylate** stabilises the action potential duration throughout the myocardium. This increases the resistance to VF and lowers the defibrillation threshold. However it is slow to act (15-20minutes) and there is a tendency for it to produce pulseless electrical activity and greater post-arrest hypotension that expected.

**Magnesium** is a critical factor in myocardial cell stability. A decreased intracellular level promotes myocardial excitability but, even in the absence of a low magnesium level, a bolus of iv magnesium will suppress ventricular ectopic beats. The use of magnesium in cardiac arrest is unproven, but it may be useful when hypokalaemia may have contributed to the arrest and a dose of 10mls of 50% magnesium sulphate may be given if this is the case.

**Calcium** has a specific indication as emergency protection against the effects of hyperkalaemia or the unusual condition of calcium channel blocker (eg verapamil) overdose. Despite its crucial role in the myocardial action

potential and contraction, its administration for any other reason appears to be ineffective, or even detrimental, as high intracellular calcium concentration are damaging to injured myocardial and neuronal cells. However, if the serum potassium level is above 6mmol/L, 10ml of 10% calcium chloride should be given.

Although the individual drugs are chosen largely for their lack of effect on myocardial contractility, administration of several antiarrhythmic agents will result in a cumulative, deleterious effect even if a perfusing rhythm is restored.

If resuscitation is successful, arrhythmias remain a likely sequel. The management of subsequent brady-arrhythmias and narrow or broad complex tachycardias are beyond the scope of this article.

### Cardiac Arrests in Special Circumstances

There are a few circumstances in which the ALS principles need adapted.

**Drowning and near-drowning** Victims of immersion who are in cardiac arrest on arrival in hospital are a difficult and controversial group to treat. Occasional reports of apparently miraculous recovery after prolonged immersion and resuscitation have been made, particularly in children. These cases have involved rapid, profound cooling in **very cold water**. Children transferred rapidly to hospital who have suffered a short duration of immersion in cold water should have rectal core temperature measurement and ECG monitoring established immediately. Often asystole is present but occasionally the ECG will show slow sinus rhythm when the patient appears to be dead. If it is thought worth attempting resuscitation:

- Resuscitation should follow standard principles with BLS.
- Early intubation and ventilation with 100% oxygen should be a priority and prolonged BLS may be needed while attempts are made to rewarm the victim to 31<sup>0</sup>C as attempts to defibrillate the hypothermic heart below this temperature are unlikely to be successful
- Although rewarming the patient can be extremely difficult and sometimes impossible without facilities for cardio-pulmonary bypass, resuscitation should

not normally cease until the core temperature has reached 31<sup>0</sup>C or attempts to achieve this have failed.

- Surface warming, heated inspired gases, warm iv fluids and intra-gastric balloons are of limited value, but must be tried. Warmed peritoneal dialysis has been recommended.

The prognosis for victims of drowning discovered in cardiac arrest is very poor. Most will die or be significantly brain damaged. With few signs to indicate the speed of cooling, triage can be difficult. Hyperkalaemia is caused by the pre-hypothermic phase of the cardiac arrest and serum potassium of >10mmol/L measured during resuscitation is incompatible with survival.

### Electrocution

The effects of electrocution depend on the conversion of electrical energy into heat energy. The degree of damage depends on:

- Energy delivered
- Tissue resistance to current flow
- Type of current. Alternating current (AC) is more dangerous. It is more likely to reach central tissues and the resulting tetanic muscle contractions prevent the victim from releasing the electrical source.
- Current pathway through the body.

Asystolic arrests are more likely with currents greater than 10 Amps but VT and VF are also common. **Rescuers must take great care to avoid receiving an electric shock.**

### Drug overdose

Deliberate overdose or poisoning should always be considered in an unconscious patient. Cardiac arrhythmias or haemodynamic effects are particularly associated with certain drugs and may require specific treatment or prolonged resuscitation. Cardiac arrhythmias following a tricyclic overdose may respond to an infusion of sodium bicarbonate to maintain the pH within the high normal range and also potassium to keep the serum potassium >4.0mmol. Bupivacaine is able to bind to the myocardium and following a cardiac arrest due to toxicity from this drug, resuscitation should be prolonged (1 hour).



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## Cardiovascular Features of Common Drug Poisoning

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### Tachyarrhythmias

anticholinergics  
tricyclic antidepressants  
cardiac glycosides  
chloral hydrate  
local anaesthetics especially bupivacaine

### Bradyarrhythmias

cardiac glycosides  
organophosphates  
calcium channel blockers  
chloroquine

### Asystole

cyanide

### Hypotension

barbiturates  
chloroquine  
theophylline

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## Cardiac Arrests during Anaesthesia

The management of intra-operative cardiac arrests differs from the standard guidelines in that the event is normally witnessed and some form of airway maintenance and intravenous access has already been established. A primary cardiac event may be the cause, but treatment is often needed for an underlying problem such as vagal stimulation, blood loss, hypoxia, bronchospasm, myocardial depression, hypokalaemia, hyperkalaemia etc. The most common cause of cardiac arrests during anaesthesia are hypoxia, vagal stimulation or hypovolaemia. Most can be prevented by careful anaesthesia and close clinical monitoring. Specific treatment should be towards the underlying cause as well as initiating resuscitation procedures.

## POST-ARREST MANAGEMENT

Following the restoration of a spontaneous cardiac output, the metabolic changes and the likelihood of the injured heart developing arrhythmias make further monitoring and intensive care essential. There may also be a need to provide a period of brain protection to maximise chances

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## Special Points for Intra-Operative Arrests (to be used in conjunction with normal guidelines and treatment of the specific problem if known)

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1. Stop all anaesthetic agents, administer 100% oxygen and ventilate the lungs.
  2. Ask the surgical team to begin chest compressions at 5 chest compressions to 1 ventilation.
  3. If the patient is pregnant, create at least 10-15° of left lateral tilt to allow CPR to be effective. Deliver fetus.
  4. Commence ECG and end-tidal CO<sub>2</sub> monitoring if not already in place.
  5. Convert the airway to a tracheal tube and check its position and patency:
    - Observe chest movement.
    - Auscultate the chest and clinically exclude a pneumothorax.
    - Observe the end-tidal CO<sub>2</sub> output if available.
    - If in any doubt, change the tube.
  6. Check the oxygen supply. If in doubt, change to a cylinder or air.
  7. Check the fresh gas delivery from the ventilator to the patient. If in doubt, change to a self-inflating bag with an oxygen reservoir
  8. If possible, send blood for arterial blood gases (ABGs), electrolytes and calcium. Aim to repeat ABGs, acid base values and potassium every 10-15 minutes.
  9. If the arrest is accompanied by significant hypothermia or is due to local anaesthetic toxicity, resuscitation is likely to be prolonged.
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of recovery. To allow this, the optimum place for a recovering arrest victim is in a high dependency, coronary care or intensive care area. Even if successful resuscitation is achieved very rapidly, the heart may still be significantly damaged and is at risk of further arrhythmias.

If the patient is alert, maintaining an airway and breathing adequately, he may be extubated and admitted to a coronary care unit for monitoring and observation. Support of the cardiac output and circulation may be required with the guidance of invasive monitoring, including central venous cannulation, if available. Monitoring of end organ function, such as urine output is also required. Post cardiac arrest investigations should include serial 12-lead ECGs, CXR and basic blood tests including electrolytes, full blood count, magnesium and cardiac enzyme measurement if available. In the case of a proven myocardial infarction (ECG, enzymes) streptokinase may be considered.

If the resuscitation was prolonged, there is a significant metabolic disturbance, if the patient is cerebrally obtunded or requires a high level of inotropic support, admission to an intensive therapy unit for mechanical ventilation may be indicated, depending on the patient's prognosis and available facilities. The detailed management of post resuscitation care is beyond the scope of this article. Failure to recover consciousness 24 hours after resuscitation indicates a poor prognosis.

### Further Reading

*ABC of Resuscitation. British Medical Association UK*

*Advanced Life Support Course Provider Manual (3<sup>rd</sup> edition) Resuscitation Council UK1998.*

*European Resuscitation Council Guidelines for Resuscitation. European Resuscitation Council (1998). Ed. L. Bossaert. Elsevier.*

## PAEDIATRIC LIFE SUPPORT

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Paediatric Resuscitation is an essential cornerstone in the practice of paediatric anaesthesia. It is fundamental that all who treat infants and children are well versed in simple basic life support and that those who are required to perform the more complex skills are taught and regularly practise the advanced life support procedures.

Resuscitation of infants and children is different from adult resuscitation. Although there may be many similarities in the methodologies used in the resuscitation protocols with those used in adults, paediatric life support is governed by the fact that it begins from a different starting point. Adult sequences are based on the observation that the majority will be primarily cardiac in origin; they are therefore rapid and immediate in onset giving little or no warning of their occurrence and usually requiring a rapid defibrillation to achieve any measure of success. In infants and children the cause is usually a primary respiratory event which leads to the final cardiac event if not recognised and dealt with promptly<sup>1-5</sup>. Primary cardiac arrest in children is rare and ventricular fibrillation and ventricular tachycardia have been reported in less than 15% of the study population in the young<sup>6-8</sup>. The aetiology and pathogenesis of sudden death in this age group is therefore important. Many children have had a relatively long 'pre-arrest' phase, cardiac arrest signalling the end of a progressive physiological decline. It could be argued in such events early recognition and

aggressive therapy could prevent many deaths in this 'pre-arrest' phase but, unfortunately, some will remain irreversible despite all the best efforts of the carers.

Trauma is the one cause of cardiac arrest where children and adults overlap. Trauma is the most common cause of death in the first four decades of life. Again, it could be argued that trauma is preventable and, even more importantly, cardiac arrest secondary to trauma can in some cases be prevented by careful correct management of the airway, breathing and circulation of the trauma victims before managing the secondary injuries.

The outcome of paediatric life support is poor. Survival rates are quoted at between 3 and 17%<sup>1,2,5,8-16</sup> and can be considered even more dismal when the majority are reported as showing significant neurological impairment after arrest. Outcome is related to the aetiology of the initial event. Better outcomes have been reported when the primary event was respiratory<sup>10, 16, 17</sup> rather than cardiac in nature.<sup>10,11,18</sup>

The audit and analysis of paediatric life support events is complex. Events are relatively uncommon and many studies have to collect data over many years and still have small sample numbers. In the BRESUS study in the United Kingdom only 2% of the victims were aged under 14 years<sup>19</sup>. In an American study surveying 15 years of

prehospital events, only 7% of victims were below 30 years of age and only 3.7% were under 8 years old<sup>6</sup>. Furthermore the definitions used in evaluating events are inconsistent and often not comparable from study to study. In an effort to improve our knowledge of paediatric life support and its outcome a revised reporting template was formulated. This, the Paediatric Utstein Style Guidelines,<sup>20</sup> it is hoped, will help to standardise the reporting of outcomes of Paediatric Life Support and thereby provide an evidence based practice of comparable data sets to establish the true worth of these events.

One conclusion that is clear is that infants and children who progress to cardiac arrest have a very poor prognosis. Because of the aetiology of resuscitation in these age groups it is important that the pre-event symptoms are recognised early and treated effectively before there is respiratory collapse and the inevitable cardiac arrest. Therefore in paediatric life support prevention and recognition of the impending event is a major factor in the overall survival.

### **Paediatric Life Support**

Guidelines for paediatric life support have been published by a number of national organisations<sup>21-24</sup>. In 1992 an International Liaison Group was established to examine the basic scientific data, analyse the national differences and to make recommendations formulated on science which would form the basis of international guidelines to be used in the future by individual national organisations. In 1997 the International Liaison Committee on Resuscitation (ILCOR), a multinational committee comprised of members representing most of the major national resuscitation organisations published a series of advisory statements including a paediatric statement<sup>25</sup>.

In 1998 the European Resuscitation Council published its revised recommendations for resuscitation of infants and children, and for the resuscitation of babies at birth<sup>26-28</sup>.

### **Age definitions**

Paediatric life support deals with the resuscitation of infants and children. Because of the wide variation in anatomy, physiology and epidemiology throughout the paediatric age band it is therefore important to define various age ranges in an effort to rationalise treatment.

### **Anatomy**

The size of a child is an obvious important consideration in determining the practical resuscitation protocol to be followed. Age will determine the finer details of the procedures to be performed especially in basic life support.

*An infant is a child under the age of one year.*

*A child is aged between 1 and 8 years of age.*

*Children over the age of 8 years should still be treated as younger children but may require different techniques to attain adequate chest compressions.*

The upper age limit of 8 years for children has been proposed as a watershed particularly in relation to the technique of chest compression. A small child under the age of eight will probably receive adequate chest compressions using a 'one-handed' technique. An older or larger child will probably require a 'two-handed' (adult) technique to achieve an adequate depth of compression. Nonetheless, because of the variability of size in children no definitive upper age limit can be stipulated and the rescuer must judge the effectiveness of the resuscitation and adapt his technique appropriately.

Adult resuscitation protocols have also been modified by the European Resuscitation Council to dovetail with the above definitions<sup>29,30</sup>. They require the rescuer to determine the cause of the arrest and where this is not primarily cardiac in origin, for example trauma or drowning, to use a protocol that more closely aligns to the paediatric recommendations.

### **Basic Life Support**<sup>26</sup>-Figure 1.

#### **Assess responsiveness**

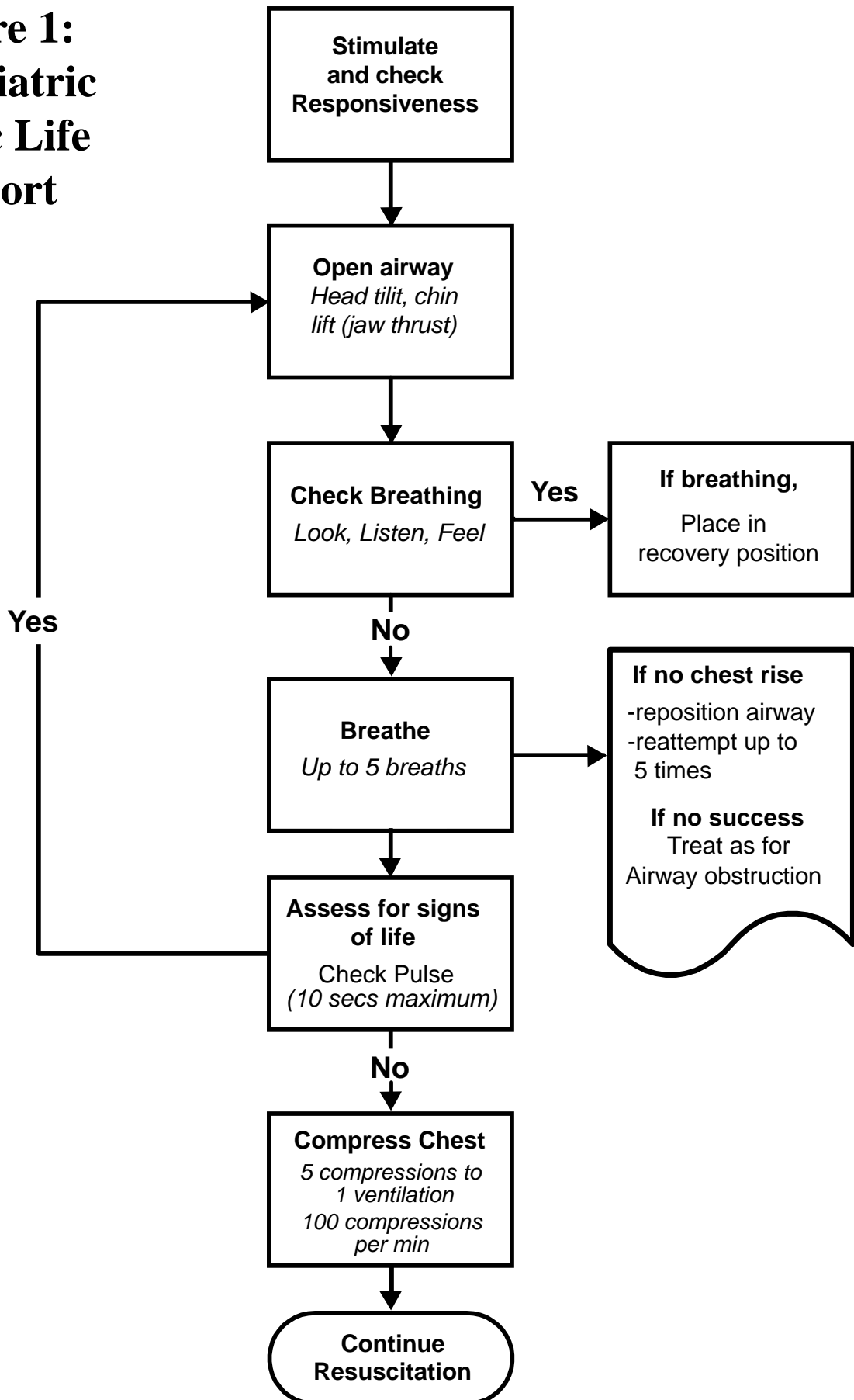
Before starting any resuscitation procedure it is vital to evaluate the situation for any danger or physical hazards. The first step in the sequence is to assess the level of responsiveness. Gentle but firm stimulation must be given and this alone may be sufficient to awaken the child or to stimulate respiration. Infants should not be vigorously shaken nor should children with a suspected spinal injury. If the child is unresponsive shout for help. Only move the child if he is in a dangerous location.

#### **Airway**

The tongue is the most common cause of airway obstruction in children. Simple head tilt - chin lift or jaw thrust manoeuvres will establish the paediatric airway, the jaw thrust procedure is particularly recommended when trauma is the cause of the collapse. A recent study has shown that there is no compromise of the airway by hyperextension of the neck<sup>32</sup>. None the less during trauma the cervical spine should be meticulously guarded by in-line cervical spine immobilisation.

If a foreign body is obstructing the airway then it should be removed carefully under direct vision. Blind probing

**Figure 1:  
Paediatric  
Basic Life  
Support**



of the child's airway is only likely to impact the object further or to directly cause tissue damage and must be discouraged. When complete upper airway obstruction has occurred as a result of inhalation of a foreign body, the object will probably be too far into the airway to either be seen or removed by simple means and will need advanced airway procedures.

### Breathing

Assessing effective breathing is very difficult<sup>33</sup> and is subject to errors. Three methods are recommended in assessing respiration:

1. *Look for chest and abdominal movement.*  
This detects physical movement, but it may not be co-ordinated and effective in moving air in and out of the lungs.
2. *Feel at mouth and nose for air movement.*  
This answers the question as to the effectiveness of the chest movement.
3. *Listen over the airway for breath sounds.*  
An important manoeuvre which will indicate whether the child has large or small airway problems. An absence or lessening of airway noise could mean that the problem is improving; alternatively it could indicate that the child is moving less air past the obstruction and that the situation is getting worse.

If the infant or child is not breathing then it is essential to commence expired air resuscitation immediately. Using the mouth of the rescuer applied to the mouth and nose of the infant has been the conventional teaching but recently the effectiveness of mouth of the rescuer applied to the nose of the infant been described<sup>34,35</sup>. In the child, mouth to mouth expired air ventilation is recommended. Because of the probable hypoxic aetiology of the event, **five** expired air ventilations are considered the optimal number of breaths to oxygenate an infant or a child. The breaths should be slow, each lasting 1 to 1.5 seconds. These slow breaths minimise gastric distension from high pressure, high flow ventilation<sup>36,37</sup>. It is also important not to ventilate with excessive tidal volumes as this may lead to gastric distension and regurgitation of gastric contents<sup>38</sup>. A simple and effective guideline is to observe the child's chest and to stop ventilation when the child's chest looks as if he had taken a deep breath. If the chest does not move when attempting ventilation then reposition the airway or consider clearing the airway using the procedure described below for a choking child.

### Circulation

Assessment of the circulation at this point in the resuscitation sequence has conventionally been by checking the pulse. Assessment of the brachial pulse is recommended<sup>39</sup> in infants and assessment of the carotid pulse in children.

The pulse check should take no longer than ten seconds and if a pulse is not felt, or the pulse rate is below 60 beats per minute in an infant, then resuscitation should continue immediately with chest compressions. Despite the apparent simplicity of a pulse check, studies have shown that both the lay rescuer and the experienced health care professional have difficulty in making an accurate pulse check<sup>40-43</sup>. The inaccuracy of the procedure has led to the validity of a pulse check in paediatric life support being challenged<sup>44</sup>. The concept of not performing a pulse check at all before commencing chest compressions is considered difficult to accept by some as it may appear to be illogical not to formally establish cardiac arrest before commencing chest compressions. Therefore the guidelines now include the statement that starting chest compressions should be considered without delaying for a pulse check in an unresponsive child who does not show obvious signs of recovery after expired air ventilation.

### Chest Compressions

Chest compressions (previously known as cardiac massage) are performed on the lower half of the sternum.<sup>45</sup> In the infant compression is performed using two fingers placed one fingers breadth below an imaginary line joining the nipples. In the child the heel of one hand is used and positioned one finger's breadth up from the xiphisternum. In the older child (over the age of 8) and in the larger young child, this one handed compression technique may be found to be inadequate and the two handed compression technique (as used in adult resuscitation) may be required to produce effective chest compression.

The depth of compression should be judged in relative rather than absolute terms. For infants and small children it is recommended to compress the chest to one third of its resting depth. The efficacy of chest compression can be judged by palpation of the femoral vessels but this may reflect venous and not arterial blood pulsation. More effective assessment can be made by analysis of the arterial waveform or evaluation of the expired carbon dioxide tracing.

The compression rate is 100 compressions per minute. A single expired air ventilation should be given after every

five compressions. This provides adequate ventilation and oxygenation for the infant or child. In the older child, where two hands are required for effective chest compression, the adult ratio of 15 compressions to 2 ventilations can be used, compressing the chest at a rate of 100/min.

### Activation of the Emergency Medical Services

Ideally the call for help given during the assessment of responsiveness should have activated the emergency medical services. In reality this is not always the case, and the priority in paediatric life support is to establish an airway, to commence effective breathing and to circulate the oxygenated blood. In paediatric life support therefore resuscitation is started and the Emergency Medical Services activated after approximately one minute of resuscitation. Thus the paediatric protocols have adopted the 'phone fast' rather than the 'phone first' philosophy based on the aetiological consideration of resuscitation event. This is considered a general recommendation but local emergency medical services circumstances or the availability of 'dispatcher-guided CPR' may override these recommendations.

**Basic life support must continue without further interruption until experienced help arrives or until signs of life return.**

### Foreign Body Airway Obstruction

Airway obstruction due to aspiration of food or vomit or the inhalation of a foreign body will compromise the paediatric airway. Spontaneous coughing to clear the material should be encouraged but if this fails back blows and chest thrusts in infants and back blows together with alternate cycles of chest thrusts and abdominal thrusts in children may provide vibration to loosen the material and enough expiratory force to expel the obstruction. Abdominal thrusts are not recommended in infants under the age of one year as damage to the abdominal contents may occur. The importance of checking the mouth, formally opening the airway and attempting expired air ventilation after each cycle has been highlighted by the need to ensure that the airway is actually obstructed. These checks are also required to assess whether the clearing manoeuvres have dislodged the material enough to allow some air pass the obstruction. The precise sequence for the relief of airway obstruction has not been formally assessed.

### Advanced Life Support<sup>27</sup> - Figure 2

The Basic Life Support sequence provides the fundamental primary treatment of an infant or child that collapses in cardiopulmonary arrest. Advanced life support is the

definitive management of the condition using complex techniques, drugs and equipment. As in basic life support, advanced life support protocols emphasise the importance of establishing an airway, oxygenation and ventilation from the outset. Although it includes a pathway for the management of ventricular fibrillation (VF) and ventricular tachycardia (VT) the emphasis is on non-ventricular fibrillation and non-ventricular tachycardia (Asystole and Pulseless Electrical Activity – previously known as Electro-Mechanical Dissociation) as these are the rhythms found in the majority of paediatric events. Ventricular fibrillation has been documented in less than 10% of paediatric events<sup>6,9,10,46-48</sup>.

### Airway

The simple basic procedures of head-tilt, chin lift or jaw thrust remain the mainstay of airway management. The insertion of a Guedel airway, correctly sized from the centre of the mouth to the angle of jaw, may be of use to aid simple airway control in the short term. Alternatively a nasal airway can be inserted.

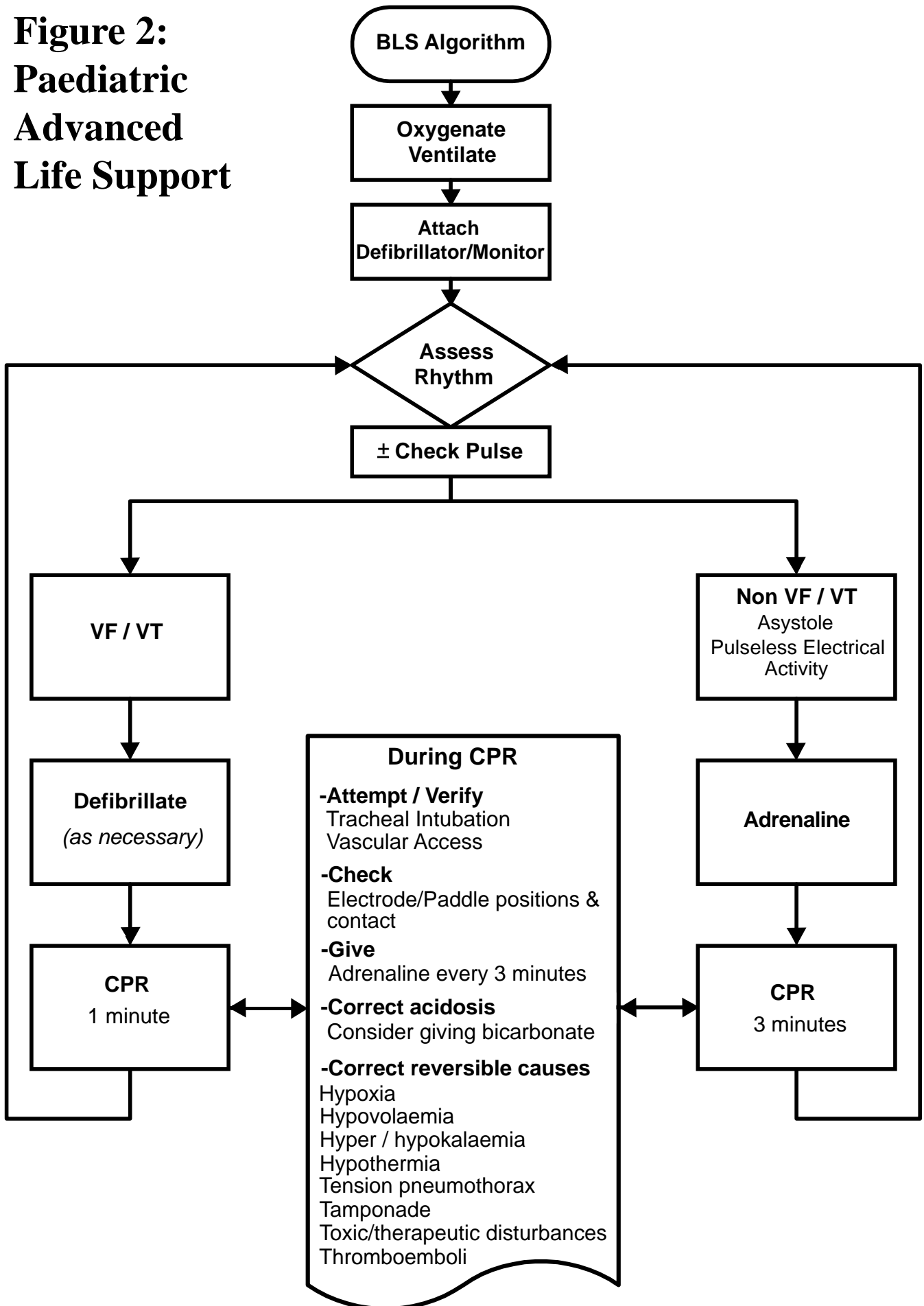
The laryngeal mask airway has been assessed as an effective airway adjunct in adult resuscitation and is a technique that can easily be taught to doctors, nurses and paramedic staff<sup>49-54</sup>. Small sized laryngeal masks are available for infants and children but their effectiveness in paediatric resuscitation has yet to be established. They will probably have their most significant effect where intubation is difficult or where the health care provider is not proficient in paediatric tracheal intubation skills.

Tracheal intubation is the most effective method of securing the paediatric airway. Using a straight bladed laryngoscope and a plain plastic tracheal tube of the appropriate size (*internal diameter (mm) = (age in years / 4) + 4*) is a technique which requires a skill only developed by formal training and regular practice. Intubation must be achieved quickly and accurately without a prolonged delay to basic life support. Any attempt lasting longer than 30 seconds should be abandoned and the child reoxygenated before a further attempt at intubation is made. Having achieved tracheal intubation the tracheal tube needs to be carefully fixed in place to prevent its accidental removal or displacement.

### Oxygenation

Although expired air resuscitation will provide some oxygenation the sooner ventilation with high-inspired oxygen levels can be established the better. Ventilation using a self-inflating bag-valve-mask with supplemented oxygen will provide higher levels of inspired oxygen.

**Figure 2:  
Paediatric  
Advanced  
Life Support**



Concentrations up to 90% can be achieved if the self-inflating bag is fitted with an oxygen reservoir system. Face masks for use with a self inflating bag should be of clear plastic so that the airway can be observed through the mask and the circular design mask with a soft seal rim have been found to be most efficient, especially in the hands of an inexperienced operator. Although many anaesthetists are experienced in the use of the Ayre's T-piece with the Jackson-Reece modification for paediatric ventilation, this circuit is not recommended for the less experienced and should not be part of the routine resuscitation equipment. Furthermore this system requires a constant flow of oxygen which may not always be immediately available. The self-inflating resuscitation bag can function independently and has the advantage of being capable of being operated safely and effectively by a much wider range of operators.

### Circulation

There are few procedures more fraught with difficulty in resuscitation than establishing venous access in an infant or small child during resuscitation. Yet circulatory access is of prime importance to effective advanced life support<sup>55,56</sup>. The intravenous or intraosseous routes of drug delivery are the preferred options. The site of venous access has to be balanced against the resuscitation skills and relative difficulty and risks of the technique. Experimental data has demonstrated that vascular access via the superior vena cava by either peripheral or central routes is preferable during resuscitation<sup>57-59</sup>. Drugs given via the inferior vena cava take longer to reach the heart<sup>58,59</sup>. Similarly drugs administered centrally do act more rapidly than those administered via the peripheral route<sup>58,60-62</sup>. Central access above the diaphragm is difficult and fraught with potential problems. Peripheral access, especially via veins in the lower limbs is usually easier especially during resuscitation. Drugs administered via the peripheral route should be followed by a fluid flush to move more rapidly into the actual circulation. Therefore when judging the advantages of the different access points and deciding which to select it must be remembered that achieving access accurately safely and rapidly is the first priority.

Intraosseous access has gained popularity in the last few years – see Update in Anaesthesia 1995;5:15-17. It is relatively easy and generally safe. Resuscitation drugs and fluids administered by this route reach the heart in a time comparable to direct peripheral venous access<sup>64-67</sup>. Although originally recommended for children under the age of six the intraosseous route has been used in older age groups and in adults during cardiac resuscitation<sup>67,68</sup>.

When establishing intraosseous access it is important to recognise the criteria for successful entry into the bone marrow. There should be a loss of resistance as the marrow cavity is entered, the needle should remain upright without support, bone marrow can be aspirated with a syringe and there is free flow of drugs and fluid without subcutaneous infiltration around the entry point<sup>69,70</sup>. Marrow aspirates can be used for estimation of haemoglobin, sodium, potassium, chloride and glucose<sup>71</sup>. Complications of intraosseous access include osteomyelitis, long bone fractures<sup>72</sup>, subcutaneous drug extravasation and compartment syndrome<sup>75-77</sup>.

The tracheal route of administration of drugs comes third to the intravenous and the intraosseous routes. It is best regarded as a route to be used where there has been, or is likely to be, a significant delay in establishing venous access and thus the administration of drugs. Therefore during resuscitation of the small infant or child it could be argued that the first important dose of adrenaline should be given by the tracheal route whilst venous access is being established<sup>78,79</sup>. There has been little research as to the efficacy of drugs administered via the tracheal route in children. The optimal dose of drug, its volume and its concentration has yet to be formally established<sup>80-83</sup>. Tracheal administration, despite its apparent simplicity, does have some disadvantages especially in the post resuscitation period. Hypertension and tachycardia, neither of which are optimal in the post arrest myocardium, have been reported and attributed to the depot storage effect of adrenaline that occurs in the lungs<sup>84-88</sup>. Severe hypertension may also be an underlying cause for a poor cerebral outcome<sup>89</sup>.

Although direct intracardiac injection is still occasionally practiced, less than 70% of injections enter the heart and serious cardiac damage may occur. It is not recommended.

### Drugs

Although many drugs have been tried in paediatric life support, few have retained their place in the resuscitation treatment protocols.

#### Adrenaline (Epinephrine)

Adrenaline is the mainstay of paediatric life support. It is used mainly for its alpha-adrenergic activity causing peripheral vasoconstriction, raising the peripheral vascular resistance, increasing the end diastolic filling pressure and thereby improving coronary blood flow<sup>13,90</sup>. Adrenaline's beta-adrenergic activity is also useful as it has a direct inotropic and chronotropic effect on the myocardium.



The recommended initial dose of adrenaline is  $10\text{mcg.kg}^{-1}$  when administered via the intravenous or intraosseous routes.  $10\text{mcg.kg}^{-1}$  is  $0.01\text{mg.kg}^{-1}$  or  $0.1\text{ml.kg}^{-1}$  of a 1 in 10,000 solution. Recent studies in infants and children have suggested the benefit of a higher dose of adrenaline for the unresponsive asystolic child<sup>91</sup>. Therefore, should the child not respond to the initial dose of adrenaline then a second dose of  $100\text{mcg.kg}^{-1}$  ( $0.1\text{ml.kg}^{-1}$  of a 1 in 1000 solution) is recommended. If the child does not respond to this or additional  $100\text{mcg.kg}^{-1}$  doses of adrenaline then the eventual outcome is likely to be poor; the results of studies show that no children have survived to discharge who have received more than two doses of adrenaline<sup>5,9,15</sup>.

### Atropine

Atropine is a parasympathetic blocking drug that will block the cardiac activity of the vagus nerve. It is used to treat bradycardia in a dose of  $20\text{mcg.kg}^{-1}$ . Atropine should be considered in the peri-arrest scenario especially as it will prevent bradycardias of vagal origin (for example a vagal bradycardia during eye surgery) before they progress to cardiopulmonary collapse. Atropine is not recommended during resuscitation from cardiac arrest as the adrenergic effects of adrenaline are considered to over-ride the parasympathetic bradycardic effects on the heart.

### Bicarbonate

Sodium bicarbonate is an alkalyzing agent used to correct the acidosis often associated with resuscitation. However, sodium bicarbonate is a solution with a high osmolarity containing a high level of sodium. The recommended dose is  $1\text{mmol.kg}^{-1}$  ( $1\text{ml.kg}^{-1}$  of an 8.4% solution). Sodium bicarbonate should only be given if the child is being effectively ventilated as any carbon dioxide that is released by the process of acid neutralisation must be removed from the body via the lungs or paradoxical intracellular acidosis will result.

### Treatment Algorithms

The administration of adrenaline plays a pivotal role in the advanced life support algorithms of paediatric life support. Establishing venous access and ventilating with oxygen are the first steps in advanced life support and form the basis for the advanced treatment protocol. The algorithm then divides into two pathways according to the presenting cardiac rhythm - Non Ventricular Fibrillation (or

Tachycardia) or Ventricular Fibrillation (or Tachycardia).

### Non Ventricular Fibrillation or Tachycardia (Asystole or Pulseless Electrical Activity)

A profound bradycardia or asystole is the most common rhythm associated with cardiac arrest in infants and children. The profound bradycardia (usually described as being a pulse beat at less than one beat per second) may precede asystole but in itself the bradycardia does not produce an adequate cardiac output. A profound bradycardia should therefore be treated in the same way as an asystole. The treatment is an initial dose of adrenaline at  $10\text{mcg.kg}^{-1}$  given by the intravenous or intraosseous route (or ten times this dose via the tracheal tube if venous access has not been established). Second and subsequent doses of adrenaline should be at  $100\text{mcg.kg}^{-1}$ .

Where there is a cardiac rhythm but no cardiac output (Pulseless Electrical Activity) it is also necessary to treat any of the underlying reversible causes of cardiac arrest. These are the 4‘H’s and 4‘T’s of cardiac arrest.

Hypoxia	Tension Pneumothorax
Hypovolaemia	Tamponade
Hyper/hypokalaemia	Toxic/Therapeutic disturbances
Hypothermia	Thromboemboli

Adrenaline should be administered every three minutes according to the schedule described previously and resuscitation should not be abandoned until a reasonable attempt has been made to correct these potentially reversible causes of cardiac arrest.

### Ventricular Fibrillation and Tachycardia

These rhythms, though common in adults, are relatively rare in infants and children. Although one study<sup>11</sup> reported an incidence of 23% ventricular fibrillation in children, other studies report an incidence of between 0 and 10%<sup>86,92,93</sup>. Therefore the physician must always be aware of the occasional need to treat ventricular fibrillation in children by defibrillation.

The recommended sequence is to give two rapid defibrillatory shocks of  $2\text{joules.kg}^{-1}$ , followed by a single shock at  $4\text{joules.kg}^{-1}$ . All further defibrillation attempts should then be made at  $4\text{joules.kg}^{-1}$  in a rapid repeated series of 3 shocks. Following the first cycle of three defibrillation attempts adrenaline  $10\text{mcg.kg}^{-1}$  should be given and, in accordance with previous explanations, a further dose of  $100\text{mcg.kg}^{-1}$  should be given following the second cycle of three shocks and between all

**Editors note:  $\text{mcg.kg}^{-1}$  is equivalent to  $\text{mcg/kg}$  and  $\text{ml.kg}^{-1}$  is the same as  $\text{ml/kg}$**

subsequent cycles. When ventricular fibrillation occurs in children there is often an underlying cause and the correction of hypothermia, drug overdose (tricyclic antidepressant overdose) and electrolyte imbalance (hyperkalaemia) should be considered.

### **Resuscitation of the Newborn<sup>28</sup>**

Newborn resuscitation specifically refers to the resuscitation procedures at or immediately after the delivery of a newly born infant. There is a specific sequence of events centred on the respiratory and circulatory changes that occur in relation to the 'First Breath'. Therefore the recommended resuscitation procedures (Figure 3) emphasise the airway and breathing manoeuvres whilst the management of the circulation is left to the trained health care provider. Resuscitation of the newborn is unique in that it is, in most cases, predictable. It is only rarely an unexpected emergency procedure. Careful assessment of maternal and foetal factors, the mode of delivery and the obstetric care will predict the majority of newborns that will require resuscitation procedures.

It has been estimated that the use of simple airway measures could prevent newborn asphyxia occurring in 900,000 infants per year world-wide. Of the five million newborn deaths per year world-wide, 56% of which occur in 'out of hospital births', 19% have 'birth asphyxia' as a cause.<sup>94</sup> In the United Kingdom newborn mortality is much lower but with the increase in 'home deliveries' it has become increasingly important for the birth attendants and other health care professionals to be not only conversant with obstetric problems but also proficient in newborn resuscitation techniques.

The majority of newborn infants cry within a few minutes of birth and require little more than careful drying and then wrapping in a warm towel to prevent heat loss. If the baby does not cry, it should be gently stimulated by more vigorous drying with a towel or flicking the soles of the feet. More vigorous stimulation is contraindicated and can be potentially dangerous. Of those that do not cry most will only need clearing the airway and ventilation, very few will need full resuscitation including intubation, circulatory access and drug administration.

The newborn baby's initial cry and subsequent efforts at breathing must be carefully assessed to ensure that they result in adequate and sustained oxygenation of the lungs. Gasping without additional efforts at breathing are usually considered inadequate. Abnormal or absent ventilatory

patterns will require immediate active intervention.

The initial assessment of the neonate is based on respiratory activity, colour and heart rate. These three parameters have been shown to be more accurate in the assessment of the newborn than the total Apgar scoring system<sup>95,96</sup>.

The newborn can be classified into three groups.

1. **Fit and healthy baby,  
Vigorous effective respiratory efforts  
Centrally pink  
Heart rate > 100/minute.**

This baby requires no intervention other than drying, wrapping in a warm towel and, where appropriate, handing to the mother. The baby will usually remain warm by skin to skin contact with mother and may be put to the breast at this stage.

2. **Breathing inadequately or apnoeic  
Central cyanosis  
Heart rate > 100/minute**

This group of babies may respond to tactile stimulation and/or facial oxygen but often need basic life support.

3. **Breathing inadequately or apnoeic  
Pale or white due to poor cardiac output and peripheral vasoconstriction  
Heart rate <100/minutes or No detectable heart rate (although this was documented up to 15 - 20 minutes before delivery.)**

These babies sometimes improve with initial basic life support but normally require immediate intubation and positive pressure ventilation progressing to chest compressions, and full advanced life support including resuscitation drugs if the baby fails to respond.

### **Newborn Basic Life Support (Figure 3)**

#### **Airway**

Open the airway by tilting the head into the neutral position and lifting the jaw upward by gentle pressure on the mandible. The airway can be cleared of residual debris and fluid by gentle suction of the mouth and nares. Aggressive pharyngeal suction can delay the onset of spontaneous breathing and cause laryngeal spasm and vagal bradycardia<sup>97</sup>. It is not indicated unless the amniotic fluid is stained with thick meconium or blood. If suction is required, a 10FG (or if preterm, 8FG) suction catheter should be connected to a suction source not exceeding -100mmHg. This should not continue for longer than 5 seconds in the absence of meconium. The catheter should normally not be inserted further than about 5cm from the lips.

## Breathing

Check for breathing by look, listen and feeling for respiratory effects. The inspired air can be supplemented with oxygen from a loose fitting facemask or funnel. Effective ventilation can only be carried out by using a well-fitting facemask that covers the mouth and nose but does not cover the eyes or overlap the chin<sup>98</sup>.

Self-inflating resuscitation bags refill independently of adjuvant gas flow. They should incorporate a pressure limited pop-off valve pre-set at 20 - 30 cm H<sub>2</sub>O. In a minority, this pressure may be inadequate to achieve lung expansion at birth and the facility to override this is useful for a few babies. The volume of the bag should be at least 500ml, so that the inflation pressure can be maintained for at least 0.5 seconds. Facemask T-piece resuscitation uses compressed air/oxygen fed to one arm of a T-piece attached to the facemask<sup>99</sup>. The baby's lungs are inflated by occluding the open arm of the T-piece. It is obviously essential to have a safety pressure release system (set at 20 - 30 cm H<sub>2</sub>O) incorporated in the gas supply tubing. A method for monitoring the peak pressures will also be required. This system has the advantage that it requires only one hand for normal operation and the inflation pressures can be maintained for longer than with the self-inflating bags. It has been traditional to use 100% oxygen as the ventilating gas for resuscitation but there is data indicating that, in term babies, 100% inspired oxygen has little advantage and may increase oxygen free radical damage. Furthermore there is evidence that newborn resuscitation is as effective with air as with 100% inspired oxygen.<sup>100-103</sup> If gas-mixing facilities are available then a 40% inspired oxygen is recommended as the ventilating gas to expand the newborn lungs, but if cyanosis persists or the heart rate falls the inspired oxygen level should be raised.

The first five or six breaths require an inspiration held for 1 to 2 seconds. This prolonged inspiration will double the inspiratory volume and is more likely to establish the functional residual capacity needed by the baby to continue to breath spontaneously<sup>104</sup>. After these initial breaths a normal ventilatory pattern can be used, ventilating at a rate of approximately 30-40 breaths per minute until spontaneous respiration is established.

If the baby does not respond to these initial face mask resuscitation manoeuvres or the heart rate falls below 100 beats per minute, the health care professional must proceed to tracheal intubation and advanced life support procedures immediately.

## Newborn Advanced Life Support (Figure 4)

Tracheal intubation is a skilled technique that requires training and practice. It is achieved, using a straight blade laryngoscope and an appropriate size of tracheal tube

### Guideline for tracheal tube size

Tracheal Tube Size (mm. Internal Diameter)	Weight (g)	Gestation (weeks)
2.5	<1000	<28
3	1000 - 2500	28 - 36
3.5	>2500	>36

These are only guidelines and tubes 0.5mm larger and smaller should always be available.

Once the tracheal tube is passed through the vocal cords its position must be carefully checked to ensure that there is equal ventilation of both lungs. The tube should then be securely fixed in position. Ventilation is continued using the self-inflating bag or T-piece system.

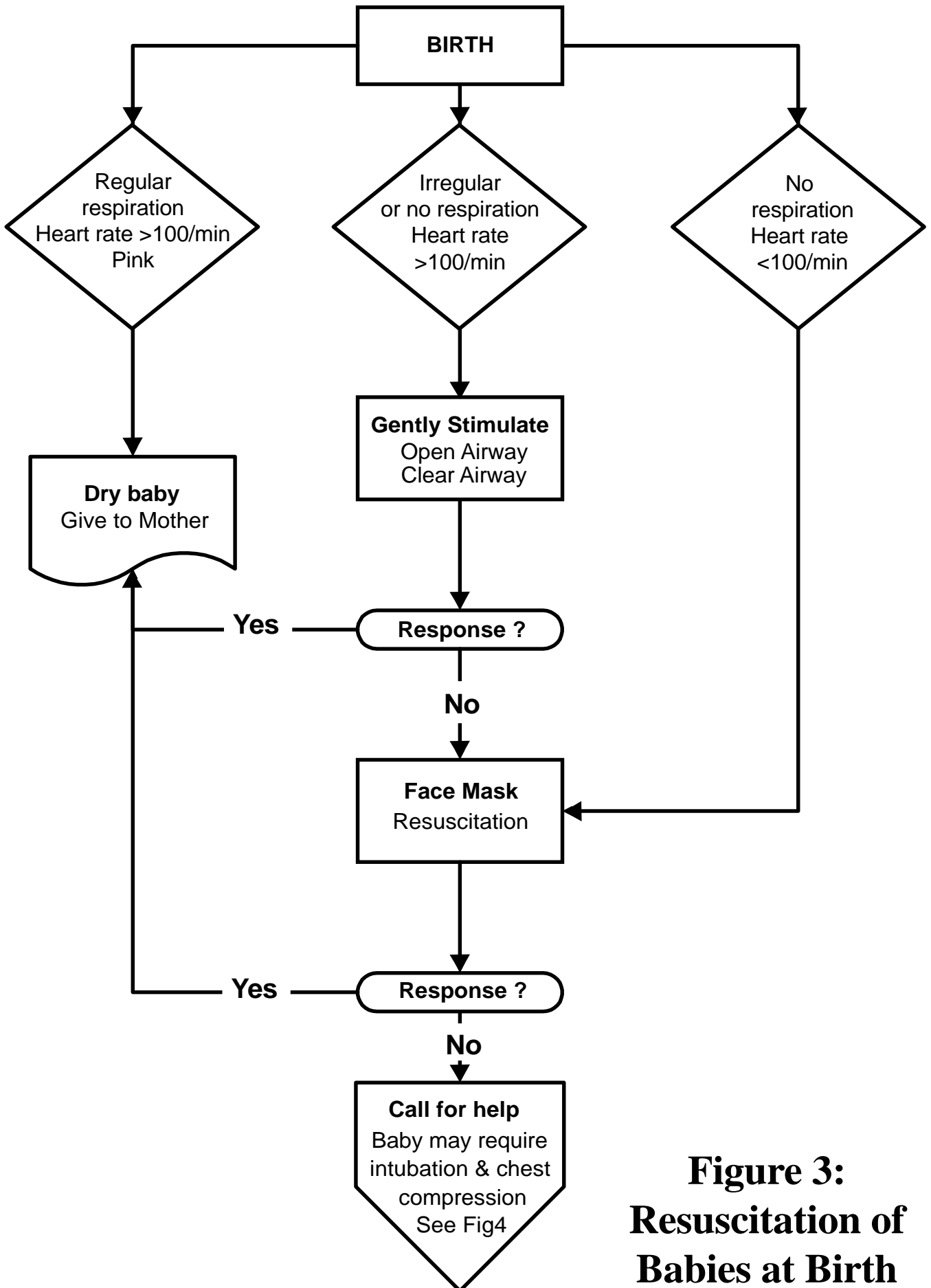
***WHEN THERE IS ANY DOUBT ABOUT THE POSITION OR PATENCY OF THE TUBE REMOVE THE TRACHEAL TUBE IMMEDIATELY AND REINTUBATE AFTER A BRIEF PERIOD OF OXYGENATION USING FACE MASK VENTILATION.***

## Circulation

The initial attempts at establishing a viable circulation are made using chest compressions. Chest compressions should be performed if:

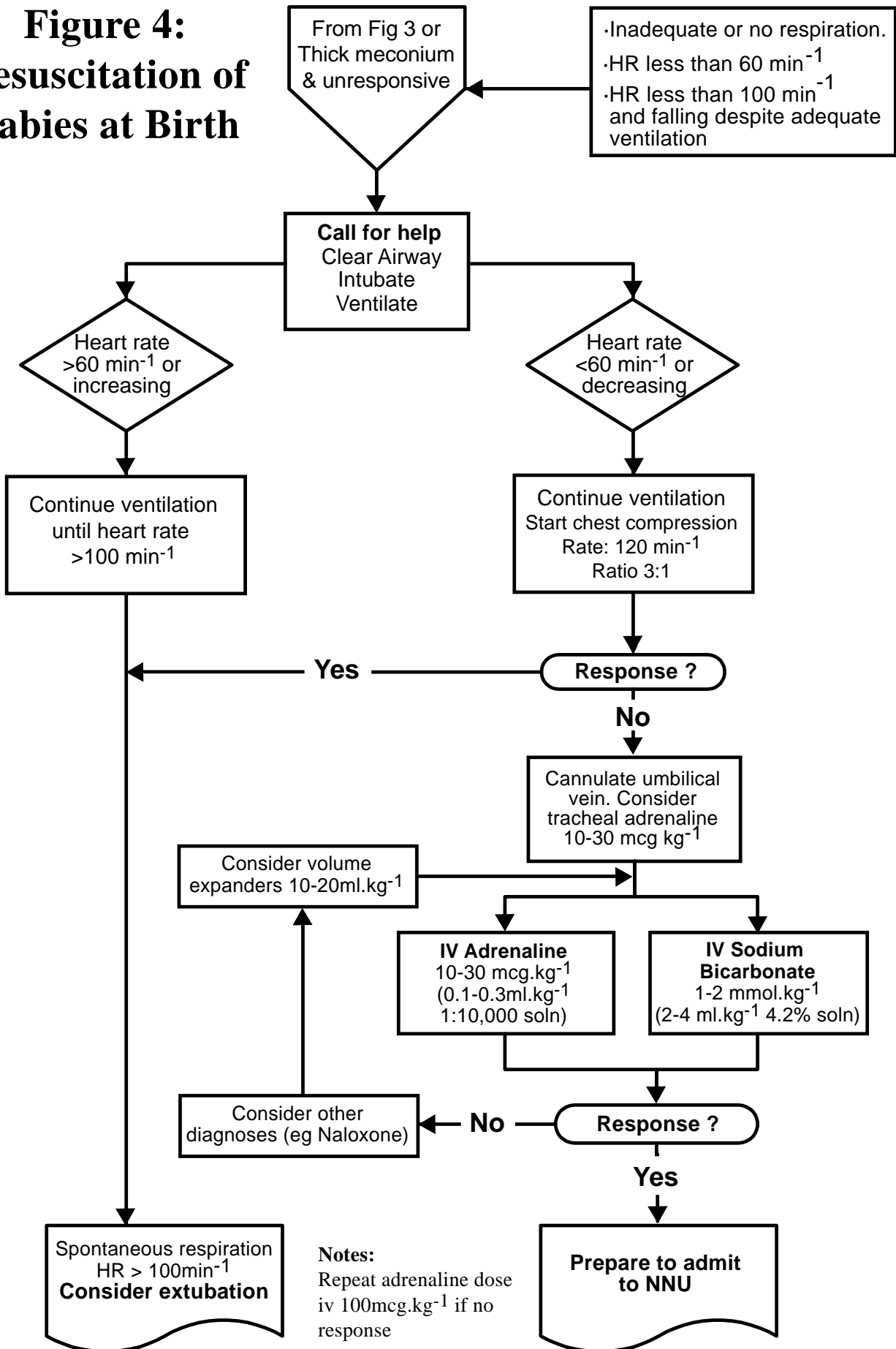
1. The heart rate is less than 60 beats/minute.
2. The heart rate is less than 100 beats/minute and falls despite adequate ventilation.

The optimal technique is to place the 2 thumbs side by side over the lower one third of the sternum with the fingers encircling the torso and supporting the back.<sup>105-107</sup> The lower third of the sternum<sup>45</sup> is compressed 2 - 3cm in a term baby at a rate of approximately 120 compressions per minute. The compressions should be smooth and not jerky and each compression should last 50% of the compression/relaxation cycle. An alternative technique is



**Figure 3:**  
**Resuscitation of**  
**Babies at Birth**

**Figure 4:  
Resuscitation of  
Babies at Birth**



to use the index and middle finger of one hand to compress the lower half of the infant's sternum. This allows the operator's free hand to perform simple resuscitation procedures whilst maintaining external chest compressions. A single ventilation should be performed after every three chest compressions. The pulse should be checked periodically and chest compressions only discontinued when the spontaneous heart rate of greater than 100 beats per minute is established.

If the infant fails to respond to active ventilation following intubation and chest compressions then venous access must be established. A failure of the infant to respond is usually as a result of inadequate ventilation and it is therefore essential to check the seal of the facemask or the position of the tracheal tube. When satisfied that there is optimal airway control and in the continuing absence of improvement, the umbilical vein should be catheterised using a 4.5 – 5 FG umbilical catheter. This is achieved by transecting the cord 1 - 2cm away from the abdominal skin and inserting the umbilical catheter until there is a free flow of blood up the catheter.

An initial dose of intravenous adrenaline,  $10\text{-}30\text{mcg.kg}^{-1}$  ( $0.1\text{ - }0.3\text{ ml.kg}^{-1}$  of  $1:10,000$  solution), should be given via the umbilical venous catheter, flushing the adrenaline through the catheter with 2ml of saline. If venous access fails, an intra-osseous needle can be inserted into the proximal tibia and this route temporarily used instead of the venous umbilical catheter. If there is a delay in establishing umbilical vein catheterisation or intraosseous access then the same dose of adrenaline,  $10\text{ - }30\text{mcg.kg}^{-1}$ , can be given through the tracheal tube. Despite the tracheal administration of adrenaline being widely practiced there is little evidence that it is effective.<sup>108-110</sup> It may be least effective if given before the lungs are fully inflated.

If there is still no response the baby should be given  $1\text{ -}2\text{ mmol.kg}^{-1}$  body weight of sodium bicarbonate slowly over 2-3 minutes. Use a 4.2% bicarbonate solution or mix a volume of 8.4% sodium bicarbonate solution with an equal volume of 5 or 10% dextrose or sterile water. This results in a concentration of  $0.5\text{mmol.ml}^{-1}$  solution. Basic life support must be continued. Sodium bicarbonate is a hyperosmolar solution and should be administered by slow infusion in preterm babies below 32 weeks because of the risk of inducing intracerebral bleeding. Further doses of bicarbonate are best given in response to the results of arterial blood gas analysis data.

Repeat doses of adrenaline should be given if the newborn

continues to fail to respond. Subsequent larger doses, up to  $100\text{mcg.kg}^{-1}$ , may be considered but there is evidence that the need for adrenaline during resuscitation is associated with a poor prognosis<sup>111</sup>.

Hypovolaemia in the newborn requires active volume replacement. Indications for intravenous fluid therapy are:

1. Evidence of acute fetal blood loss.
2. Pallor that persists after oxygenation.
3. Faint pulses with a good heart rate and poor response to resuscitation including adequate ventilation.
4. Fluid replacement, at  $10\text{-}20\text{mls.kg}^{-1}$ , can be given as 4.5% albumin, whole blood or plasma.

Finally, intramuscular naloxone ( $100\text{mcg.kg}^{-1}$ ) should be considered in the apnoeic newborn who rapidly becomes pink and who obviously has a satisfactory circulation on resuscitation. Naloxone is a narcotic antagonist and is specifically indicated where there is a history of recent therapeutic administration of opiates to the mother.

### Conclusion

Paediatric life support is an essential part of the resuscitation cycle. To be effective, those practising paediatric resuscitation at basic or advanced levels need to be properly trained and practised in the skills of the procedure. Delay or hesitation in recognising the need for or performing resuscitation will have dire consequences.

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## TOXICITY FROM LOCAL ANAESTHETIC DRUGS

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### Introduction

Toxic side effects of local anaesthetic drugs occur when excessive blood levels occur. This is usually due to:

- Accidental rapid intravenous injection.
- Rapid absorption, such as from a very vascular site ie mucous membranes. Intercostal nerve blocks will give a higher blood level than subcutaneous infiltration, whereas plexus blocks are associated with the slowest rates of absorption and therefore give the lowest blood levels.
- Absolute overdose if the dose used is excessive.

### Reducing the risk of toxicity

- Decide on the **concentration** of the local anaesthetic that is required for the block to be performed. Calculate the **total volume** of drug that is allowed according to the table below.
- Use the least toxic drug available.
- Use lower doses in frail patients or at the extremes of ages.
- Always inject the drug slowly (slower than 10ml / minute) and aspirate regularly looking for blood to indicate an accidental intravenous injection.
- Injection of a test dose of 2-3ml of local anaesthetic containing adrenaline will often (but not always) cause a significant tachycardia if accidental intravenous injection occurs.

- Most nerve blocks are more dependent on **volume** of drug injected than the **total dose**. Therefore if more volume is needed it is better dilute the local anaesthetic with 0.9% saline than to add more local anaesthetic and increase the dose unnecessarily.
- Add adrenaline (epinephrine) to **reduce the speed of absorption**. The addition of adrenaline will reduce the maximum blood concentration by about 50%. Usually adrenaline is added in a concentration of 1:200,000, with a maximum dose of 200 micrograms. This is made up by taking an ampoule of adrenaline with a concentration of 1:1,000 = 1mg/ml = 0.1mg%. From this you take 0.1ml (zero point one millilitres) and add it to each 20ml of the local anaesthetic. **The addition of adrenaline will make no difference to the toxicity of the local anaesthetic if it is injected intravenously.**
- Make sure that the patient is monitored closely by the anaesthetist or a trained nurse during the administration of the local anaesthetic and the following surgery.

### Signs and Symptoms of Local Anaesthetic Toxicity

The systemic toxic effects due to local anaesthetic overdose primarily involve the central nervous and cardiovascular systems. In general the Central Nervous System (CNS) is more sensitive to local anaesthetics than the Cardiovascular System (CVS). Therefore CNS manifestations tend to occur earlier. Brain excitatory effects occur before the depressant effects.

Drug	Maximum dose for infiltration	*Maximum dose for plexus anaesthesia
Lignocaine	4mg/kg	5mg/kg
Lignocaine with adrenaline	7mg/kg	7mg/kg
Bupivacaine	2mg/kg	2mg/kg
Bupivacaine with adrenaline	3mg/kg	3mg/kg
Prilocaine	6mg/kg	7mg/kg
Prilocaine with adrenaline / octapressin	8mg/kg	8mg/kg

\* when performing intercostal blocks, reduce the dose for infiltration by 25%

### CNS signs & symptoms

*Early or mild toxicity:* light-headedness, dizziness, tinnitus, circumoral numbness, abnormal taste, **confusion** and drowsiness. Patients often will not volunteer information about these symptoms unless asked. **Throughout the injection talk to the patient asking them how they feel. Any suggestion of confusion should alert you to the possibility of toxicity and you should stop any further injection.**

*Severe toxicity:* tonic-clonic convulsion leading to progressive loss of consciousness, coma, respiratory depression, and respiratory arrest.

Depending on the drug and the speed of the rise in blood level the patient may go from awake to convulsing within a very short time.

### CVS signs & symptoms

*Early or mild toxicity:* tachycardia and rise in blood pressure. This will usually only occur if there is adrenaline in the local anaesthetic. If no adrenaline is added then bradycardia with hypotension will occur.

*Severe toxicity:* Usually about 4 - 7 times the convulsant dose needs to be injected before cardiovascular collapse occurs. Collapse is due to the depressant effect of the local anaesthetic acting directly on the myocardium. Bupivacaine is considered to be more cardiotoxic than lignocaine. Severe and intractable arrhythmias can occur with accidental iv injection.

The acute toxicity of local anaesthetics is due to the speed of rise of blood concentration. Therefore a rapid injection of a small volume may cause toxicity.

### Essential Precautions

- **Always** secure intravenous access before injection of any dose that may cause toxic effects.
- **Always** have adequate resuscitation equipment and drugs available **before** starting to inject.

### Treatment of Toxicity

If a patient you are attending shows any signs or symptoms of toxicity during injection of local anaesthetic **stop the injection and assess the patient.**

**Treatment is based on the A B C D of Basic Life Support**

**Call for help while treating the patient**

**A.** Ensure an adequate **airway**, give **oxygen** in high concentration if available.

**B.** Ensure that the patient is **breathing** adequately. Ventilate the patient with a self inflating bag if there is inadequate spontaneous respiration. Intubation may be required if the patient is unconscious and unable to maintain an airway.

**C.** Treat **circulatory failure** with intravenous fluids and vasopressors such as ephedrine (10mg boluses) if hypotension occurs. Adrenaline may be used cautiously intravenously in boluses of 0.5 - 1ml of 1:10,000 (1mg in 10ml) if ephedrine is either not available or not effective in correcting the hypotension. Treat arrhythmias.

Start chest compressions if cardiac arrest occurs.

**D. Drugs** to stop fitting such as Diazepam 0.2-0.4mg/kg intravenously slowly over 5 minutes repeated after 10 minutes if required, or 2.5mg - 10 mg rectally. Thiopentone 1-4 mg/kg intravenously may also be used in theatre.

Observe the patient closely after any reaction.

Treatment of local anaesthetic toxicity is likely to have a good outcome if toxicity is recognised and basic resuscitation is started early. Monitor patients closely when using local anaesthetics. If a reaction occurs:

- Prevent hypoxia which will cause brain damage and make fitting or arrhythmias more difficult to control.
- Ensure that hypotension and arrhythmias are treated early.
- Ensure that fits are adequately treated.
- Most reactions are short-lived if the above advice is followed.

### Case History

A 20 year old mother who had just delivered a baby started to fit and then developed a cardiac arrest whilst a midwife was injecting lignocaine prior to suturing her episiotomy. Prompt resuscitation with airway, intubation and ventilation, chest compressions, intravenous fluid and adrenaline saved her life. When the ampoule of lignocaine was checked it was found that the midwife had used 10mls of 10% lignocaine for infiltration (1000mg), more than 5 times the maximum permitted dose for infiltration.

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# PAIN *Clinical Updates*

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## PAIN IN SICKLE CELL DISEASE

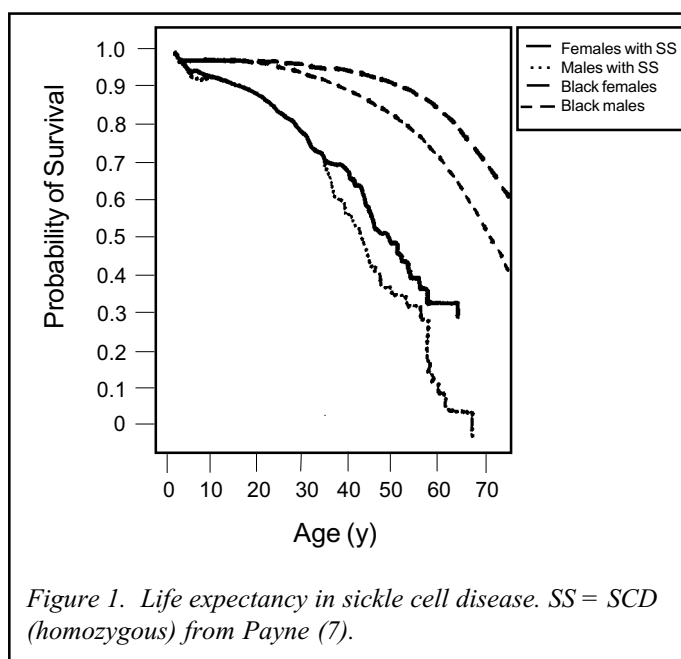
Pain in sickle cell disease (SCD) presents unique challenges for patients, families, and health care professionals. Pain is the most frequent problem experienced by people with SCD. It has profound effects upon comfort and function in work, school, play and social relationships(1). The frequency and severity of painful episodes are highly variable among patients. Some patients have pain daily but others only occasionally (1,2). Painful episodes may start in the first year of life and continue thereafter. The episodes last from hours to weeks followed by a return to baseline. Onset and resolution can be sudden or gradual. Dehydration, infection, stress, fatigue, menses, and cold (including air conditioning and swimming in cold water) can precipitate painful episodes (3). However, the majority of painful episodes have no clear precipitant. Patients experience a wide variety of symptoms spanning acute and chronic pain and assessment and management must be suitable for both. Because pain and SCD itself are lifelong problems that have profound effects upon the quality of life, understanding of individual development and adopting a biopsychosocial approach are crucial(4). The experience of pain varies with each developmental phase(5,6) as should assessment and treatment.

Painful episodes are often termed "crises." Some avoid the term to emphasize that a major goal of treatment is to take the sense of catastrophe out of the crisis. However, replacing a word does not change perceptions. The same is true for the strength, resilience, and vulnerability of each patient, as reflected in coping skills, mood, social life, and function.

Pain—the most frequent problem experienced by people with SCD—profoundly affects comfort and function in work, school, play, and social relationships.

### Barriers to Care

Many barriers may impede humane and competent assessment and management of SCD-related pain(4,8). First, most patients with SCD are of African ancestry, but the majority of health care professionals in developed nations are not. Patients and health care professionals often differ in culture and socio-economic standing. Cross-racial and cross-cultural communications can be fraught with difficulty, and there is no reason to assume that the medical arena is immune from the conflicts of society(9). Second, access to health care may be difficult



for many with SCD. For example, non-pharmacologic treatments for chronic pain and chronic illness may be unavailable, unaffordable, or not covered by health insurers. Third, many patients are treated in large tertiary care hospitals where continuity of care is poor and a biomedical orientation overshadows attention to the psychosocial aspects of pain and chronic illness. Fourth, risks of addiction to analgesic medication are overstated frequently(4,10). Fifth, the variability and unpredictability of pain in SCD may make it difficult for the patient to cope and this may lead to the adversarial relationship often observed between patients with SCD and health care professionals.

The term “painful episode” is now used in preference to “crisis”, which suggests catastrophe.

## Assessment

Assessment sets the tone for the therapeutic alliance, includes the patient and family in the treatment process, and underlines the respect and concern of the health care professional. It is the foundation of intervention. Assessment during the acute event is brief and focused to guide pharmacotherapy. For a patient with more frequent painful episodes, comprehensive assessment is necessary(11,12). This is best performed when the patient feels well and free of severe pain during an outpatient visit or at discharge from the hospital. Assessment includes physiological, psychological, social, cultural, and spiritual aspects of the pain. It considers not only the patient, but also the family and, where appropriate, the health care system. Assessment of the health care system is often forgotten, but difficult pain problems cannot usually be resolved until this is explored. Such assessment involves a review of the entire network treating the patient, as well as

specific groups and individuals such as nurses, physicians, and social workers. Strengths and problems are identified. Including the health care system in any assessment includes everyone involved in the process of care, beyond just patient and family, and reinforces the mutual responsibility inherent in therapeutic relationships.

The basic principles of pain assessment are universal(11,13). Cognitive and affective states and developmental level must be considered. Frequent reassessment is essential to titrate treatment of pain that rapidly waxes and wanes. The accuracy of the assessment rests on many factors, including past experiences with the health care system(8). Patients do not automatically trust health professionals, especially when they have encountered untrustworthiness. Patients whose pain has been managed inconsistently and inadequately by an ever-changing roster of health care professionals may, for example, always rate their pain as 10 out of 10 or exhibit unusual pain behavior in an attempt to obtain adequate analgesia. Consistent, marked differences between the verbal report and observed behavior warrant further investigation. Factors influencing such discrepancy could include stoicism, past experiences of disbelief and inadequate analgesia, learned coping skills, emotional distress, family dysfunction, or entrenched adversarial relationships with the health care system(12).

## Treatment

Medication is the mainstay of treatment for the acute episode but it is only one part of an integrated and individualized treatment plan. Patients and families must be included in the development of any plan. Such inclusion is itself therapeutic, as it reinforces self-efficacy and control(11,12). Consistency is important in treating this unpredictable and inconsistent pain problem, and will serve to reduce anxieties about the type and amount of analgesics to be given.

Medication includes nonsteroidal anti-inflammatory drugs (NSAIDs) and acetaminophen/paracetamol, opioids, adjuvants such as tricyclic antidepressants and invasive approaches such as epidural analgesia. The World Health Organisation ladder for the treatment of mild, moderate, and severe cancer-related pain is applicable to the management of acute episodes of pain associated with SCD(14). NSAIDs or acetaminophen/paracetamol alone are used for mild pain. A mild opioid is added for moderate pain, and strong opioids, sometimes administered by the parenteral route, for severe pain. In general, the types of drugs, route of administration, and schedule (e.g., patient-controlled analgesia) follow recommendations for opioid management of other acute pain problems(11,12,13,15).

Although meperidine/pethidine has commonly been used for SCD-related pain, it is no longer recommended because of the risks of seizures and dysphoria(16). However, many adults with SCD have used meperidine/pethidine successfully for years without side effects. A

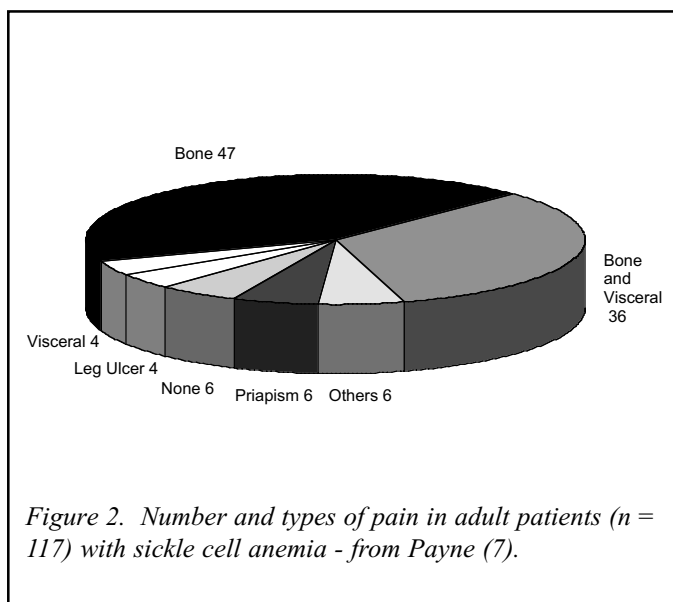
recommendation for abrupt change, especially during an acute painful episode by a physician who is not a primary or trusted caregiver, may evoke resistance and scepticism from the patient. Adherence to guidelines should not supersede concern and respect for the patient's beliefs and past experiences; maintenance of the therapeutic alliance is of prime importance after safety.

We must try to identify high-risk patients early, and provide them with effective, individualized interventions before dysfunction and adversarialism are entrenched.

NSAIDs are often considered benign and preferable to opioids. However, particular risks of NSAID use exist in SCD. Blood loss from occult gastritis, although often unnoticed in other patients, may destabilize precarious haemodynamic compensation in chronic anaemia. Since NSAIDs and acetaminophen/paracetamol are used throughout life, the risk of analgesic nephropathy must be considered, especially in patients who are already at risk of renal failure from SCD(17). Patients and physicians may overuse NSAIDs and acetaminophen/paracetamol in an effort to avoid opioids. This potentially dangerous practice reflects widespread, unfounded fear of opioid addiction.

Average opioid requirements are higher for patients with SCD than for other patients with acute pain(18). Patients often become tolerant with frequent opioid use. Also, it should be emphasised that SCD pain can be exceptionally severe; patients with SCD who have undergone surgery often rate their SCD pain as more severe than postoperative pain(17). Since the degree of opioid tolerance in any single patient is usually unknown, the initial dose must be chosen with concern for both analgesia and safety. After the initial dose, rapid titration to effect is necessary. Severe pain in SCD is an emergency, and patients must be made comfortable as soon as possible. Titration to effect should take only one to two hours, not one to two days.

The majority of painful episodes are managed at home(1). Severe episodes generally require parenteral analgesics. Some patients, however, manage even severe pain with strong oral opioids at home. Parenteral administration is necessary if the patient is vomiting, obstructed, experiencing intractable pain, or otherwise unable to take or benefit from oral agents. Pain management at home has advantages and disadvantages(19). The patient remains in familiar circumstances. Support from the family may facilitate partial continuation and early return to activities of daily living, such as work, school, and social interaction. Without a supportive family at home, safe and effective care is impossible. However, health professionals may overlook the deleterious effects on mood and function of frequent painful episodes inadequately managed at home.



For example, children and adolescents may miss considerable amounts of school, ultimately developing school avoidance, failure, or dropout, without adequate educational intervention by the health care team(1). Also, pain may accompany and mask life-threatening events. Chest pain may herald acute chest syndrome and respiratory failure. Therefore, despite pressure to reduce costs of hospitalization, managing patients at home may potentially be deleterious to comfort and safety.

The use of chronic daily opioids in patients with frequent or daily severe pain is controversial. Many clinicians report improved comfort and function in patients who were formerly debilitated by daily pain(20). Others are concerned about perpetuation of the pain syndrome by symptoms of withdrawal, along with exacerbation or lack of recognition of interacting factors such as depression, anxiety, or intolerable life stresses. Patients for whom daily chronic opioids are considered should undergo in-depth evaluation of physical, psychological, social, and spiritual factors contributing to the pain, and have treatable problems addressed(21). Tricyclic antidepressants may be indicated for treatment of both pain and depression. Clinicians often face the dilemma of treating chronic pain in patients with SCD who could benefit from well-coordinated multidisciplinary interventions that are inaccessible because of economic or geographic barriers. Inequities in the availability of appropriate pharmacologic and non-pharmacologic care raise ethical issues involving allocation of resources, justice, and beneficence.

Physicians greatly overestimate the incidence of addiction in patients with SCD(10,22). This bias results in inadequate dosing, as-needed rather than around-the-clock administration and use of only nonopioid or mixed agonist-antagonist medications for moderate to severe pain. Patients resent being labeled by health care professionals who assume that their request for medication in specific

doses and routes represents addiction rather than the patient's experience and knowledge of what works(23). Having SCD seems neither to increase nor to decrease vulnerability to addiction. Certainly some patients with SCD are addicted. Yet addiction exists in almost every subpopulation in society and our current knowledge is that the incidence in patients with sickle cell disease parallels that of the general population, and may be less than the incidence in health care professionals.

Clinicians have employed epidural analgesia for painful episodes but this practice is controversial(24). This approach can be effective in the short run, and in some cases, such as acute chest syndrome, may be life-saving by providing adequate analgesia without hypoventilation. However, before recommending any intervention the chronicity of the pain must be considered. The goal is not just to manage current pain, but also to enhance future pain management and improve coping with the pain and illness. The potential benefits and drawbacks must be weighed against a lifetime of pain. Problems experienced with long term use may include deleterious side effects such as epidural adhesions at the treatment site. In addition, high-technology approaches can be onerous and frightening for patients and families, so that even as pain itself is reduced during the episode, overall quality of life might suffer. The author recommends epidural analgesia only for particularly severe episodes refractory to the patient's ordinary treatment.

Having SCD seems neither to increase nor to decrease vulnerability to addiction.

Psychological and spiritual approaches and modalities are presumed useful in SCD related pain, as for other acute and chronic pain syndromes. A *sine qua non* is continuity and predictability of care within a trusting relationship. Without this, individual approaches applied in a random manner may be ineffective. Endorsement and support of the primary care physician for cognitive, behavioral, and psychological approaches are essential. Otherwise the patient often resists, fearing (sometimes accurately) dismissal, labeling of the pain as "not real" and of himself or herself as "mentally ill" or undesirable, or substitution of these approaches for adequate medication.

In SCD, although genetic and physiologic factors determine disease severity, psychosocial factors and coping skills interact with pathophysiology to determine the severity of pain and its impact on the individual and the family(25). Certain coping skills correlate with reduced impact of the pain and the illness. However, these are associations and not a mathematical model. As pain becomes more severe and frequent, the greater its impact on the individual, and the harder it will be for that individual to use effective coping skills.

Advancing knowledge [alone] will not benefit the majority of patients without concurrent changes in beliefs and attitudes.

Physical approaches include but are not limited to TENS, heat, positioning, and splints for a painful extremity. Cold exacerbates vaso-occlusion and worsens pain so in general should not be used.

## Future Directions

Research on SCD-related pain is sparse, but undertreatment of pain in patients with SCD is not due to lack of knowledge. Instead, barriers to implementing available knowledge in clinical practice are the major reason for this shortfall(4,8,10,22) As has been found for the treatment of cancer pain, advancing knowledge through research, while clearly desirable, does not benefit the majority of patients without concurrent changes in beliefs and attitudes(26).

Current health care systems inadequately address the needs of patients with SCD-related pain. Many painful episodes cannot and (at least initially) should not be managed at home, but the environment of acute care usually is not conducive to timely and adequate analgesia. Although emergency departments are the gateway to care for patients with SCD-related pain who cannot manage at home, most provide neither consistent care nor adequate broadly-based assessment and follow up. Some hospitals have established short-stay or day-treatment facilities where patients with SCD receive parenteral analgesia early in the course of the painful episode(27). This approach provides immediate, accessible treatment that may shorten the duration and severity of the episode and obviate the need for acute care hospitalization.

Some patients function well in life despite frequent and intense painful episodes. Others do not. Patients whose functioning is impaired often display psychosocial dysfunction by adolescence. Signs of high-risk status include increasing numbers of hospitalizations for pain, increasing school absences, school failure, increased use of analgesics, family discord and dysfunction, and mood changes(4). An adversarial relationship with health care professionals often develops and intervention is necessary before this becomes entrenched and function is reduced. We must try to identify high-risk patients early, and provide them with individualized, multi-faceted interventions. Waiting to do so until adulthood may doom all attempts to failure.

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## MANAGEMENT OF THE MAJOR BURN

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The medical management of burns is both urgent and predictable.

### DEFINITION OF A MAJOR BURN

A major burn can be defined as any burn that requires intravenous fluid resuscitation (10% Body Surface Area (BSA) in a child, 15% in an adult) and / or a burn to the airway.

### TYPES/CLASSIFICATION

Beyond simple erythema, burns are either partial or full thickness depending on whether the basement membrane has been lost. On examination a full thickness (3<sup>rd</sup> degree) burn is usually pale, bloodless and insensitive to the firm touch of a sterile needle. Partial thickness burns can be further divided into superficial (1<sup>st</sup> degree) and deep (2<sup>nd</sup> degree), which refers to the depth at which the dermal layer is injured. Sensation is preserved and healing of the skin more likely.

The mechanism of the burn can be classified into six categories

- *Contact* - direct contact with a hot surface.
- *Scald* - hot fluid/gas usually causing a superficial burn.
- *Flash* - a brief burn, usually partial thickness
- *Flame* - usually full thickness
- *Chemical*
- *Electrical*

### BURN PATHOLOGY

#### Local

Within moments the capillaries of the injured tissue become leaky. Plasma is lost, drawing water with it. This continues for between 3 and 36 hours and results in oedema of the tissues involved. Local airway swelling may lead to loss of the airway by both internal and external oedema. Chest wall oedema may make ventilation difficult and oedema of the limbs may cause ischaemia leading to limb loss (especially if the burn is circumferential).

Hypovolaemia and haemoconcentration of the blood leads to a rising haematocrit which will result in poor systemic tissue perfusion. This is 'Burns Shock'. Red blood cells are lost both directly in the burn and as a result of increased fragility.

### Systemic

Damaged tissue will release 'middle molecules' (leukotrienes, prostaglandins, oxygen free radicals and histamine) into the circulation leading to a systemic increase of capillary permeability.

**Burn Shock** is defined as the inability of the circulation to meet the needs of tissues for oxygen and nutrients and the removal of the metabolites. The clinical picture of severe shock consists of pale cold skin and a rapid yet thready pulse. Respiration is rapid and shallow leading to gasping ('air hunger'). Urine output falls and the patient becomes increasingly restless and disorientated. Often consciousness is lost only as a preterminal event.

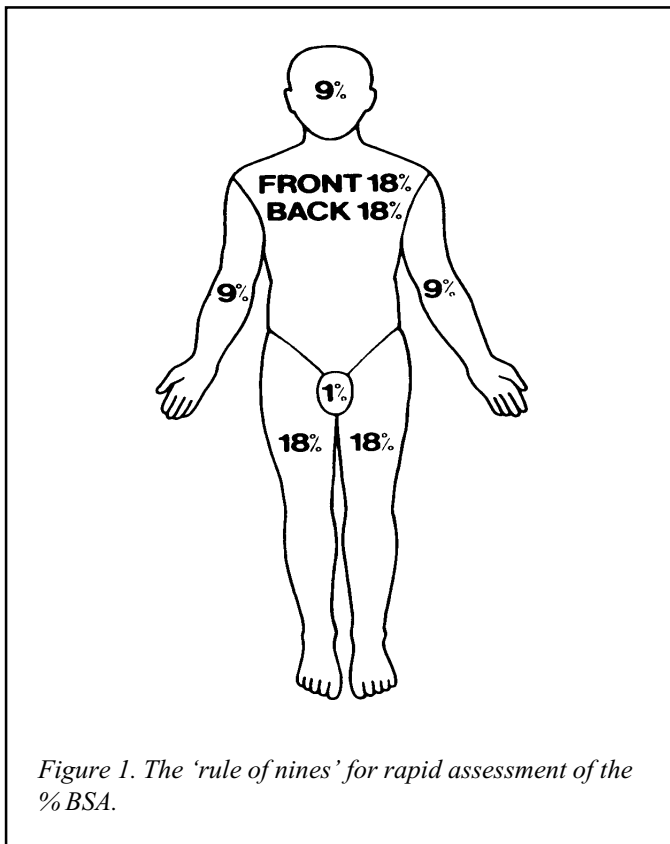
### MANAGEMENT PRINCIPLES

#### First aid

Remove the casualty from further injury. Extinguish flames, remove clothing, turn off the electrical source, or douse the chemically burnt patient with water. Flames ascend so lie the patient down. Cover the burn with a clean dressing, avoid the patient getting cold and transfer to a hospital as soon as possible. Additional oxygen should be given during transfer.

#### Primary management

- *Airway* - check the airway is clear. Endotracheal intubation is necessary if there are deep burns to the face and neck, soot in the nostrils, burns of the tongue and pharynx, stridor or hoarseness.
- *History* including time and nature of the incident (Wet or dry burn /chemical /electrical /inside or outside).
- *Weigh* the patient.
- *Examine* the burn and assess the size with the 'rule of nines' (figure 1) to give a %BSA.
- *Intravenous access* - obtain large bore venous access, even through burnt tissue.
- *Blood sampling* - samples for haematocrit, electrolytes, crossmatch, arterial blood gases and carboxyhaemoglobin levels.
- *Analgesia* - intravenous morphine, ketamine, or Entonox
- *Catheterise* - assess urine output as a gauge of tissue perfusion and adequate resuscitation.



With colloid resuscitation, less volume is required and the blood pressure is better supported. However they are expensive, often unavailable and tend to leak out of the circulation and may result in later oedema especially in the lungs.

Controversy remains as to which fluid should be used. Inhalational injury may increase fluid requirements by 50%. Both regimes require regular assessment as to the adequacy of resuscitation. This includes blood pressure, pulse, capillary return, urine output, level of consciousness and haematocrit. Additional fluid should be given if resuscitation is inadequate.

CHART FOR ESTIMATING SEVERITY OF BURN WOUND

NAME \_\_\_\_\_ WARD \_\_\_\_\_ NUMBER \_\_\_\_\_ DATE \_\_\_\_\_  
AGE \_\_\_\_\_ ADMISSION WEIGHT \_\_\_\_\_

LUND AND BROWDER CHARTS

IGNORE SIMPLE ERYTHEMA

Partial thickness loss (PTL)  
Full thickness loss (FTL)

REGION	%	
	PTL	FTL
HEAD		
NECK		
ANT. TRUNK		
POST. TRUNK		
RIGHT ARM		
LEFT ARM		
BUTTOCKS		
GENITALIA		
RIGHT LEG		
LEFT LEG		
TOTAL BURN		

RELATIVE PERCENTAGE OF BODY SURFACE AREA AFFECTED BY GROWTH

AREA	AGE 0	1	5	10	15	ADULT
A=1/2 OF HEAD	9 1/2	8 1/2	6 1/2	5 1/2	4 1/2	3 1/2
B=1/2 OF ONE THIGH	2 3/4	3 1/4	4	4 1/2	4 1/2	4 3/4
C=1/2 OF ONE LEG	2 1/2	2 1/2	2 3/4	3	3 1/4	3 1/2

Figure 2. The Lund and Browder chart for accurate assessment of the %BSA.

- Reassess the patient thoroughly at regular intervals and also the burn.

### Fluid resuscitation

This should be instituted as soon as possible. There are two simple protocols that both depend upon the %BSA, time passed since injury and patients weight. The rule of nines may over-estimate the BSA, but the Lund and Browder chart gives a more accurate assessment. (Figure 2.) Fluid requirements may be greater than the protocols suggest.

**Parklands:** Crystalloid resuscitation with Hartmanns

24 hour fluid requirement =  $4 \times \%BSA \times Wt (Kg)$

Give half over the first 8 hours, and the remainder over the next 16 hours

Although there may be pronounced generalised oedema initially, as large volumes are required, it is cheap and produces less respiratory problems later on.

**Muir and Barclay:** Colloid resuscitation with plasma

The first 36 hours are divided into time periods of 4,4,4,6,6,12 hour intervals

Each interval =  $0.5 \times \%BSA \times Wt (Kg)$

Water loss is related to evaporative and other extrarenal losses and may lead to a hypernatraemia. Salt intake should be balanced against the plasma sodium concentration, but is usually about  $0.5 \text{ mmol/kg}/\%BSA$ . If the burn is left exposed in an hot environment, sodium free water intake must be increased, but only to achieve a moderate hypernatraemia. Aggressive water load may lead to a low plasma sodium and result in 'burn encephalopathy'. Hyperkalaemia usually associated with severe muscle damage may require correction with insulin and dextrose.

### Airway management

A high index of suspicion is required regarding the patient's

airway. Laryngeal oedema develops from direct thermal injury leading to early loss of the airway. With signs of an airway burn (soot in the nostrils/stridor/hoarse voice) consider early intubation of the patient. If in doubt it is better to protect the airway (and be able to provide tracheo-bronchial toileting) than to risk losing the airway altogether. A tracheostomy may be necessary if there is any delay in securing the 'at risk' airway.

The airway is further endangered by an associated loss of respiratory drive due to a depressed level of consciousness (eg head injury or carbon monoxide poisoning). Again intubation may be required.

## BURN MANAGEMENT

**Dressings** are necessary to reduce infection and adsorb exudate. Bactericidal agents, such as silver sulphadiazine 1% and silver nitrate are used. Antibiotic preparations should be avoided to prevent resistant colonisation developing. Regular, often daily, dressing changes are recommended, and the patient should be washed with clean warm water.

### Surgery

Circumferential burns will require immediate surgery to improve circulation to distal extremities or to permit adequate breathing if the chest wall is burnt. Early excision and grafting is preferred as it minimises infection and hastens wound healing. The whole burn should be excised within 48 hours. Regular dressing changes, further excision and grafting may be required. It should be remembered that blood loss may be excessive at these times. Blood loss can be reduced by using diathermy and/or applying gauze soaked in adrenaline (1:200,000) during the burn excision.

The problem with excision of a large burn is often the lack of donor skin to cover the excised burn. The patient's donor skin can be meshed, so as to increase the size. It can then be covered with cadaveric skin which acts as a biological dressing with growth stimulating properties. Artificial bovine skin (such as *Integra*®) may also be used but are expensive.

### Anaesthesia

If there is any concern over the airway a gas induction following pre-oxygenation or a fibre-optic intubation are the safest options. Suxamethonium should be avoided after the first 48 hours up to 2 years after a major burn because it may result in a large increase in serum potassium. Analgesia requirements are increased. Give Entonox, ketamine or morphine (titrated to response).

Consideration should be given to altered pharmacokinetics:

- Volume of distribution increases for water soluble drugs (resistance to non-depolarising agents occurs.)
- Increased extracellular fluid:intracellular fluid ratio
- Albumin falls - less protein binding
- Increased metabolic rate / temperature leading to altered half life

Monitoring must include vital signs, temperature and urine output. Invasive monitoring may be necessary. The ECG leads can be placed with the use of staples if the chest wall is burnt.

Postoperatively the patient should be admitted to a high dependency unit, so that the continuing fluid loss following burn excision can be maintained.

## SYSTEMIC MANAGEMENT

**Pulmonary** Smoke may damage the lungs in three ways:

- Intoxication/hypoxaemia
- Respiratory tract injury due to irritants
- Thermal damage

**Carbon monoxide** has a higher affinity for haemoglobin than oxygen, and will lead to tissue anoxia. The severity of the poisoning depends on the proportion of Hb combined with carbon monoxide. Symptoms range from a throbbing headache with nausea and vomiting (<30%) to coma, convulsions and cardiac arrest (>60%). 100% oxygen is the initial treatment and may necessitate intubation and ventilation. The half-life of carboxyhaemoglobin (COHb) in air is 4 hours and in 100% oxygen, 80 minutes. Hyperbaric oxygen may be useful especially if there are significant neurological signs but may be difficult in severely burnt patients. Pulse oximeters are inaccurate in the presence of carboxyhaemoglobin and cannot be relied upon.

Other gases such as hydrogen sulphide, hydrogen cyanide and hydrogen chloride may be inhaled, particularly if paints or furniture have been involved in the fire. Cyanide poisoning is difficult to diagnose but a high index of suspicion should be had if the patient is apnoeic and has a metabolic acidosis indicating tissue hypoxia. Treatment is beyond the scope of this article but ventilation and antidotes (sodium thiosulphate) will be necessary. It is also important to consider the rescuers who may have been exposed to these gases.

Soot is an irritant that will cause chemical injury if left in the airway. It can be treated with Bicarbonate (1.8%) nebulisers. Chemical toxins formed from combustion include chlorine, ammonia and phosgene. They have variable penetration into the respiratory tract but may cause serious airway injury.

Thermal injury from flame or hot gases may also injure the upper respiratory tract although it is rarely severe as the upper airway will rapidly cool 'dry heat' and thereby minimise injury. Steam inhalational, however, can cause lower alveolar damage and carries a poor prognosis.

Suspicion of a potential inhalational injury should be aroused if there is a history of a fire in an enclosed space, disturbed consciousness, facial burns, coughing, hoarse voice, airway soot and cyanosis. Specific investigations include COHb levels, arterial blood gases, and fiberoptic bronchoscopy. The management involves oxygen therapy and mechanical ventilation along with physiotherapy and tracheo-bronchial toileting. Increased fluid requirements will be necessary. If acute respiratory distress syndrome develops the patient will require specialist intensive care support.

**Renal** failure may occur as a complication of renal hypoperfusion (inadequate resuscitation), septicaemia or haemoglobinuria/myoglobinuria. The latter may require alkalinisation of the urine in addition to maintaining an adequate perfusion pressure and the administration of mannitol (1g/Kg). Early renal dialysis may become necessary. Renal failure in association with burns has a high morbidity and mortality.

**Haematology** Intravascular haemolysis along with wound losses will increase blood transfusion requirements. Haemoglobinuria may occur.

**Nutrition** Patients are profoundly catabolic with a BMR peaking at 4 days. These patients require early and aggressive feeding preferably enterally to maintain intestinal mucosal integrity. Glutamine rich feeds may further reduce mucosal breakdown and also improve immune function. Curling's ulcers are associated with severe burns but can be prevented by early feeding and antacid prophylaxis.

**Cerebral** Hyponatraemia complicating resuscitation may result in the *burn encephalopathy syndrome*. This is seen as cerebral irritability.

### Sepsis

Burn injury is associated with a generalised loss of immunocompetence, and sepsis remains a major cause of death in burns. Early sepsis (1-3 days post burn) is usually streptococcal or staphylococcal. Late sepsis is usually due to Pseudomonas, acinetobacter and fungi. Prophylactic antibiotics should be avoided and instead regular cultures taken and appropriate therapy given when indicated. Some specialist burns centres recommend systemic decontamination of the digestive tract (SDD). Tetanus toxoid must be given.

### Other problems

Consider the possibility of underlying medical conditions that may have led to the burn injury for example epilepsy, a cerebrovascular event, hypoglycaemia, drug or alcohol overdose. Always attach an ECG if there is a history of heart disease or carbon monoxide inhalation.

### Conclusion

Effective management of the burnt patient depends upon early and adequate fluid resuscitation, a high level of suspicion over the airway safety and continuous assessment of the whole patient. Early transfer to a specialist centre is desirable.

#### Example of fluid management:

A 70kg patient with 50% body surface area burn would require:

$$4 \times 50 \times 70 = 14000 \text{mls of Hartmanns solution over 24 hours.}$$

Therefore 7 litres should be given in the first 8 hours and 7 over the following 16 hours.

(Calculated with the Parklands formula)

Regular reassessment of the adequacy of resuscitation should be performed. Blood products and colloid may also be given in addition to these requirements.

## OXYGEN CONCENTRATORS FOR DISTRICT HOSPITALS

*Michael B Dobson, John Radcliffe Hospital, Oxford, UK*

Oxygen is one of the most basic drugs we have. In many acute illnesses such as acute respiratory infections, asthma, fetal asphyxia, and shock the availability of an oxygen supply can save a patient's life. Many small or remote hospitals have difficulty obtaining a reliable oxygen supply because of logistical and cost factors.

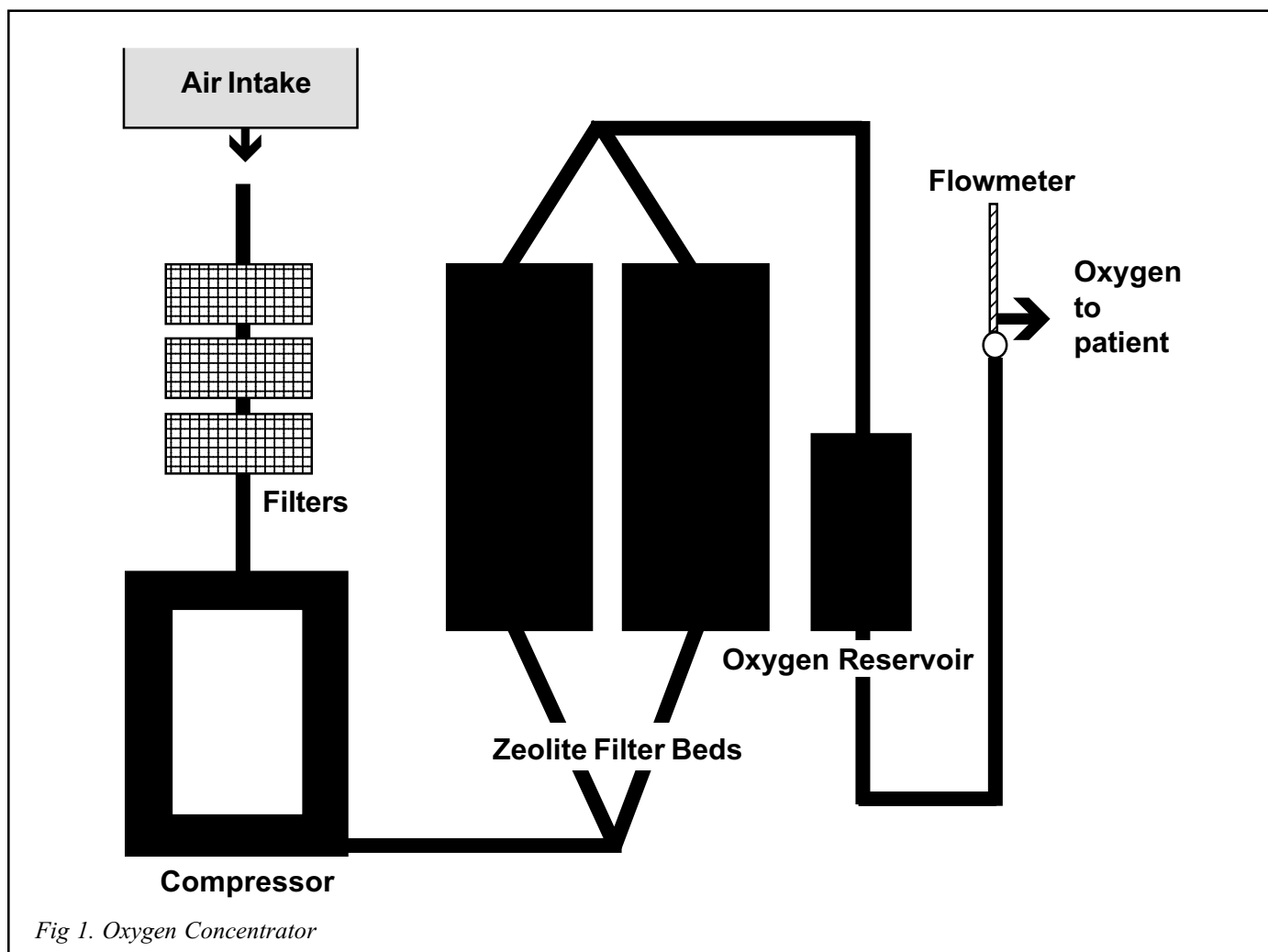
Oxygen supplies in cylinder form require a reliable system for supply and transportation. In many countries the cylinders themselves have to be purchased rather than rented, and losses in transit may be considerable. The cost of oxygen itself is considerable - it is relatively expensive to manufacture because of the high energy costs of the processes used to make it. An additional cost premium is usually levied on "Medical" oxygen, although it is generally indistinguishable from the industrial grade. In Tanzania, perhaps a typical developing country, a recent survey showed that 75% of the district hospitals had an oxygen supply for less than 25% of the year.

The physical separation of gases from a mixture at room temperature is the underlying principle of oxygen concentrators. This low energy method has been developed in recent years to allow separation of oxygen from air, and has been applied to all scales of size from an incubator to a complete steelworks.

The largest application of oxygen concentrators in medicine has been in the provision of portable, domiciliary machines for the provision of long term home oxygen therapy to patients with chronic lung disease. The prime reason for this development was the dramatic cost savings which could be achieved. The purchase price of a concentrator is about half the cost of a year's supply of oxygen from cylinders.

### How Concentrators Work (See Figure 1)

Room air is drawn into the machine through a series of filters, compressed to a pressure of 4 atmospheres, and passed into a column containing zeolite, a "molecular sieve"



*Fig 1. Oxygen Concentrator*

of aluminium silicate. Nitrogen binds to the zeolite, while oxygen passes through. Before the column becomes saturated with nitrogen the flow of air is switched to a second column; the first column is vented to atmosphere, discharging most of the nitrogen, and the remainder of the nitrogen is removed by back flushing the column with a small flow of oxygen from the second column. As the second column approaches saturation the process is reversed. The life of the zeolite crystals can be expected to be at least 20,000 hours which in most situations would give about 10 years' use. The gas emerging from the columns (95% oxygen) passes through a small reservoir chamber, and a flow control system to the patient. Most domiciliary sized machines can produce a flow of up to 4 litres/minute of oxygen. Higher flows result in a loss of concentration, and most machines are flow-limited to prevent this from occurring.

### Are Concentrators Reliable?

In recommending technology from the western world to the developing world it is important to have an established reliability record, and oxygen concentrators certainly meet this requirement. Domiciliary concentrators have been in regular use for more than ten years and have in general proved very reliable. They do, of course, need regular servicing, usually after every 5000 hours of use. (As a comparison, after 5000 hours of driving at an average speed of 50 km/hr a car will have covered 250,000 km). Servicing is not complicated and can if necessary be carried out by the user.

There are great differences in environment between the home of a western patient with respiratory failure and a remote hospital in the tropics. For this reason a group of clinicians and engineers under the auspices of the World Health Organisation and the World Federation of Societies of Anaesthesiology has established a set of tests and performance criteria which a small concentrator has to fulfil to be of maximum value in a small hospital.

### The WHO Performance Standard

An International standard (ISO) already exists for small concentrators, relating mainly to safety in use. To reach the WHO standard, the machine must first meet the ISO standard, and in addition to this it must be capable of functioning in adverse circumstances, including:

- ambient temperature of up to 40°C
- relative humidity up to 100%
- unstable mains voltage

- excessively dusty environment

It must pass stringent tests including military standard shock, vibration, and corrosion tests and be incapable of delivering an output oxygen concentration of less than 70%.

Every machine must be supplied with a comprehensive service manual and at least 2 years supply of replacement spares.

### Results in the Field

Oxygen concentrators have been field tested in a number of developing countries, including Malawi, Mongolia and Egypt (a future article will describe the results from these studies) and have proved themselves to be both reliable and effective. It is difficult to imagine more different environments than Europe, Malawi, Egypt and Mongolia - yet the concentrators have been successfully used in all of them. One of the main reasons for the success of the projects has been the training given to users and technicians - concentrators are not hard to service, but like any machine they do need to be looked after and kept clean.

### Should our hospital buy one?

Experience from the field trials, and a cost study based on Papua New Guinea have shown very considerable cost savings to be possible, as well as the year-round availability of oxygen which is lacking in so many hospitals. The power consumption of a concentrator is only 350w, so even hospitals without a mains supply can use one. A portable 600w petrol generator is a sufficient power source, as is a truck battery fitted with an oscillator. The outlet pressure of a concentrator is only sufficient to ensure flow of oxygen to the patient. Small concentrators are not intended, nor are they suitable as a power source for devices such as a compressed gas anaesthesia (Boyle's) machine or a lung ventilator (although they can of course provide a supply of oxygen for use with an independently powered ventilator). Remember also that if the power fails, the oxygen reservoir inside the machine will last only 2-3 minutes.

The minimum number of concentrators should be two per hospital. Remember that no piece of apparatus will last for ever, especially if neglected. Hospitals using concentrators need to plan for regular maintenance, order a reserve of spare parts, and assign the task of looking after the machine to a named, trained individual. Make sure you buy an approved machine, (the list of approved machines is available from the Program for Health Technology, WHO, 1211 Geneva 27, Switzerland). It is

wise to choose from this list a machine whose manufacturer has a reliable local agent in your country.

The use of concentrators can bring both a substantial improvement in the availability of treatment and cost savings for the hospital. Even so it is still advisable to have the odd spare cylinder of oxygen available for emergencies. One 140 bed hospital in Nepal did just that when it installed concentrators in its operating theatres some 12 years ago; the 3 large reserve cylinders remain unopened - it would

be difficult to find a better recommendation for this technology!

## References

1. Dobson MB Oxygen concentrators for the smaller hospital—a review. *Tropical Doctor* 1992;22:56-8
2. Dobson MB Oxygen concentrators offer cost savings for developing countries. A study based on Papua New Guinea. *Anaesthesia*. 1991;46:217-9

## SELF ASSESSMENT QUESTIONS

*Dr Andrew Longmate, Edinburgh Royal Infirmary UK.*

The following MCQ questions assess your knowledge about issues covered in Update in Anaesthesia. Write your answers on a sheet of paper, and then check the answers and comments on page 65.

**1. A low alveolar PO<sub>2</sub> may be caused by**

- a) shivering
- b) breathing a hypoxic mixture of gases
- c) a decreased minute volume
- d) ventilation / perfusion mismatch
- e) uncomplicated cardiac failure

**2. Blood in the following vessels usually has an oxygen haemoglobin saturation greater than 90%**

- a) pulmonary artery
- b) aorta
- c) inferior vena cava
- d) pulmonary veins
- e) halfway along the pulmonary capillary

**3. Oxygen consumption**

- a) is increased in malignant hyperpyrexia
- b) is increased under general anaesthesia
- c) in health, is critically dependent on oxygen delivery
- d) is approximately 2L/min in the resting adult
- e) when increased, causes a decrease in the mixed venous PO<sub>2</sub> (assume oxygen delivery remains constant)

**4. Oxygen stores**

- a) are increased slightly by pre-oxygenation
- b) are large because oxygen is so important for cellular function
- c) depend in part on blood volume and haemoglobin concentration

- d) are large in pregnant women compared to non-pregnant women
- e) can be accurately assessed with a pulse oximeter

**5. The following commonly contribute to poor oxygenation during general anaesthesia**

- a) hyperventilation
- b) atelectasis
- c) ventilation / perfusion mismatch
- d) hypersensitive chemoreceptors
- e) increased metabolic rate

**6. 40% oxygen via a facemask is appropriate oxygen therapy for a patient with**

- a) a shunt equivalent to 40% of cardiac output due to pneumonia
- b) a reduced minute volume due to opioid analgesia
- c) complete upper airway obstruction
- d) hypovolaemic shock
- e) ischaemic heart disease following uncomplicated major surgery

**7. Pre-oxygenation**

- a) can be started in the ward prior to coming to theatre
- b) as part of a rapid sequence induction occurs following induction of anaesthesia but before intubation of the trachea
- c) causes a significant increase in the oxygen bound to haemoglobin in the blood
- d) should take place through an anaesthetic circuit and a high oxygen flow rate and the mask held just off the face
- e) allows for acceptable oxygenation during 10 minutes of apnoea

**8. The PO<sub>2</sub>**

- a) in the trachea while breathing air is about 150 mmHg

- b) in the arterial blood while breathing air is about 100 mmHg (13 kPa)
- c) in the alveoli can exceed 600 mmHg when breathing 100% oxygen
- d) of venous blood may fall when cardiac output is very low
- e) in the mitochondria in the brain is higher than in venous blood
- 9) ***After recent significant head injury with loss of consciousness and a period of decreased GCS, suitable anaesthetic techniques for fixation of fractured elbow include:***
- a) local anaesthetic block
- b) spontaneous ventilation with 2% halothane
- c) intravenous ketamine
- d) ventilation with a low concentration of whatever volatile agent is available to you (eg 0.5% halothane)
- e) avoidance of suxamethonium
- 10) ***Difficult intubation is associated with:***
- a) a short thick neck
- b) limited mouth opening
- c) dental abscess
- d) limited neck movements
- e) a Mallampati grade 1.
- 11) ***Strategies that can be used in patients likely to be a difficult intubation include:***
- a) regional anaesthesia
- b) inhalational induction and spontaneous ventilation with a volatile agent
- c) paralysis with alcuronium or pancuronium after intravenous thiopentone.
- d) awake intubation
- e) cautious intravenous anaesthesia using ketamine.
- 12) ***Interscalene block***
- a) is good for hand surgery
- b) is likely to work when paraesthesia or twitches occur over the shoulder.
- c) is useful for operations on the shoulder or upper arm
- d) can be used to reduce dislocated shoulder
- e) frequently blocks the phrenic nerve
- 13) ***A fit occurring in association with placement of a major local anaesthetic block***
- a) may be due to direct injection of local anaesthetic into a blood vessel
- b) may be because too big a dose of local anaesthetic has been given
- c) should be managed by giving oxygen and maintaining the airway
- d) if the fit does not stop may be treated with thiopentone or diazepam
- e) may be associated with cardiac arrhythmias or arrest
- 14) ***Pre-oxygenation in pregnant women is especially important because:***
- a) intubation may be difficult
- b) they have a reduced lung volume (decreased FRC-functional residual capacity)
- c) they use up oxygen faster than non pregnant patients
- d) can be achieved by breathing oxygen for 3 minutes with a tight fitting face mask.
- e) can be achieved by performing 3 vital capacity breaths of 100% oxygen
- 15) ***Agents which may be given at the time of induction of general anaesthesia for Caesarean section when there is pre-eclampsia include:***
- a) thiopentone
- b) etomidate
- c) magnesium sulphate
- d) alfentanil
- e) ketamine
- 16) ***A pregnant patient complains of difficulty breathing and tingling in the arms soon after a spinal with heavy bupivacaine for Caesarean section. Actions to perform include:***
- a) place the patient head down
- b) give oxygen and be ready to control the airway
- c) speed the iv infusion up
- d) place a pillow under the head and shoulders
- e) leave the room to continue scrubbing up for surgery.
- 17) ***Contraindications to the use of NSAIDs (non-steroidal anti-inflammatory drugs) for the control of pain include:***
- a) renal failure
- b) all asthmatic patients
- c) patients actively bleeding
- d) old and frail patients
- e) patients with peptic ulcer



**18) Concerning pain relief in children:**

- a) they have no need for pain relief
- b) morphine is too dangerous to give
- c) NSAIDs should not be used
- d) paracetamol is too weak to be useful
- e) local anaesthetic blocks are useful

**19) Features of an anaphylactic reaction****include:**

- a) low blood pressure
- b) diarrhoea
- c) wheezing
- d) flushed appearance
- e) swelling around the face and airway

**20) Treatment of an anaphylactic reaction****includes:**

- a) adrenaline
- b) steroids

- c) iv fluids
- d) antihistamine
- e) antibiotics

**21) Causes of a fast pulse (greater than 100)****include:**

- a) pain
- b) ketamine
- c) hypovolaemic shock
- d) atropine
- e) fever

**22) Causes of a slow pulse (less than 50 beats per minute) include:**

- a) hypoxia
- b) neostigmine
- c) cervical dilatation
- d) vasovagal episodes
- e) may be normal in very fit people

**EXTRACTS FROM THE JOURNALS**

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**Fluid Resuscitation With Colloid or Crystalloid Solutions**

Fluid resuscitation of critically ill patients is a subject of considerable debate. Currently there is much interest in the result of a recent meta-analysis which suggests that colloid therapy for hypovolaemia is associated with an increased risk of death when compared with crystalloid.<sup>(1)</sup>

The purpose of this paper was to identify all available unconfounded evidence of the effect on mortality in critically ill patients of colloid compared with crystalloids for volume replacement. The authors studied 37 trials of fluid resuscitation in critically ill patients but based their analysis on the 19 trials (with 1315 participants) which reported mortality.

The paper discusses that for decades there has been controversy over the relative benefits of colloid and crystalloid solutions for fluid resuscitation of hypovolaemic patients. In this review of randomised controlled trials the use of colloids compared with crystalloids was associated with an increase in absolute risk of mortality of

4%. There was no evidence that different types of injury necessitating fluid resuscitation had different findings. Although more expensive than crystalloids, use of colloids far exceeds current recommendations

Following publication of this meta-analysis considerable debate has been published from both supporters of crystalloid resuscitation and colloid users. It is difficult to know how to apply these findings at present.

Conditions in many parts of the developing world means that the choice of intravenous fluids is often limited. It is of interest that this analysis could show no benefit for the more expensive, and often fashionable, colloids.

Although there is still doubt about the optimal type of fluid replacement in hypovolaemic shock, it is well recognised that adequate volumes of intravenous fluid are required. Evidence for the safety of each type of fluid will need more work, particularly in the different groups of patients such as trauma, sepsis, anaphylaxis, cardiac etc and the different types of fluid available.

1. Schierhout G, Roberts I. Fluid resuscitation with colloid or crystalloid solutions in critically ill patients: a systematic review of randomised trials. *BMJ* 1998; 316:961-964.

### Pain experienced by infants

The study of pain in children has developed dramatically in the past 10 years. Although anaesthetists have been responsible for much of the research and the increase in public and professional awareness of the problem, surgeons, nurses and parents have also been the driving force for change in many places.

The key to successful prevention and treatment of pain in children rests in reliable measurement techniques. Facial expression scoring, cry duration and visual analogue scale scores are used as research tools for pain measurement. Facial expressions of pain following an injection are brow lowering, eyes closure, deepened nasolabial furrows and mouth opening. These signs are consistently seen from 2 to 18 months of age. (1)

The effect of neonatal circumcision on pain response during subsequent routine vaccination was studied by Anna Taddio and her colleagues from the Hospital of Sick Children in Toronto (2). Patients who had undergone neonatal circumcision showed a more marked pain response to subsequent routine vaccination than uncircumcised infants. The application of the topical local anaesthetic EMLA during circumcision reduced the pain

of circumcision but had little effect during tightening the clamp on the foreskin. However during subsequent vaccination there was a significant trend for EMLA treated infants to have an intermediate (compared with uncircumcised infants and circumcised without EMLA application) pain response across all the three (facial action, cry duration and V.A.S. scores) measurements of pain.

These papers add to the considerable evidence already available that even very small children experience pain which may be clinically detected and, to an extent quantified. Effective methods of controlling pain in this group of patients should be the subject of further research. Study of the vaccination pain response of infants who received more effective and more available circumcision pain management (like dorsal penile nerve block or caudal block) would be interesting.

1. Lilley CM, Craig KD, Grunau RE. The expression of pain in infants and toddlers: developmental changes in facial action. *Pain* 1997; 72:161-170

2. Taddio A, Katz J, Hersich AL, Koren G. Effect of neonatal circumcision on pain response during subsequent routine vaccination. *Lancet* 1997; 349:599-603

## ANKLE BLOCKS

*Dr B A McCormick, Southmead Hospital, Bristol.*

### Introduction

The ankle block is a safe and effective method for obtaining anaesthesia and analgesia of the foot for surgical procedures on bones and soft tissues.

### Indications

- ♦ Surgical anaesthesia of the foot especially when general, epidural or spinal anaesthesia is contra-indicated.
- ♦ For post-operative analgesia.

### Anatomy

Five nerve branches supply sensation to the foot. All are branches of the sciatic nerve, except the **saphenous nerve**, which is the terminal branch of the femoral nerve. The sciatic nerve divides into the tibial nerve and the common peroneal nerve at a variable point between the buttock and the popliteal fossa. The tibial nerve then divides into the **posterior tibial** and **sural nerves**, and the common peroneal nerve into the **deep** and **superficial peroneal**

**nerves**. The posterior tibial nerve finally divides into the **medial and lateral plantar nerves**.

Figure 1 shows the sensory distributions of these nerves. Of particular note;

- ♦ The posterior tibial nerve innervates all but one of the intrinsic muscles of the foot, via its terminal branches, the medial and lateral plantar nerves. Blockade of this nerve is important for surgery to deeper structures.
- ♦ The deep peroneal nerve innervates the first web-space and so must be blocked for anaesthesia of the great toe.
- ♦ Surgery is unusual in the territory of the sural nerve therefore it is not often blocked.

Figure 1 shows the anatomical relations of these five nerves. Note:

- ♦ The **posterior tibial nerve** lies immediately posterior to the posterior tibial artery, at the medial malleolus.

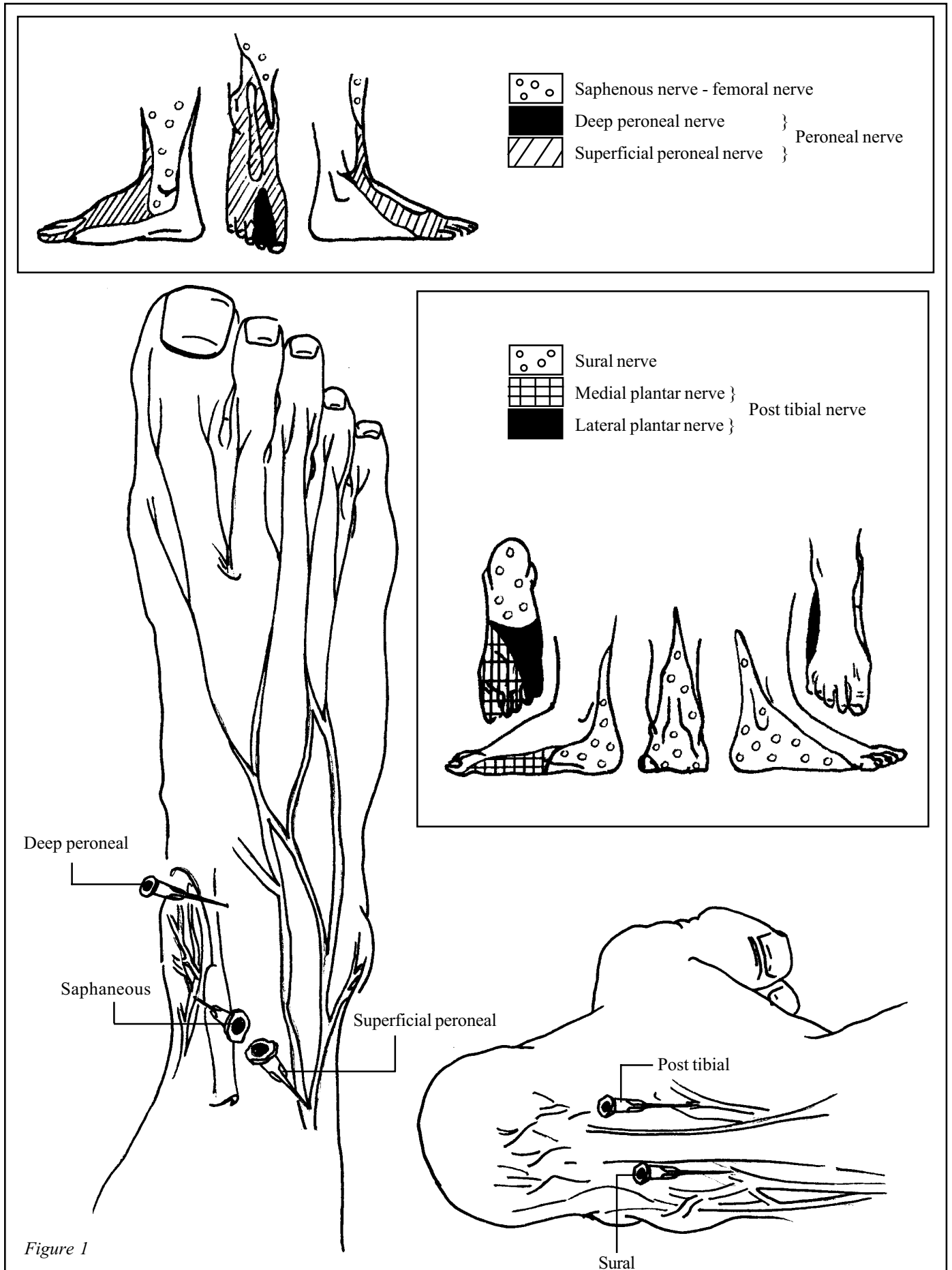


Figure 1

- ♦ The **superficial peroneal nerve** divides into terminal branches anterior to the ankle, necessitating a wide fan of infiltration for blockade.
- ♦ If paraesthesia is felt, inject 3-5ml LA. If not, advance to contact the tibia, withdraw 0.5cm and then inject 5-7ml LA.

### Preparation

1. Check resuscitation equipment and drugs.
2. Perform block in an anaesthetic or operating room.
3. Explain procedure to patient and obtain consent.
4. Establish IV access.
5. Full monitoring is advised where available (ECG, pulse oximetry, NIBP).

### Technique - General

- ♦ As performing the block can be painful, remember to inject the local anaesthetic slowly. Heating the local anaesthetic to body temperature may also help to reduce pain. Sedation may be required.
- ♦ All five nerves can be blocked with the patient supine and the foot on a padded support. Some prefer to block the **posterior tibial** and **sural nerves** with the patient prone. To block the posterior tibial nerve in a supine position, flex the knee and place the ankle on top of the contralateral shin. This allows easy access to the medial and lateral malleolus.
- ♦ As four of the nerves are almost entirely sensory an infiltration technique is used. Where available, nerve stimulation can be used to localise the posterior tibial nerve. Stimulation will produce movement of the big toe. A 23G needle, 4cm in length, is appropriate for all injections. It is important always to aspirate prior to injection of local anaesthetic, to exclude intravascular injection.
- ♦ The aim is sensory block alone and so low concentrations of local anaesthetic (LA) are sufficient (e.g. 0.25% bupivacaine) in most cases.

The five nerves are blocked by injections that form a ring of infiltration around the ankle at the level of the malleoli.

### Posterior tibial nerve

- ♦ Introduce the needle along the medial aspect of the Achilles tendon, at the level of the cephalic (towards head) border of the medial malleolus.
- ♦ Advance, in an anterior direction, towards the posterior border of the tibia (nerve lies just posterior to the posterior tibial artery).

### Sural nerve

- ♦ Introduce the needle along the lateral border of the Achilles tendon at the level of the cephalic border of the lateral malleolus.
- ♦ Advance anteriorly towards the fibula.
- ♦ If paraesthesia is felt inject 3-5ml LA. If not, inject 5-7ml LA as the needle is withdrawn. This gives subcutaneous infiltration from the Achilles tendon to the fibula.

Infiltration around the remaining three nerves can be performed from a single site. The needle is inserted 1cm lateral to the tendon of extensor hallucis longis (or just lateral to the anterior tibial artery, if palpable), at the level of the cephalic borders of the malleoli. This tendon is prominent on the dorsum of the foot, during extension of the big toe.

### Deep peroneal nerve

- ♦ From the position described above, advance the needle posteriorly (i.e. at 90° to the skin). Inject 3-5ml LA deep to the fascia, on either side of the anterior tibial artery.

### Superficial peroneal nerve

- ♦ After blocking the deep peroneal nerve, withdraw the needle to just stay in the skin.
- ♦ Turn the needle towards the lateral malleolus and inject 5ml LA in a subcutaneous band between the lateral malleolus and the anterior border of the tibia. This should reach all the branches of this nerve.

### Saphenous nerve

- ♦ Again withdraw the needle to just stay in the skin and turn the needle to point towards the medial malleolus.
- ♦ Infiltrate 5ml LA subcutaneously as the needle is advanced towards the medial malleolus. The great saphenous vein lies in this area, just antero-medial to the medial malleolus, in order to infiltrate around the vein, without causing damage, it may be necessary to make a further skin puncture lateral to the vein.

**Notes:**

- ◆ Check the block prior to surgery, testing sensation to pinprick.
- ◆ If anaesthesia is inadequate, identify which nerve supplies the relevant area and repeat the infiltration of that nerve.
- ◆ Within the limits of maximal local anaesthetic doses, advise the surgeon to infiltrate locally during surgery to augment the block.

**Cautions**

1. It is best to avoid adrenaline in the LA. There are theoretical risks to the foot from the vasoconstrictor effect.
2. Although systemic absorption from the subcutaneous tissues of the ankle is low, and toxicity is therefore unlikely, total recommended maximum total dose of local anaesthetic should not be exceeded.

**ANSWERS to MCQ questions on page 59.****1. TTTFF**

The PO<sub>2</sub> of alveolar gas is a balance between the oxygen supplied by breathing (decreased by low minute volume or hypoxic inspired mixture of gases) and the oxygen removed by the blood and used in metabolic processes in the body (increased by shivering). Ventilation / perfusion mismatch will cause a low arterial PO<sub>2</sub> as described in the text but the alveolar PO<sub>2</sub> will be unaffected. In cardiac failure pulmonary congestion may cause ventilation / perfusion mismatch but alveolar PO<sub>2</sub> does not fall unless the minute volume is reduced.

**2. FTFTT**

The transfer of oxygen from the alveoli to the blood is usually complete by the time the blood has passed a third of the way along the pulmonary capillary. Pulmonary veins take oxygenated blood back to the heart which is then pumped into the aorta. Deoxygenated blood returns to the heart from the lower half of the body via the inferior vena cava. It is then pumped from the right ventricle into the pulmonary arteries.

**3. TFFFT**

Approximately 250 ml of oxygen is consumed by the resting body per minute. Malignant hyperpyrexia increases the metabolic rate and therefore increases oxygen consumption. General anaesthesia causes a reduction in metabolic rate and therefore a reduction in oxygen consumption. In health, oxygen consumption is supply independent (see fig 3). Increased oxygen consumption causes more oxygen to be extracted from the arterial blood (increased extraction ratio) and therefore the venous PO<sub>2</sub> is lower.

**4. FTTF**

Normally the amount of oxygen in the body is only sufficient to sustain life for a few minutes. Oxygen stores are

increased dramatically by pre-oxygenation (see figure 4). They depend on blood volume and haemoglobin concentration and on the functional residual capacity (decreased in pregnant women) and the alveolar concentration of oxygen. A pulse oximeter only measures arterial haemoglobin saturation.

**5. FTTF**

Anaesthesia is commonly associated with hypoventilation. The chemoreceptor response to hypoxia and hypercapnia is reduced by anaesthesia. Uncomplicated general anaesthesia results in a reduction in metabolic rate.

**6. FTFFT**

A shunt equivalent to 40% of cardiac output is very large and would need more than 40% oxygen to correct the resultant hypoxia (see figure 6). Hypoxia due to hypoventilation is relatively easily corrected by increasing the inspired oxygen concentration but complete upper airway obstruction requires manoeuvres to clear the airway. Hypovolaemic shock requires 100% oxygen and fluid replacement. 40% oxygen would be suitable prophylactic therapy for a patient with ischaemic heart disease.

**7. FFFFF**

Preoxygenation involves three minutes breathing 100% oxygen through an anaesthetic circuit with the facemask firmly applied to the face. If performed as part of a rapid sequence induction it should occur before induction of anaesthesia and the increased store of oxygen in the functional residual capacity can result in a normal PO<sub>2</sub> for up to 8 minutes of apnoea.

**8. TTTT**

The PO<sub>2</sub> in the trachea while breathing air is about 150 mmHg and in the arterial blood is about 100 mmHg (13 kPa). Atmospheric pressure is 760 mmHg and when breathing 100% oxygen the only other gases in the alveoli are carbon dioxide (40 mmHg) and water vapour (47

mmHg). The PO<sub>2</sub> of oxygen could therefore be as high as  $760 - (40 + 47) = 673$ . When cardiac output is low, oxygen delivery is low and more oxygen is extracted from each unit of blood (high oxygen extraction). This causes a low venous PO<sub>2</sub>. The PO<sub>2</sub> in mitochondria is very low.

### 9. *TFFTF*

Remember that the risk of the patient having a surgically treatable intracranial haematoma must be considered. If this is possible then its investigation and or treatment will take precedence over less urgent surgery. However life saving surgery (e.g. to stop haemorrhage) takes priority over the head injury.

Local anaesthesia is ideal in this situation though spinal anaesthesia is contraindicated if there is any risk of raised intracranial pressure. Spontaneous ventilation and high concentrations of volatile agents are a bad mix after significant head injury and can lead to a rise in intracranial pressure and a worsening of the condition. Likewise ketamine will increase intracranial pressure and is contraindicated. Although many anaesthetic textbooks say that suxamethonium can increase intracranial pressure this is a minor point and its benefits in being a quick acting muscle relaxant allowing the anaesthetist to rapidly secure the airway far outweigh any theoretical disadvantage.

### 10. *TTTTF*

Answers a-d are all associated with difficult intubation, but a Mallampati 1 score is associated with an easy intubation.

### 11. *TFFTT*

Regional anaesthesia is an ideal way to avoid the difficulties of an awkward airway. Spontaneous ventilation allows for maintenance of the airway and anaesthesia can be maintained in this manner or an attempt at laryngoscopy and intubation can be made when the patient is deeply anaesthetised. Don't use long acting paralysis in patients who may be difficult to intubate. Ketamine can be used slowly and incrementally to induce and maintain anaesthesia allowing the patient to continue breathing and the airway to be maintained. This is a useful drug if you are inexperienced and unable to consider some of the other options.

### 12. *FFTTT*

The hand is better anaesthetised with axillary or supraclavicular block. Twitches or paraesthesiae should be felt in the arm or hand when placing the block as sensation over the shoulder are often due to superficial nerve stimulation. Paralysis of the diaphragm on the

ipsilateral (same) side means that this block may not be suitable for patients with very poor respiratory function.

### 13. *TTTTT*

Remember ABC (airway, breathing, circulation) in any emergency. Although local anaesthetics can cause cardiac arrhythmias and cardiac arrest, another important cause is hypoxia related to airway obstruction or stopping breathing (apnoea). Give oxygen and gently maintain the airway. You may need to ventilate the patient if they stop breathing. Place the patient in the lateral position so that aspiration is less likely should they regurgitate. Many fits are self terminating but if they last for longer than 1-2 minutes they can be terminated with drugs. Thiopentone usually stops fitting quite quickly - give just enough to stop the fit. Diazepam takes a minute or two to work - give 5-10 mgs iv initially. Both these drugs may cause respiratory depression so be ready to maintain the airway.

### 14. *TTTTT*

Preoxygenation is especially important in pregnancy for the reasons given in a-c. A vital capacity breath is from maximal expiration to maximal inspiration.

### 15. *TTTTF*

a & b are suitable agents, c & d can be used to reduce the pressor response. Do not use ketamine in pre-eclampsia as it may cause dangerous rises in blood pressure leading to stroke or heart failure. Spinal anaesthesia is perfectly acceptable in pre-eclampsia especially if this is a familiar technique for you.

### 16. *FTTTF*

The patient is developing a high spinal block. Tingling in the arms represents local anaesthetic spread to low cervical levels. The patient is at risk of apnoea (stopping breathing) if the anaesthetic spreads to higher cervical levels and blocks the nerve supply to the diaphragm. They are also likely to be hypotensive. Head down position encourages flow of the heavy bupivacaine towards the head and will cause total spinal. By raising the height of the head and shoulders you will prevent the local anaesthetic spreading further and possibly prevent a total spinal. The patient may need to be ventilated / intubated if they develop a total spinal.

### 17. *TFTTT*

NSAIDs worsen renal function and should not be used in renal failure and used with caution in the old and frail. Some asthmatic patients are made worse by NSAIDs but if they do not currently have wheeze and have taken aspirin uneventfully in the past it is reasonable to give them a

NSAID for pain. NSAIDs inhibit platelets and should not be given if problems controlling bleeding are anticipated.

### 18. *FFFFT*

Children have the same need for good analgesia as adults though their recovery times after surgery can be quicker. As well as being cruel to leave a child without pain relief, it makes management more difficult as a child in pain will be restless, thrashing about the cot and pulling out drips. A well analgesed child will be settled, breathing quietly and much easier to look after as well as being much happier. Paracetamol is a useful agent- give 20mg/kg as an initial dose then 15mg/kg 6 hourly. NSAIDs may also be usefully used. Morphine works well and 0.1-0.2mg/kg can be given im or pethidine 1-2 mg/kg. Local anaesthetic is very useful in children but be careful not to use toxic doses.

### 19. *TTTTT*

Any or all of these may occur after an anaphylactic reaction

### 20. *TTTTF*

After any suspected anaphylactic reaction the agent precipitating it should be discontinued. Adrenaline is the agent of choice to stop anaphylaxis. Apart from antibiotics the other agents are useful in the management of anaphylaxis

### 21. *TTTTT*

### 22. *TTTTT*

The most important thing to exclude and treat when faced with bradycardia is hypoxia. A slow pulse may be caused by a variety of surgical stimuli including cervical dilatation and traction on the eyeball. Unopposed neostigmine causes bradycardia which is why atropine should be given at the same time. Suxamethonium may also cause bradycardia especially when a second dose is given so atropine should also be given in this situation.

#### **Editorial Note:**

In Update in Anaesthesia No:9; 17-22 the authors for the article "The role of the Anaesthetist in the Management of Pre-eclampsia" should have been GJ Torr and MFM James, Department of Anaesthesia, University of Capetown, South Africa. Apologies for this omission.

## NEWS

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### **Plastic and Reconstructive Surgery Course – Accra, Ghana March 2000**

A training course designed for local surgeons working in developing countries and aims in one week to cover the relevant core areas of burns and plastic surgery. The speakers are international coming from UK, USA Sweden, Nigeria, Switzerland, Germany, Kenya, Malawi and Slovenia.

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**Venue:** Accra International Conference Centre, Accra, Ghana

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