



# UPDATE IN ANAESTHESIA



*A journal for anaesthetists in developing countries*

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## Editorial

Welcome to UPDATE! Many colleagues working in developing countries have few textbooks or journals. UPDATE in anaesthesia is a new add on journal being produced by "World Anaesthesia" for anyone administering anaesthetics in such difficult circumstances. The contributors and editors all have experience of working in developing countries; in fact many are working and teaching there at the present time. The first issue has been kindly sponsored by the World Federation of Societies of Anaesthesiologists and we are grateful to Mr Alex Morris and Mrs Carol Wilson for their help with the illustrations.

Initially the journal is being produced twice a year and will cover all aspects of anaesthesia including the basic sciences, general and regional anaesthesia and practical topics such as maintenance of equipment. It will be supplied free of charge to distributors within your countries, usually your National Society of Anaesthetists.

As editors we need to know if you would like to receive the journal, which organisation we can send it to in your country and what sort

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of subjects you would like us to cover. The more criticism and suggestions received, the better the journal will become. Please address letters to Dr Iain Wilson, Department of Anaesthetics, Royal Devon and Exeter Hospital (Wonford), Exeter EX2 5DW, Great Britain. We look forward to hearing from you!

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# INTRAVENOUS REGIONAL ANAESTHESIA (BIER'S BLOCK)

Dr W.F. Casey, Consultant Anaesthetist, Gloucestershire Royal Hospital, Gloucester.

**History.** This simple method of providing anaesthesia of the distal arm or leg was first described by August Bier in 1808. After a period of wide popularity, it fell into disuse until repopularised by Holmes in 1963.

**Clinical application.** Intravenous regional anaesthesia (IVRA) is indicated for any procedure on the arm below the elbow or leg below the knee that will be completed within 40-60 minutes. Onset of anaesthesia is rapid and reasonable muscle relaxation can be obtained. Its use is limited to procedures lasting less than an hour because of increasing discomfort from the tourniquet.

**Contra-indications.** The technique should not be employed if a tourniquet cannot safely be used, for example, in patients with severe Reynaud's or homozygous sickle cell disease. IVRA is not contraindicated in patients who are heterozygous for sickle cell disease. Caution should be employed in patients who have sustained crush injuries of the relevant limb as potentially viable tissue will be subjected to a further period of hypoxia.

**Equipment.** The only equipment necessary to perform this procedure successfully is a tourniquet which does not leak and that can be inflated to a pressure at least 50mmHg above the patient's systolic blood pressure, and a cannula inserted in a distal vein. As with all other local anaesthetic blocks, resuscitation equipment should be immediately available.

**Drugs.** The drug of choice for IVRA is prilocaine as it is the least toxic local anaesthetic and has the largest therapeutic index. If prilocaine is not available, lignocaine is an acceptable alternative. It is essential that plain and not adrenaline-containing solutions are used. Bupivacaine (Marcain) is not suitable and should never be employed as it is too toxic, particularly to the myocardium. A suitable dose to use in an arm is 40ml of 0.5% prilocaine (or 0.5% lignocaine). This can be increased to 50ml in muscular individuals or decreased to 30ml in small or frail patients. Larger volumes are necessary in the leg eg. 50-60ml. Maximum recommended volumes for a 60-70 kg patient are 400mg prilocaine (80ml 0.5% solution) or 250mg lignocaine (50ml 0.5% solution).

**Technique.** Before beginning to perform the block, the patient's blood pressure should be measured.

An intravenous cannula or butterfly needle is then inserted in a distal vein in the limb scheduled for surgery (eg. in the dorsum of the hand or foot). It is good practice to place a cannula in another limb as

well in case any complications (see below) occur, which may require intravenous drug administration. The tourniquet is then applied to the upper arm or thigh. It should never be placed on the forearm or lower leg as adequate arterial compression cannot then be obtained.

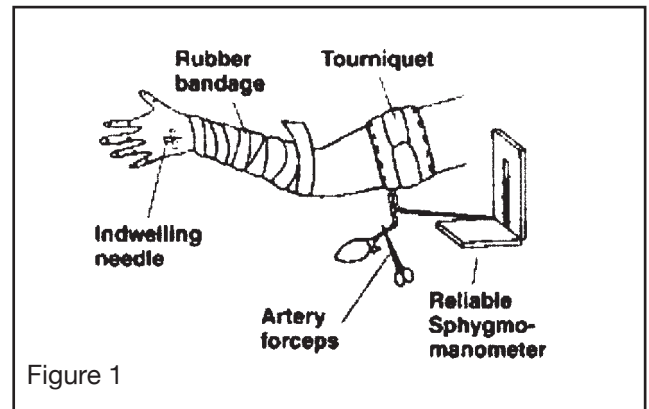


Figure 1

A more effective block is obtained if the limb is exsanguinated (blood removed) before the tourniquet is inflated. Traditionally, this is done by tightly wrapping the distal part of the limb with an Esmarch rubber bandage, before inflating the tourniquet. However, if this is likely to cause pain to the patient eg. if the limb is fractured, it is acceptable to simply elevate the arm or leg for 20-30 seconds whilst applying firm digital pressure on the brachial (or femoral) artery. This will allow venous blood to drain from the limb whilst preventing further arterial blood entering. The tourniquet is then inflated to a pressure of 50mm Hg or more above the patient's systolic blood pressure.

The local anaesthetic solution is then slowly injected into the indwelling cannula and the patient warned that the limb may start to feel hot and that the skin will take on a mottled appearance. Analgesia will occur within 3-4 minutes and surgery can then commence. Even if the surgery is completed within a few minutes, on no account should the tourniquet be deflated until at least 15 minutes has passed since the injection of the local anaesthetic or serious toxic side-effects may occur. The pressure in the tourniquet must be constantly observed and maintained at least 50mm Hg above the patient's systolic blood pressure.

If the operation is prolonged, the patient may complain of pain due to pressure from the tourniquet. This may be reduced either by the subcutaneous infiltration of a few mls of local anaesthetic above the tourniquet or by the use of a "double tourniquet technique." If this method is used, two tourniquets are placed on

the patient's arm or leg. Initially, the more proximal (upper) one is inflated and the local anaesthetic agent injected. If the patient becomes uncomfortable, the distal (lower) tourniquet is inflated and then the proximal one is deflated.

At the end of the procedure, the tourniquet is deflated and normal sensation quickly returns. It has been suggested that if the tourniquet is reinflated again 20-30 seconds, the rate of washout of local anaesthetic from the limb and hence the incidence of side-effects is decreased. In any event, the patient should be warned that they may experience tinnitus, dizziness or transient drowsiness following deflation of the tourniquet.

**Complications.** Intravenous Regional Anaesthesia is extremely safe and problems are few. The most important complications are due to the toxicity of local anaesthetics and will occur if the tourniquet suddenly deflates soon after the local anaesthetic has

been injected. These will range from dizziness and tinnitus to muscle twitching, loss of consciousness and convulsions. Serious cardiac side-effects are rare and occur if convulsions are inadequately treated or if bupivacaine is used.

Convulsions due to local anaesthetic toxicity are treated in the standard way: oxygen is administered, the airway is protected, by endotracheal intubation if necessary, and the convulsions terminated with intravenous diazepam or thiopentone. Deaths have been reported when convulsions have been inadequately or incorrectly managed.

**Conclusion.** IVRA is a simple and valuable technique that is easy to learn and perform. It is very safe provided excessive doses of local anaesthetic are avoided, if the tourniquet pressure is carefully monitored and if resuscitation equipment is always immediately available.

# DRAW-OVER ANAESTHESIA PART 1 - THEORY

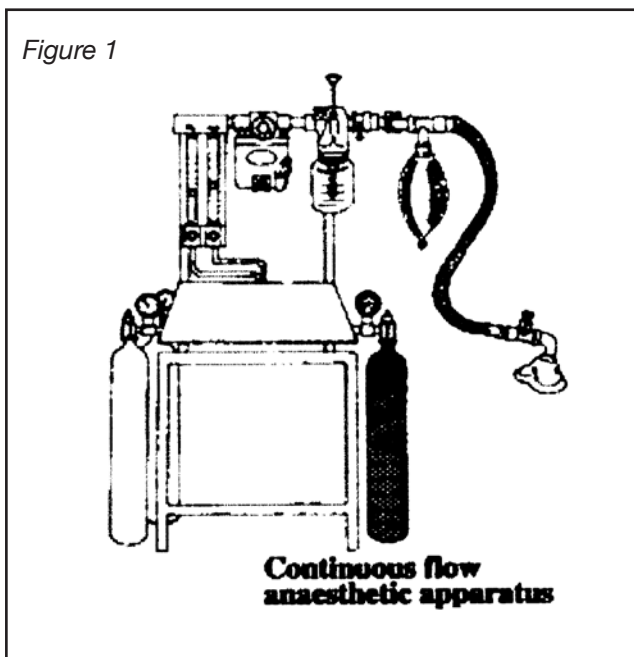
Mr G Kamm, Anaesthetist, Muchami Hospital, Tanzania and Dr IH Wilson, Consultant Anaesthetist.

## Introduction

In many parts of the world a regular supply of compressed anaesthetic gases cannot be maintained. Shortages of nitrous oxide are common and in some places oxygen is also unavailable. Anaesthetists working in such environments, whether in a developing country or in a disaster situation, may still be faced with patients requiring surgery necessitating techniques of anaesthesia not dependent on a supply of compressed gases. Suitable techniques include drawover anaesthesia, local anaesthesia and ketamine anaesthesia. This article considers the theory of drawover anaesthesia. Future editions of Update will contain articles covering the use of drawover systems for both adults and children, and care of the apparatus.

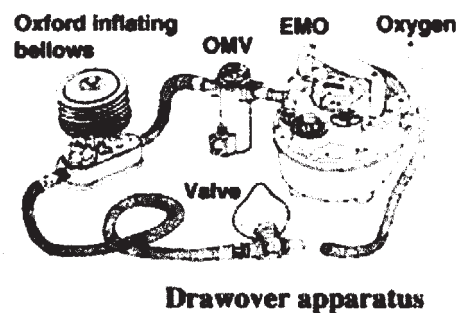
## How does drawover anaesthesia differ from anaesthesia given with a Boyles machine?

During anaesthesia using a Boyles machine (figure 1), compressed gases (oxygen and nitrous oxide or air) pass from cylinders mounted on the machine to rotameters, (a type of flow meter for gases), and then through the vaporizer where a volatile agent such as halothane is added to the gas mixture. The resulting mixture is delivered to the patient via an anaesthetic circuit, such as the Magill system. This type of anaesthesia system, known as "a continuous flow apparatus", is dependent on a supply of compressed gases. If these run out during an operation, so does the anaesthetic!



A drawover system (figure 2) is designed to provide anaesthesia without requiring a supply of compressed gases. Atmospheric air is used as the main carrier gas and is drawn by the patient's inspiratory effort through the vaporizer, where the volatile agent, normally ether or halothane, is added. The mixture is then inhaled by the patient via a non-rebreathing valve. The components of a drawover circuit are illustrated in figure 2.

Figure 2



## Features of drawover apparatus:

1. Robust, compact and portable
2. Low purchase price and running costs
3. Straightforward maintenance
4. Not dependent on compressed gases

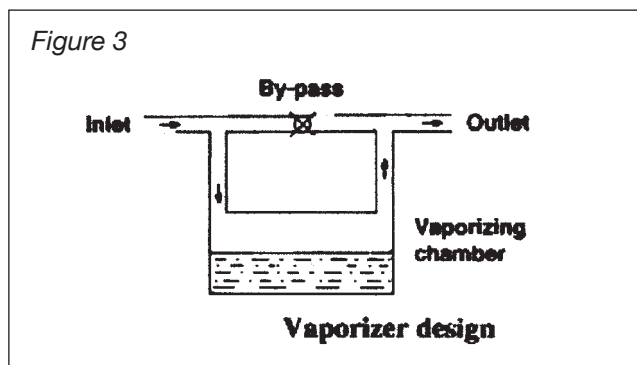
## Function of the components of a drawover system vaporizer

During drawover anaesthesia the patient moves, (or "draws"), air through the vaporizer which must have a very low resistance to the intermittent gas flow which is generated. The volume of air passing through the vaporizer is determined by the patient's tidal volume (the volume of air in a single breath) and the respiration rate. Considerable variations in flow through the vaporizer occur, depending on the type and depth of anaesthesia, the age of the patient and whether the patient is breathing spontaneously or being artificially ventilated. These conditions of gas flow require the drawover vaporizer to be specially designed.

Vaporizers designed for continuous flow anaesthesia should never be used in a drawover system as the high internal resistance to gas flow is too great. They are designed to work under a continuous high pressure and flow, and are called **plenum** vaporizers.

As air flows into the vaporizer it is directed either to the vaporizing chamber where it collects vapour from the

volatile agent being used, or into a bypass chamber which does not come into contact with the volatile agent (figure 3). The air from the two chambers mixes as it leaves the vaporizer.



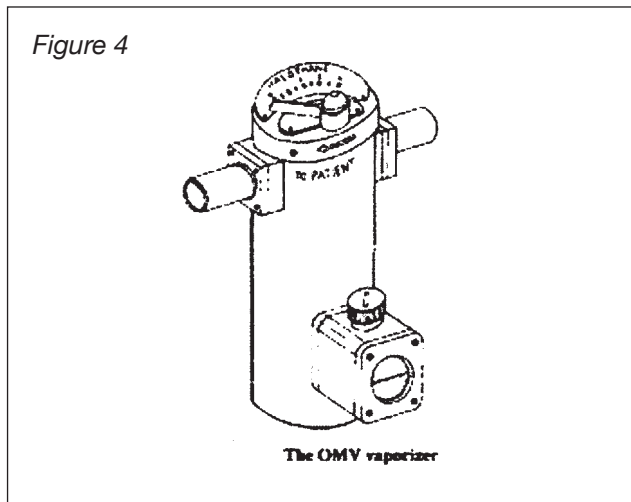
The ratio of air flow going to the different chambers determines the final concentration of volatile agent leaving the vaporizer, and is determined by the concentration control. The process of vaporisation removes heat from the volatile agent and vaporizer, due to the latent heat of vaporisation. This heat loss reduces the efficiency of vaporisation, and may result in a fall in concentration of volatile agent being delivered by the vaporizer. Some vaporizers compensate for cooling by a temperature operated valve which automatically increases the ratio of air directed through the vaporizing chamber as cooling occurs. Vaporizers with this facility are said to be thermo-compensated. Other vaporizers partially compensate for heat loss by containing a substance (such as water or copper) which delay changes in vaporizer temperature by providing a reservoir of heat. Vaporizers using this system are described as thermally buffered. Some vaporizers, such as the EMO, utilise both systems. All vaporizers require regular maintenance, but schedules vary both in frequency and complexity. Some models can be maintained by the anaesthetist, provided the essential tools are available, others require to be returned to the supplier for maintenance.

The most widely available drawover vaporizers are the EMO (Epstein, Macintosh, Oxford), OMV (Oxford Miniature vaporizer) and the TEC series (previously known as the PAC series). A few details of the vaporizers are set out below; an article in the next issue of Update will describe their use more fully.

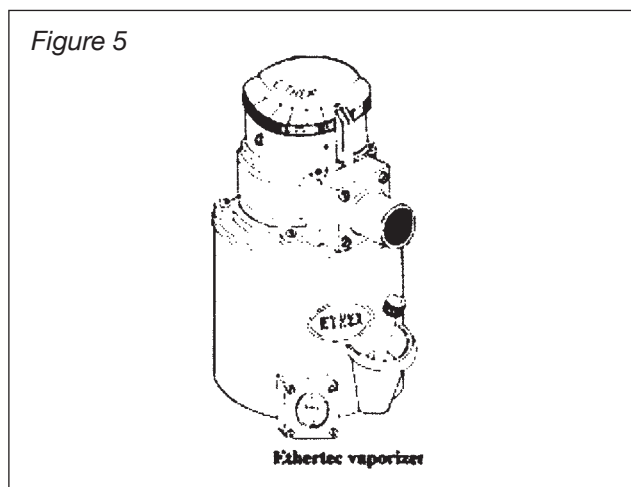
The **EMO** (figure 2) is a temperature compensated vaporizer which produces an accurate output of 0 to 20% ether. It is usually used in conjunction with the Oxford Inflating Bellows (OIB) which is incorporated as a part of the EMO system. Manufacturer Penlon (UK) Ltd.

The **OMV** (figure 4) is a small thermally buffered vaporizer which was originally produced to be used together with the EMO in order to speed the induction of anaesthesia. Original models contained only

20mls of volatile agent, more modern ones 50mls. A variety of volatile agents may be used with the OMV including halothane, trichlorethylene, enflurane, methoxyflurane and isoflurane. Different scales are available for each agent so that after draining the vaporizer the anaesthetist may use a different volatile agent. Manufacturer Penlon (UK) Ltd.



The **TEC or PAC** (figure 5) vaporizers consist of a range of thermo-compensated drawover vaporizers with different models available for ether, halothane, methoxyflurane and trichlorethylene. Manufacturer-Ohmeda (UK) Ltd.



**Self-inflating bags or bellows** allow controlled ventilation of the patient during anaesthesia or resuscitation. They should be the correct size for the patient to allow for an adequate tidal volume. Bellows and self-inflating bags incorporate a non-return valve through which they fill ensuring that fresh gas is always delivered to the patient. When there is an oxygen port on the bag or bellows this should be occluded, and oxygen added through a separate T piece (figure 2). Self-inflating bags and bellows are used with a non-rebreathing expiratory valve at the patient end to allow inspiration from the bag and expiration to atmosphere.

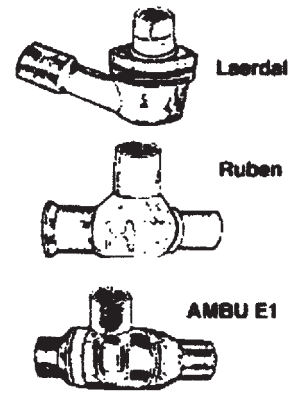
The Oxford inflating bellows (OIB) is popular with many anaesthetists using drawover anaesthesia. Unlike the self-inflating bags the OIB can be seen to move during spontaneous respiration. Two non-return flap valves are contained in the base of the OIB. The distal flap valve needs to be immobilised when the OIB is used with one of the non-rebreathing valves mentioned below. A magnet is supplied with the bellows for this purpose and its use will be fully described in a future article Practical drawover anaesthesia.

**Connecting tubing** should be of the antistatic type when ether is used and connections conform to the international standards of 22mm and 15mm tapered connections.

**Patient expiratory valve.** This should be a non-rebreathing valve such as an AMBU E1, Laerdal or Rubin's valve (figure 6). These valves allow either spontaneous or controlled respiration without adjustment. They need regular cleaning to prevent them becoming sticky and should be resterilised if used with a patient with chest infection.

**Oxygen T attachment.** To add oxygen to a drawover system a standard T piece is mounted on the intake

Figure 6



side of the vaporizer (figure 2). If it is mounted on the output side of the vaporizer a dilution of the volatile agent will occur. A reservoir tube (at least a metre in length) allows oxygen to accumulate during the expiratory phase. An oxygen flow of one litre/minute results in an inspired oxygen concentration of around 30-40% and a flow of four litres/minute a concentration of 60-80%.

# THE DIAGNOSIS AND TREATMENT OF HAEMORRHAGIC SHOCK

Dr IH Wilson, Consultant Anaesthetist and Dr PJF Baskett, President, Association of Anaesthetists of Great Britain & Ireland.

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Anaesthetists frequently care for patients in haemorrhagic shock, and must be capable of judging its severity. This article will discuss the assessment and clinical signs associated with hypovolaemia, and the management of the shock state.

## Pathophysiology

Shock produces a reduction in tissue perfusion resulting in hypoxic metabolism, acidosis and deterioration in organ function. The body responds to hypovolaemia through the sympathetic nervous system which causes tachycardia and vasoconstriction in an attempt to maintain cardiac output and blood pressure. To preserve blood flow to the vital organs (brain, heart, kidneys and liver), there is marked vasoconstriction of cutaneous and other peripheral blood vessels. Oliguria (defined as a urine output of less than 0.5ml/kg/ hour) occurs as the body actively retains fluid. The patient feels thirsty. As blood loss progresses there is increasing organ failure shown by dyspnoea (lungs), aggression or drowsiness (brain) and myocardial depression.

## Classification of blood loss

Haemorrhage may be classified according to the actual amount of blood lost, or as a percentage of the circulating blood volume.

The circulating blood volume may be estimated using the formula in figure 1.

<i>Figure 1. Calculating the circulating blood volume</i>
Circulating blood volume @ 70mls/kg
e.g. A 70kg man has: $70 \times 70 = 4,900\text{mls}$
e.g. A 20kg child has: $70 \times 20 = 1,400\text{mls}$

The clinical signs of shock in adults are listed in table 1 according to the amount of blood lost.

After haemorrhage the diastolic blood pressure changes before the systolic, due to active arterial vasoconstriction. The capillary refill test indicates the condition of the peripheral circulation. It is assessed by squeezing the finger nailbed and observing how long it takes for the circulation to return. Normally it is less than 2 seconds. Note that patients who are drowsy or unconscious due to shock have lost at least 2000mls, or 40% of their blood volume.

Young fit adults can vasoconstrict intensely in response to blood loss maintaining a relatively normal systolic blood pressure even after 1500-2000mls.

Therefore always assess the systolic blood pressure in conjunction with the other clinical signs of shock. Remember that during resuscitation with intravenous fluids, the restoration of systolic blood pressure does not mean that the blood volume has returned to normal-there may still be class 2 shock with severe volume depletion.

Not all clinical signs are present in every patient. For example elderly people may not develop a tachycardia, especially if they are taking a beta adrenergic blocker such as propranolol. Like patients with heart disease (ischaemic or valvular) they may become hypotensive after relatively little blood loss. In patients with pre-existing hypertension care must be taken when interpreting blood pressure. For example a systolic blood pressure of 110mmHg would be normal in a young person, but may reflect serious hypotension in the adult with hypertensive disease. Pain and cold may also produce some of the clinical signs of shock. Patients with extensive tissue damage lose a considerable amount of their circulating volume by oedema formation.

## Management of haemorrhagic shock

Remember the ABC of resuscitation. Check and correct any problems with the airway and breathing. Give oxygen in a high inspired concentration by face mask. Intubate patients who are unconscious. Control external haemorrhage by elevating the limb and by direct firm pressure with a clean pad over the bleeding site.

Insert a large cannula (14 gauge) into a suitable vein, use two when shock is worse than class one. When it is difficult to find veins, cannulate the external jugular or femoral vein or perform a cutdown at the ankle or antecubital fossa. In small children the intra-osseous route has been used with success. Do not use leg veins when intra-abdominal haemorrhage is suspected, or cannulate veins in an injured arm or shoulder. Take a sample for blood crossmatching when the first cannula is inserted.

## Choice of Intravenous Fluids

The choice of fluids will often be determined by what is available. There are 3 types of intravenous fluid: crystalloid, colloid and blood ( table 2). Dextrose 5% is not effective in the treatment of shock as it leaves the circulation rapidly. It should only be used as a last resort.

Crystalloids are distributed rapidly between the circulation and the extracellular (interstitial) fluid. When

Figure 1: Types of Intravenous Fluids used in Shock

	<b>Class 1</b>	<b>Class 2</b>	<b>Class 3</b>	<b>Class 4</b>
<b>Blood Loss</b> Volume (mls) in adult	750mls	800 - 1500mls	1500 - 2000 mls	>2000mls
<b>Blood loss</b> % circ. blood volume	<15%	15-30%	30-40%	>40%
<b>Systolic Blood Pressure</b>	No change	Normal	Reduced	Very low
<b>Diastolic Blood Pressure</b>	No change	Raised	Reduced	Very low / unrecordable
<b>Pulse</b> (beats/ min)	Slight tachycardia	100 - 120	120 (thready)	>120 (very thready)
<b>Capillary Refill</b>	Normal	Slow (>2s)	Slow (>2s)	Undetectable
<b>Respiratory Rate</b>	Normal	Normal	Raised(>20/min)	Raised (>20/min)
<b>Urine Flow</b> (mls/hr)	>30	20-30	10-20	0-10
<b>Extremities</b>	Normal	Pale	Pale	Pale & cold
<b>Complexion</b>	Normal	Pale	Pale	Ashen
<b>Mental State</b>	Alert, thirsty	Anxious, or agressive, thirsty	Anxious or aggressive or drowsy	Drowsy, confused or unconscious

treating shock give three times the estimated blood loss to allow for this. i.e. when replacing 1000mls blood loss give 3000mls of crystalloid.

Colloids remain within the circulation for a longer time (typically 4-8 hours) and should be administered in an equal volume to the blood loss.

Blood transfusion is required in previously healthy patients when estimated blood loss is greater than 30% of the circulating blood volume (1500mls in an adult). In previously anaemic patients transfusion is required with less severe haemorrhage. In grade 4 haemorrhage early transfusion with uncrossmatched blood is often necessary. After blood transfusions of 8 units or more coagulation factors may become deficient requiring fresh frozen plasma (if available).

Intravenous fluid replacement should be given to replace the estimated losses rapidly. Suitable fluid regimes for differing degrees of blood loss are shown in table 3. Many readers will not have access to colloids; the correct response is to give more crystalloid in a volume of three times the estimated loss, plus blood transfusion as described above. If facilities allow, warm the fluids (especially blood) using a blood warmer.

Monitor the patient's response to treatment by careful observation and recording of the clinical signs in table 1. Pass a urinary catheter and measure urine output to assist in your assessment. Aim for a urine flow of 0.5-1ml/kg/hour. Clinical improvement will be sustained if you have replaced the correct amount of

blood and the rate of haemorrhage is lessening. When the patient fails to respond appropriately, consider whether there are other sources of haemorrhage that you have not identified, or that haemorrhage is continuing unabated into the chest or abdomen or pelvis. A tension pneumothorax, pericardial tamponade or cardiac contusion can compound the signs of shock. In these situations measuring central venous pressure (normal 4-10cmH20) is helpful as it will indicate if pump failure is present.

Investigations may be needed to assist the diagnosis of injuries eg chest or other X-rays. A haemoglobin or haematocrit estimation helps in the decision for blood transfusion, which should maintain the haemoglobin around 8-10g/dl. However, if the sample is taken before resuscitation, a misleadingly high result may be obtained as haemodilution will not have occurred. Occasionally the serum potassium levels may be altered.

**Life saving surgery**

When there is severe haemorrhage (eg ruptured spleen or ectopic pregnancy) the patient may require an immediate operation to save life. Delaying surgery for prolonged resuscitation wastes resources and may be fatal.

Resources. In many parts of the world resources such as intravenous fluids or blood are in short supply. The sooner shock is treated, and the underlying cause diagnosed and managed, the better the outcome (and less resources consumed).

**Summary**

The key to successful management of haemorrhagic shock is awareness, identification and careful assessment of the problem and treatment with adequate fluid replacement. Attention to the airway and ventilation with oxygen is vital. Early precise diagnosis and definitive surgery should follow rapid resuscitation.

Figure 2: Treating Shock

Airway clear?	Be careful of suspected cervical spine injury
Breathing adequate?	Give oxygen and support ventilation
Circulation	Stop external bleeding I.V. Cannulation Cross match sample Resuscitate with IV fluids
Diagnose problem	Undress and expose
Establish	Treatment priorities for definitive care

Table 2: Types of Intravenous Fluids Used in Shock

Crystalloid	Ringers lactate Saline
Colloid	Gelofusine Haemaccel Dextran 70* Hetastarch Plasma or albumen solutions
Blood	Whole blood Packed cells Plasma reduced blood

\* Maximum of 1500mls/day is usually recommended for Dextran 70 as platelet function may be affected with larger amounts.

Table 3: Suitable Blood Replacement Regimes for Previously Healthy Adults

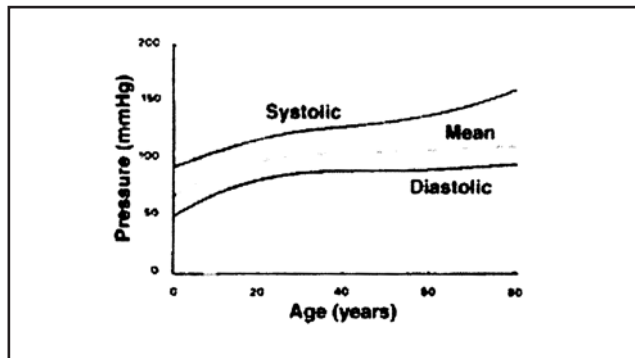
Estimated blood loss	Suitable fluid regime
1000 mls	3000 mls crystalloid or 1000mls colloid
1500 mls	1500mls crystalloid & 1000mls colloid or 4500mls crystalloid
2000mls	1000mls crystalloid, 1000mls colloid & 2 units blood or 3000mls crystalloid & 2 units blood

# CONTROL OF ARTERIAL BLOOD PRESSURE

Dr S. Sharma, formerly Lecture in Anaesthesia, Tribhuvan University Teaching Hospital, Kathmandu, Nepal; presently Plymouth General Hospital

The blood pressure is the force that causes blood to flow through the arteries, capillaries, and finally veins back to the heart.

It is closely regulated via several physiological mechanisms to ensure an adequate tissue blood flow. Both systolic and diastolic pressures increase with age (figure 1).



The blood pressure is determined by the rate of blood flow produced by the heart (cardiac output), and the resistance of the blood vessels to blood flow. This resistance is produced mainly in the arterioles and is known as the systemic vascular resistance (SVR) or the peripheral vascular resistance (PVR). The interactions between blood flow, pressure and SVR are shown in the equation in figure 2:

Figure 2

$$\text{Blood pressure} = \text{cardiac output} \times \text{SVR}$$

Using the formula in figure 2 we can see that the blood pressure can be raised either by increasing cardiac output or SVR. Conversely the blood pressure is reduced by a fall in cardiac output or SVR.

**Physiological mechanisms to maintain normal blood pressure** are listed below:

1. Autonomic nervous system responses
2. Capillary shift mechanism
3. Hormonal responses
4. Kidney and fluid balance mechanisms

**The autonomic nervous system** is the most rapidly responding regulator of blood pressure and receives continuous information from the baroreceptors (pressure sensitive nerve endings) situated in the carotid sinus and the aortic arch. This information is relayed to the brainstem to the vasomotor centre (VMC). A decrease in blood pressure causes activation of the sympathetic nervous system resulting in

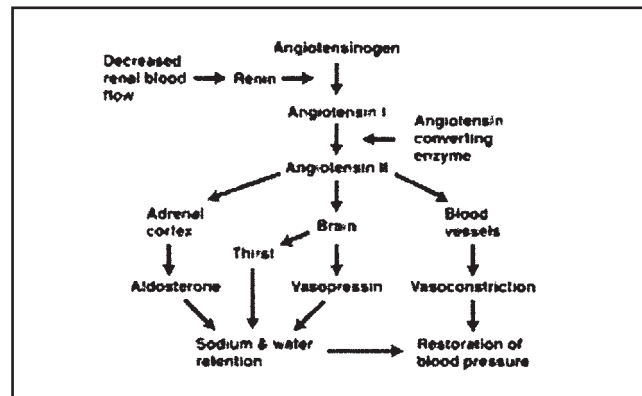
increased contractility of the heart (beta receptors) and vasoconstriction of both the arterial and venous side of the circulation (alpha receptors).

**The Capillary fluid shift mechanism** refers to the exchange of fluid that occurs across the capillary membrane between the blood and the interstitial fluid. This fluid movement is controlled by the capillary blood pressure, the interstitial fluid pressure and the colloid osmotic pressure of the plasma. Low blood pressure results in fluid moving from the interstitial space into the circulation helping to restore blood volume and blood pressure.

**Hormonal mechanisms** exist both for lowering and raising blood pressure. They act in various ways including vasoconstriction, vasodilation and alteration of blood volume. The principal hormones raising blood pressure are:

(a) Adrenaline and noradrenaline secreted from the adrenal medulla in response to sympathetic nervous system stimulation. They increase cardiac output and cause vasoconstriction and act very rapidly.

(b) Renin and angiotensin production is increased in



the kidney when stimulated by hypotension (figure 3). Angiotensin is converted in the lung to Angiotensin II, which is a potent vasoconstrictor. In addition these hormones stimulate the production of aldosterone from the adrenal cortex which decreases urinary fluid and electrolyte loss from the body.

This system is responsible for the long term maintenance of blood pressure but is also activated very rapidly in the presence of hypotension.

The kidneys help to regulate the blood pressure by increasing or decreasing the blood volume and also by the renin-angiotensin system described above. They are the most important organs for the longterm control of blood pressure.

In conclusion blood pressure is controlled by several physiological mechanisms acting in combination. They ensure that the pressure is maintained within

normal limits by adapting their responses both in the short and long term to provide an adequate perfusion to the body tissues.

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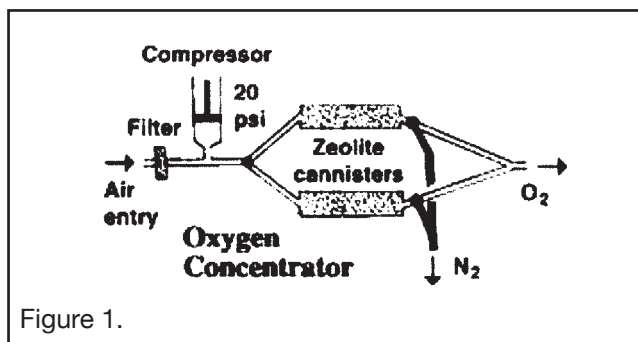
# THE OXYGEN CONCENTRATOR

Dr Roger Eltringham, Consultant Anaesthetist, Gloucestershire Royal Hospital, Gloucester.

A reliable source of oxygen is essential wherever anaesthetics are administered both to supplement the inspired gas mixture and also for resuscitation. It is traditionally supplied in cylinders which are both bulky and expensive. In isolated areas transportation of cylinders is difficult and may be unreliable, in military situations and disaster areas it may be dangerous or impossible. In many parts of the world the supply of oxygen may fail altogether leaving the anaesthetist with the unenviable task of providing anaesthesia for emergency surgery without access to oxygen, thus putting the patient at considerable risk of hypoxia and death.

Atmospheric air consists of approximately 80% nitrogen and 20% oxygen. An oxygen concentrator uses ambient air as a source of oxygen by separating these two components. It utilises the property of zeolite granules to selectively absorb nitrogen from compressed air.

Atmospheric air is entrained by the concentrator (Fig 1), filtered and raised to a pressure of 20 pounds per square inch (P.S.I.) by a compressor.



The compressed air is then introduced into one of the canisters containing zeolite granules where nitrogen is selectively absorbed leaving the residual oxygen available for patient use. After about 20 seconds the supply of compressed air is automatically diverted to the second canister where the process is repeated enabling the output of oxygen continue uninterrupted. While the pressure in the second canister is at 20 P.S.I. the pressure in the first canister is reduced to zero. This allows nitrogen to be released from the zeolite and returned into the atmosphere. The zeolite is then regenerated and ready for the next cycle. By alternating the pressure in the two canisters so that first one and then the other is at 20 P.S.I., a constant supply of oxygen is produced while the zeolite is continually being regenerated. Individual units have an output of up to five litres per minute with an oxygen

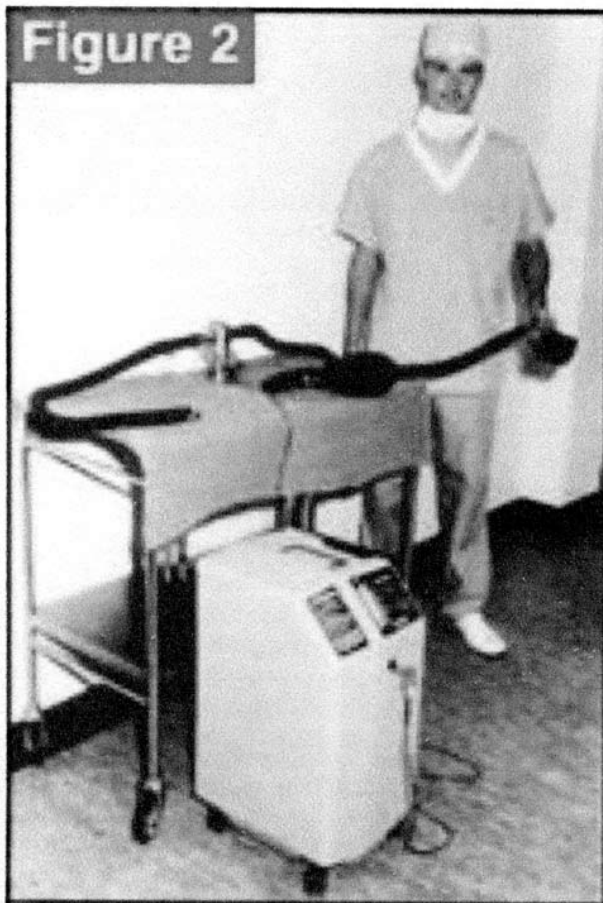
concentration of up to 95%.

Although this principle has been used on a large scale in units designed to supply entire hospitals with oxygen, interest has recently been focused on smaller units for individual patients. These have already proved their worth for domiciliary use and are now employed with great success in the wards, operating theatres and recovery units of isolated hospitals throughout the world.

The World Health Organisation has introduced minimal safety standards of performance under extreme conditions of temperature, humidity, vibration and atmospheric pollution. Manufacturers have been invited to submit units for testing and so far (September 1991) only the Puritan Bennett model 492A has successfully met all these standards. It is powered electrically from the mains or if this fails a small generator will suffice. The output is continually analysed and the user is alerted by an orange warning light on the front panel if the output concentration falls below 85%. If the oxygen concentrator falls below 70% a red warning light is illuminated indicating malfunction and the unit automatically shuts down. The concentrator is extremely easy to operate, the controls consisting simply of an on/off switch and a flow meter. A pressure alarm sounds when the unit is first turned on and for the next few seconds while the pressure is initially building up to 20 P.S.I. after which the alarm remains silent. It only sounds subsequently if the pressure falls: this usually means the filters need changing. The noise of the compressor is subdued and does not disturb even the most sensitive of surgeons.

Routine maintenance consists merely of changing the filters at regular intervals as directed by the manufacturers and this can be easily achieved using skills available locally. Providing these recommendations are observed the unit requires no other attention and will continue to function for many years.

As the output pressure from the concentrator is low it is not suitable for powering apparatus such as the Manley ventilator or for use with a standard Boyles anaesthetic machine. However it is extremely effective in supplementing air when using a drawover anaesthetic system such as the E.M.O. or Triservice apparatus (see figure 2) or when mechanically ventilating a patient using an electrically powered ventilator such as the East Radcliffe.



In these situations the oxygen is fed via a side-arm into the reservoir tube and the flow rate adjusted to the oxygen concentration required. During anaesthesia with a concentrator flow rate of one litre per minute the inspired oxygen concentration is approximately 35% and with a five litre flow an inspired oxygen concentration greater than 70% can be achieved. It is important that the oxygen is introduced upstream of the vaporizer (see figure 2 Drawover) and not downstream when it will dilute the inspired vapour

concentration. It is small and easily transportable and can also be used in the wards and in the recovery room for oxygen therapy and with incubators. A flow splitter is available to allow oxygen to be supplied to up to four separate sites simultaneously if required.

With its record of dependability, ease of maintenance and minimal running costs the initial purchase price of U.S. \$2,000 is an excellent investment and will rapidly lead to great savings of money. Even more important it will enable a source of oxygen to be constantly available and reduce anxiety for anaesthetists and risks to patients enabling anaesthesia to be given safely in situations where it might otherwise be extremely hazardous. Ideally no anaesthetist should be placed in a situation where he is expected to provide anaesthesia without access to a reliable source of oxygen. If oxygen is unavailable or supplies are unreliable, the anaesthetist should seek the help of his surgical colleagues and the hospital administrator as a matter of urgency, drawing attention to the advantages of oxygen concentrators.

Further information about the Puritan Bennett model can be obtained from Ian Chapman, Puritan Bennett, Heathrow Causeway, 152-176 Great South West Road, Hounslow, Middlesex TW4 6JS, U.K. Telephone 0181 577 1870.

A nationwide supply of oxygen dependent entirely on concentrators has successfully been set up in Malawi funded by the Danish Aid Organisation DANIDA. Advice on projects of this scale can be obtained from Dr John Pederson, Hostrups Have 5, Idh, DK 1954, Frederiksberg, Denmark. Telephone and fax 45-313 56694.

Information on other makes of oxygen concentrators can be obtained from Dr Michael Dobson, Nuffield Dept of Anaesthesia, John Radcliffe Hospital, Oxford, U.K.

# SUXAMETHONIUM

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**Preparation.** Suxamethonium is available as the chloride, bromide, or in some countries, as the iodide salt, and is dissolved in sterile water for injection. It is also known as succinylcholine. Suxamethonium chloride is supplied as a solution and deteriorates with storage, especially in hot countries, and should be stored in a refrigerator at 4° if possible. Suxamethonium bromide is supplied as a powder and dissolved in sterile water just before use, and can be stored for longer. All solutions of suxamethonium are destroyed by alkali, and should not be mixed with thiopentone.

**Uses.** Suxamethonium is a short acting muscle relaxant. The main uses in anaesthesia are to allow intubation of the trachea or to maintain relaxation for short surgical procedures.

**Main Effects.** Suxamethonium is a muscle relaxant and works by temporarily altering proteins in the muscle fibre membrane such that the muscle is not able to contract. The muscle relaxation may be preceded by brief irregular muscle contractions called fasciculations. The onset of muscle relaxation will be rapid after intravenous injection (30-60 seconds), and lasts 5-10 minutes. The muscle paralysis can be continued with intermittent intravenous boluses, using about 25% of the initial dose. The total dose should not exceed 6-8 mg/kg, or recovery may be very slow. The patient must be kept anaesthetised and the lungs artificially ventilated until recovery occurs.

In an emergency suxamethonium may be administered intramuscularly, but the onset of action is slower and less predictable than when given intravenously.

**Dose:**

Intravenous 1-2 mg/kg

Intramuscular 3 mg/kg

**Contraindications.** Suxamethonium is contraindicated in patients with recent burns or spinal cord trauma causing paraplegia (can be given immediately after the injury, but should be avoided from approximately day 10 to day 100 after the injury), raised potassium levels, severe muscle trauma, or a history of malignant hyperpyrexia.

Suxamethonium should be used with caution in patients with atypical plasma cholinesterase, or with muscle diseases. There may be prolonged paralysis or dangerous rises in potassium levels.

**Advantages.** The advantages of suxamethonium compared with non-depolarising muscle relaxants are the faster onset of relaxation, the greater degree of relaxation, and the short duration. It is suited for rapid sequence inductions where the trachea must be intubated as soon as possible.

## Adverse effects

**Cardiovascular:** suxamethonium can cause bradycardia (= slow heart rate), especially if a second or further doses are given. This can be prevented by the prior administration of atropine. Children develop this complication more commonly than adults.

**Metabolic:** the potassium level in the serum will rise by about 1 mmol/L in normal patients and by much more in patients with recent burns, paraplegias or severe muscle trauma.

**Raised intracranial and intraocular pressure:** there is a transient rise in intracranial and intraocular pressure after suxamethonium. This is of no importance in patients without eye or intracranial disease, but the drug should be avoided in patients with these conditions if possible.

**Prolonged paralysis:** this can occur in patients with abnormal plasma cholinesterases; if suxamethonium is given in excessive doses eg. by repeat injections or infusion; in patients having certain drugs eg. some antibiotics.

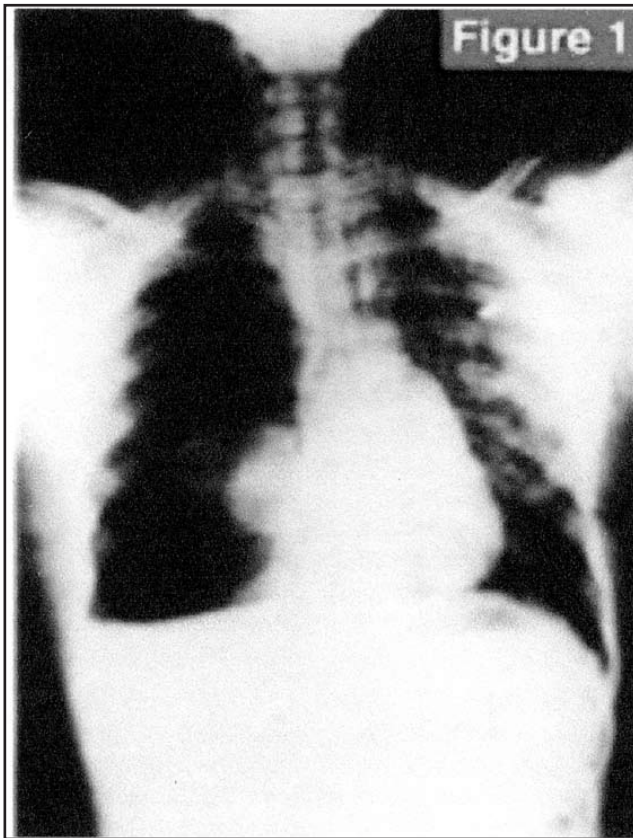
**Anaphylaxis:** suxamethonium can cause allergic reactions, which range in severity from minor flushing of the skin to cardiac arrest and severe bronchospasm.

**Malignant hyperthermia:** suxamethonium can trigger the onset of malignant hyperthermia in those patients who have this genetic muscle disorder.

**Muscle pains:** the fasciculations seen before the onset of muscle paralysis can cause muscle pain post-operatively, especially in young adults.

**Metabolism.** Suxamethonium is metabolised by an enzyme in the blood called plasma cholinesterase. Metabolism is normally complete within 5-10 minutes. Some patients lack this enzyme or have an altered enzyme that does not metabolise the suxamethonium as rapidly. These patients may remain paralysed for many hours after a standard dose of suxamethonium, and must be kept anaesthetised and ventilated until the suxamethonium has been eliminated by other slower methods.

## CLINICAL DILEMMAS



You are asked to anaesthetise a 30, year old man who has been involved in a road traffic accident. He requires a laparotomy for a suspected ruptured spleen. In your preoperative assessment you notice that he is dyspnoeic (breathless) and has reduced air entry on the right side. His chest X ray is shown in figure 1.

### Questions

1. Why is he dyspnoeic?
2. Why is his problem important to the anaesthetist?
3. What would you do about it?

### Answers

1. The patient is dyspnoeic because he has a tension pneumothorax. The X-ray shows the compressed outline of the right lung and an absence of normal lung markings in the right side of the chest. The mediastinum has been pushed over to the left. Air is also seen in the mediastinum and in the soft tissues of the neck and chest wall.

2. A tension pneumothorax makes normal respiration impossible and causes hypoxia. It also causes a shift of the mediastinum and a severe reduction in cardiac output and blood pressure due to impaired venous return to the heart. Under anaesthesia a pneumothorax may rapidly increase in size, particularly with the use of intermittent positive pressure ventilation or nitrous oxide (which difruses into the pleural space). A small asymptomatic pneumothorax may rapidly develop into a life threatening tension pneumothorax during anaesthesia.

3. Insert a chest drain with an under water seal prior to induction of anaesthesia.

### Comment

A pneumothorax may be diagnosed clinically by the signs of reduced air entry and an increased or “hyper-resonant” percussion sound on the affected side. It should always be suspected in patients who have had a chest injury and is best confirmed by a chest X-ray. It may be difficult to diagnose during anaesthesia, but if missed may rapidly progress and may be fatal. Always suspect the diagnosis in patients at risk of pneumothorax, and remember it may present in the anaesthetised patient as hypotension which is caused by the developing tension in the pleural space. Because of these difficulties most anaesthetists will insert a chest drain before induction in any patient with fractured ribs who requires anaesthesia. In an emergency situation where a chest drain is not readily available, a wide bore needle may be inserted into the pleural cavity to relieve a tension pneumothorax.

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