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EDITORIAL

This is the 5th edition of Update in Anaesthesia which is now sent to over 5000 anaesthetists in 100 countries. We are grateful for all your comments and suggestions and will print those of interest to other readers. This edition contains a questionnaire about your practice of anaesthesia. The answers to this will help us develop Update to be as practical as possible - we look foward to hearing from you.

Our aim is to produce an edition of Update twice a year. For a variety of reasons we have not managed to keep to this schedule, but the journal is produced as soon as a suitable mixture of articles is available. If you wish to receive the journal please contact Dr Ray Sinclair, Intensive Care Unit, Royal Cornwall Hospital, Treliske, Truro, Cornwall, TR1 3LJ, UK.

We have received requests for translations of Update in Anaesthesia into Russian, Spanish and French.

Contents: No 5

This is likely to be an extremely expensive exercise but we are currently investigating ways of achieving it so that Update can reach more people.

Dr Iain Wilson
Dr Roger Eltringham

PULSE OXIMETRY

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Pulse oximetry is a simple non-invasive method of monitoring the percentage of haemoglobin (Hb) which is saturated with oxygen. The pulse oximeter consists of a probe attached to the patient's finger or ear lobe which is linked to a computerised unit. The unit displays the percentage of Hb saturated with oxygen together with an audible signal for each pulse beat, a calculated heart rate and in some models, a graphical display of the blood flow past the probe. Audible alarms which can be programmed by the user are provided. An oximeter detects hypoxia before the patient becomes clinically cyanosed.

How does an oximeter work? A source of light originates from the probe at two wavelengths (650nm and 805nm). The light is partly absorbed by haemoglobin, by amounts which differ depending on whether it is saturated or desaturated with oxygen. By calculating the absorption at the two wavelengths the processor can compute the proportion of haemoglobin which is oxygenated. The oximeter is

dependant on a pulsatile flow and produces a graph of the quality of flow. Where flow is sluggish (eg hypovolaemia or vasoconstriction) the pulse oximeter may be unable to function. The computer within the oximeter is capable of distinguishing pulsatile flow from other more static signals (such as tissue or venous signals) to display only the arterial flow.

Calibration and Performance. Oximeters are calibrated during manufacture and automatically check their internal circuits when they are turned on. They are accurate in the range of oxygen saturations of 70 -100% (+/-2%), but less accurate under 70%. The pitch of the audible pulse signal falls with reducing values of saturation.

The size of the pulse wave (related to flow) is displayed graphically. Some models automatically increase the gain of the display when the flow decreases and in these the display may prove misleading. The alarms usually respond to a slow or fast pulse rate or an oxygen saturation below 90%. At this level there is a marked fall in PaO_2 representing serious hypoxia.

In the following situations the pulse oximeter readings may not be accurate.

1. A reduction in peripheral pulsatile blood flow produced by peripheral vasoconstriction (hypovolaemia, severe hypotension, cold, cardiac failure, some cardiac arrhythmias) or peripheral vascular disease. These result in an inadequate signal for analysis.

2. Venous congestion, particularly when caused by tricuspid regurgitation, may produce venous pulsations which may produce low readings with ear probes. Venous congestion of the limb may affect readings as can a badly positioned probe. When readings are lower than expected it is worth repositioning the probe. In general, however, if the waveform on the flow trace is good, then the reading will be accurate.

3. Bright overhead lights in theatre may cause the oximeter to be inaccurate, and the signal may be interrupted by surgical diathermy. Shivering may cause difficulties in picking up an adequate signal.

4. Pulse oximetry cannot distinguish between different forms of haemoglobin. Carboxyhaemoglobin (haemoglobin combined with carbon monoxide) is registered as 90% oxygenated haemoglobin and 10% desaturated haemoglobin - therefore the oximeter will overestimate the saturation. The presence of methaemoglobin will prevent the oximeter working accurately and the readings will tend towards 85%, regardless of the true saturation.

5. When methylene blue is used in surgery to the parathyroids or to treat methaemoglobinaemia a shortlived reduction in saturation estimations is registered.

6. Nail varnish may cause falsely low readings. However the units are not affected by jaundice, dark skin or anaemia.

Pulse oximeters may be used in a variety of situations but are of particular value for monitoring oxygenation and pulse rates throughout anaesthesia. They are also widely used during the recovery phase. The oxygen saturation should always be above 95%. In patients with long standing respiratory disease or those with cyanotic congenital heart disease readings may be lower and reflect the severity of the underlying disease.

In intensive care oximeters are used extensively during mechanical ventilation and frequently detect problems with oxygenation before they are noticed clinically. They are used as a guide for weaning from ventilation and also to help assess whether a patient's oxygen therapy is adequate. In some hospitals oximeters are used on the wards and in casualty departments. When patients are sedated for procedures such as endoscopy, oximetry has been shown to increase safety by alerting the staff to unexpected hypoxia.

Oximeters give no information about the level of CO_2 and therefore have limitations in the assessment of patients developing respiratory failure due to CO_2 retention. On rare occasions oximeters may develop faults and like all monitoring the reading should always be interpreted in association with the patient's clinical condition. Never ignore a reading which suggests the patient is becoming hypoxic. There is no

doubt that pulse oximetry is the greatest advance in patient monitoring for many years and it is hoped that their use will eventually become routine during anaesthesia and surgery world wide. Since pulse oximeters cost at least £1200 their purchase will depend mainly on economic considerations.

THE AUTONOMIC NERVOUS SYSTEM

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The nervous system is divided into the somatic nervous system which controls organs under voluntary control (mainly muscles) and the Autonomic Nervous System (ANS) which regulates individual organ function and homeostasis, and for the most part is not subject to voluntary control. It is also known as the visceral or automatic system.

The ANS is predominantly an efferent system transmitting impulses from the Central Nervous System (CNS) to peripheral organ systems. Its effects include control of heart rate and force of contraction, constriction and dilatation of blood vessels, contraction and relaxation of smooth muscle in various organs, visual accommodation, pupillary size and secretions from exocrine and endocrine glands. Autonomic nerves constitute all of the efferent fibres which leave the CNS, except for those which innervate skeletal muscle. There are some afferent autonomic fibres (i.e. transmit information from the periphery to the CNS) which are concerned with the mediation of visceral sensation and the regulation of vasomotor and respiratory reflexes, for example the baroreceptors and chemoreceptors in the carotid sinus and aortic arch which are important in the control of heart rate, blood pressure and respiratory activity. These afferent fibres are usually carried to the CNS by major autonomic nerves such as the vagus, splanchnic or pelvic nerves, although afferent pain fibres from blood vessels may be carried by somatic nerves.

The ANS is primarily involved in reflex arcs, involving an autonomic or somatic afferent limb, and then autonomic and somatic efferent limbs. For instance, afferent fibres may convey stimuli from pain receptors, or mechanoreceptors and chemoreceptors in the heart, lungs, gastrointestinal tract etc.

There may then be a reflex response to this involving autonomic efferent fibres causing contraction of smooth muscle in certain organs (e.g. blood vessels, eyes, lungs, bladder, gastrointestinal tract) and influencing the function of the heart and glands. The efferent limbs of these reflexes may also involve the somatic nervous system (e.g. coughing and vomiting). Simple reflexes are completed entirely within the organ concerned, whereas more complex reflexes are controlled by the higher autonomic centres in the CNS, principally the hypothalamus.

The ANS is divided into two separate divisions called the Parasympathetic and Sympathetic Systems, on the basis of anatomical and functional differences. Both of these systems consist of myelinated preganglionic fibres which make synaptic connections with unmyelinated postganglionic fibres, and it is these which then innervate the effector organ. These synapses usually occur in clusters called ganglia. Most organs are innervated by fibres from both divisions of the ANS, and the influence is usually opposing (e.g.the vagus slows the heart, whilst the sympathetic nerves increase its rate and contractility), although it may be parallel (e.g. the salivary glands). The responses of major effector organs to autonomic nerve impulses are summarised in Table 1.

Organ	Sympathetic stimulation	Parasympathetic stimulation
Heart	↑ heart rate β_1 (and β_2) ↑ force of contraction β_1 (and β_2) ↑ conduction velocity	↓ heart rate ↓ force of contraction ↓ conduction velocity
Arteries	Constriction (α_1) Dilatation (β_2)	Dilatation
Veins	Constriction (α_1) Dilatation (β_2)	
Lung	Bronchial muscle relaxation (β_2)	Bronchial muscle contraction ↑ bronchial gland secretions
Gastrointestinal tract	↓ motility (β_2) Contraction of sphincters (α)	↑ motility Relaxation of sphincters
Liver	Glycogenolysis (β_2 and α) Gluconeogenesis (β_2 and α) Lipolysis (β_2 and α)	glycogen synthesis
Kidney	Renin secretion (β_2)	
Bladder	Detrusor relaxation (β_2) Contraction of sphincter (α)	Detrusor contraction Relaxation of sphincter
Uterus	Contraction of pregnant uterus (α) Relaxation of pregnant and non-pregnant uterus (β_2)	
Eye	Dilates pupil (α)	Constricts pupil ↑ lacrimal gland secretions
Submandibular and parotid glands	Viscous salivary secretions (α)	Watery salivary secretions

PARASYMPATHETIC NERVOUS SYSTEM

The preganglionic outflow of the parasympathetic nervous system arises from the cell bodies of the motor nuclei of the cranial nerves III, VII, IX and X in the brain stem and from the second, third and fourth sacral segments of the spinal cord. It is therefore also known as the cranio-sacral outflow.

Preganglionic fibres run almost to the organ which is innervated, and synapse in ganglia close to or within that organ, giving rise to postganglionic fibres which then innervate the relevant tissue. The ganglion cells may be either well organised (e.g. myenteric plexus of the intestine) or diffuse (e.g. bladder, blood vessels).

The cranial nerves III, VII and IX affect the pupil and salivary gland secretion, whilst the vagus nerve (X) carries fibres to the heart, lungs, stomach, upper intestine and ureter. The sacral fibres form pelvic plexuses which innervate the distal colon, rectum, bladder and reproductive organs.

In physiological terms, the parasympathetic system is concerned with conservation and restoration of energy, as it causes a reduction in heart rate and blood pressure, and facilitates digestion and absorption of nutrients, and consequently the excretion of waste products.

The chemical transmitter at both pre and postganglionic synapses in the parasympathetic system is Acetylcholine (Ach). Ach is also the neurotransmitter at sympathetic preganglionic synapses, some sympathetic postganglionic synapses, the neuromuscular junction (somatic nervous system), and at some sites in the CNS. Nerve fibres that release Ach from their endings are described as

cholinergic fibres.

The synthesis of Ach occurs in the cytoplasm of nerve endings and is stored in vesicles in the presynaptic terminal. The arrival of a presynaptic action potential causes an influx of calcium ions and the release of the contents of several hundred vesicles into the synaptic cleft. The Ach then binds to specific receptors on the postsynaptic membrane and increases the membrane permeability to sodium, potassium and calcium ions, which results in an excitatory post-synaptic potential. The action of Ach is terminated by hydrolysis with the enzyme Acetyl Cholinesterase.

The specific Ach receptors have been subdivided pharmacologically by the actions of the alkaloids muscarine and nicotine. The actions of Ach at the preganglionic synapses in both the parasympathetic and sympathetic systems is mimicked by nicotine, and all autonomic ganglia are therefore termed nicotinic. Nicotinic transmission also occurs at the neuromuscular junction, in the CNS, the adrenal medulla and at some sympathetic postganglionic sites (see later). However, the actions of Ach at the parasympathetic postganglionic nerve ending is mimicked by muscarine. Muscarinic transmission also occurs at certain sites in the CNS.

SYMPATHETIC NERVOUS SYSTEM

The cell bodies of the sympathetic preganglionic fibres are in the lateral horns of the spinal segments T1-L2, the so called thoraco-lumbar outflow. The preganglionic fibres travel a short distance in the mixed spinal nerve, and then branch off as white rami (myelinated) to enter the sympathetic ganglia. These are mainly arranged in two paravertebral chains which lie anterolateral to the vertebral bodies and extend from the cervical to the sacral region. They are called the sympathetic ganglionic chains. The short preganglionic fibres which enter the chain make a synapse with a postsynaptic fibre either at the same dermatomal level, or at a higher or lower level, and then the longer postganglionic fibres usually return to the adjacent spinal nerve via grey rami (unmyelinated) and are conveyed to the effector organ.

Some preganglianic fibres do not synapse in the sympathetic chains but terminate in separate cervical or abdominal ganglia, or travel in the greater splanchnic nerve and directly synapse with chromaffin cells in the adrenal medulla. As discussed above, Ach is the neurotransmitter via a nicotinic receptor at the preganglionic synapse. The adrenal medulla is innervated by preganglionic fibres and therefore adrenaline is released from the gland by stimulation of nicotinic Ach receptors.

At most postganglionic sympathetic endings, the chemical transmitter is noradrenaline, which is present in the presynaptic terminal as well as in the adrenal medulla. In sweat glands, however, postganglionic sympathetic fibres release Ach and this transmission is nicotinic.

In contrast to the parasympathetic system, the sympathetic system enables the body to be prepared for fear, flight or fight. Sympathetic responses include an increase in heart rate, blood pressure and cardiac output, a diversion of blood flow from the skin and splanchnic vessels to those supplying skeletal muscle, increased pupil size, bronchiolar dilation, contraction of sphincters and metabolic changes such as the mobilisation of fat and glycogen.

Adrenaline and noradrenaline are both catecholamines, and are both synthesized from the essential amino acid phenylalanine by a series of steps, which includes the production of dopamine. The terminal branches of the sympathetic postganglionic fibres have varicosities or swellings, giving them the appearance of a string of beads. These swellings form the synaptic contact with the effector organ, and are also the site of synthesis and storage of noradrenaline. On the arrival of a nerve impulse, noradrenaline is released from granules in the presynaptic terminal into the synaptic cleft. The action of noradrenaline is terminated by diffusion from the site of action, re-uptake back into the presynaptic nerve ending where it is inactivated by the enzyme Monoamine Oxidase in mitochondria or metabolism locally by the enzyme Catechol-O-Methyl-Transferase.

The synthesis and storage of catecholamines in the adrenal medulla is similar to that of sympathetic postganglionic nerve endings, but due to the presence of an additional enzyme the majority of noradrenaline is converted to adrenaline. The adrenal medulla

responds to nervous impulses in the sympathetic cholinergic preganglionic fibres by transforming the neural impulses into hormonal secretion. In situations involving physical or psychological stress, much larger quantities are released.

The actions of catecholamines are mediated by specific postsynaptic cell surface receptors. Pharmacological subdivision of these receptors into two groups (α and β) was first suggested by Ahlquist in 1948, based upon the effects of adrenaline at peripheral sympathetic sites. These have since been further subdivided on functional and anatomical grounds. Thus β_1 adrenoceptor mediated effects in the heart (increased force and rate of contraction) have been differentiated from those producing smooth muscle relaxation in the bronchi and blood vessels (β_2 effects). Similarly, α–adrenoceptor mediated effects such as vasoconstriction have been termed, α_1 effects, to differentiate them from the feedback inhibition by noradrenaline on its own release from presynaptic terminals, which is mediated by α_2 adrenoceptors on the presynaptic membrane.

However, further research now shows that the classification is not as simple as this. For instance, many organs have both β_1 and β_2 adrenoceptors. (e.g. in the heart, there is one β_2 adrenoceptor to every three β_1 adrenoceptors). The receptors also show differing responses to adrenaline and noradrenaline. At β_1 adrenoceptors in the heart, adrenaline and noradrenaline appear to have an equal effect, whereas at β_2 adrenoceptors in smooth muscle are more sensitive to circulating adrenaline than noradrenaline.

AXILLARY BRACHIAL PLEXUS BLOCK

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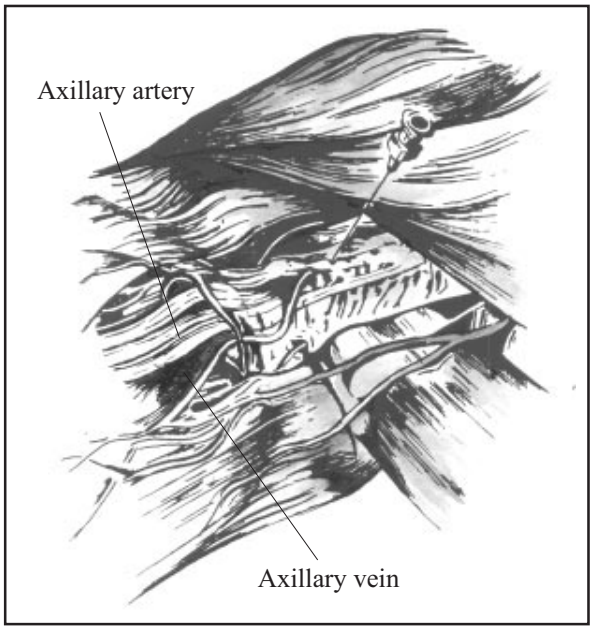
An axillary block is the most commonly performed variety of brachial plexus block. The landmarks are easy to identify and it is associated with fewer complications than other approaches to the brachial plexus. The technique may be used to provide anaesthesia for a variety of surgical procedures on the hand and forearm. Although in some patients the block may extend above the elbow it does not do this reliably.

Anatomy

The brachial plexus is derived from the cervical roots C5, C6, C7, C8 and the thoracic root T1. The plexus runs from the neck to the axilla passing between the clavicle and the first rib. In the axilla the plexus forms 3 cords which surround the axillary artery - the posterior, lateral and medial cords. The cords form the nerves to the arm - the median, ulnar, radial and the musculocutaneous nerve. The musculocutaneous nerve arises first and is often incompletely anaesthetised during an axillary block. Alongside the axillary artery runs the axillary vein. The vessels and nerves are contained in a connective tissue sheath (figure 1).



Technique

After checking your resuscitation equipment insert an intravenous cannula in the other arm in case of emergencies. With the patient lying supine the arm is abducted to about 90 degrees, externally rotated and flexed at the elbow. A pillow should be used to ensure that the arm is in a relaxed position. Prepare the axilla using a skin sterilising solution, shaving is not necessary. Palpate the axillary artery and place a finger on it as high in the axilla as possible. Raise a skin weal superficial to the artery with 1% lignocaine. Advance the needle slowly at about 30

degrees to the skin through the weal towards the side of the artery. The needle will enter the sheath which is sometimes felt as a click. The click is best felt when a short bevelled needle is used. Correct placement in the sheath is confirmed if the needle gently pulsates indicating close proximity to the artery or if the patient complains of parasthesia (a feeling of "pins and needles" caused by the needle touching the nerve). Aspirate to exclude intravascular placement of the needle and then inject the local anaesthetic solution. During the injection aspirate again to ensure that the needle has not changed position and entered a vessel. The injection is most conveniently performed with an extension set between the syringe and the needle. This allows the syringe to be changed without moving the needle. Some anaesthetists place a cannula into the sheath to allow further top ups of local anaesthesia. If firm pressure is placed over the sheath below the point of injection the local anaesthetic solution is encouraged towards the axilla and a better block will result.

Allow plenty of time for the block to develop (15 - 30 minutes). If it is planned to use a tourniquet during surgery some subcutaneous infiltration of local anaesthetic on the medial side of the upper arm will often improve the comfort of the patient.

Volumes of local anaesthetic

This will depend on the size of the patient and the drugs available. The following doses are suggested:

Adult: 30-40ml
1% lignocaine with adrenaline 1:200,000

Teenagers: 40-60kg 20-25ml
1% lignocaine with adrenaline 1:200,000

Children: 25-35kg 14-20ml
1% lignocaine with adrenaline 1:200,000

An alternative is to use 1% prilocaine or 0.25% bupivacaine.

Contraindications

Damage or disease of the brachial plexus. Sepsis in the axilla. Allergy to local anaesthetic.

Complications

Arterial puncture. If this occurs slowly withdraw the needle until blood cannot be aspirated - the needle now lies in the sheath. Slowly inject the local anaesthetic. Check the needle position by aspirating twice during the injection.

Venous puncture. Withdraw the needle, put pressure over the injection site for 5 minutes and then start again.

USING VOLATILE ANAESTHETIC AGENTS

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There are a variety of anaesthetic techniques available and various clinical situations demand different techniques of anaesthesia. A trained anaesthetist should be able to decide the most appropriate method for each case as an anaesthetic decision (not a surgical decision) and have the necessary skills to use the technique of choice. Anaesthetists should practise their skills as widely as possible and not give the same anaesthetic to every single patient. This is of particular importance where supplies of drugs and equipment are unreliable.

Volatile anaesthetic agents are commonly used and have an important safety feature in that agents which enter the circulation via the lungs can leave by the same route. Therefore the concentration of anaesthetic at the brain can be rapidly reduced as long as the patient is breathing adequately.

Basic pharmacology of volatile agents

An agent inhaled into the lungs will first enter the circulation and is then carried to all tissues of the body. We are primarily interested in the concentration reaching the brain because this produces the state of anaesthesia. The exact mechanism of anaesthesia is poorly understood but it seems that the nerve cells absorb the agent and in so doing their ability to

conduct impulses to each other is reduced.

The more soluble the agent is in blood the longer it takes to build up an effective concentration in the brain and the slower the onset of unconsciousness. Thus with a very soluble agent such as ether, the induction of anaesthesia is prolonged. On the other hand an agent such as nitrous oxide is relatively insoluble in blood; the blood becomes saturated quickly, the brain concentration rises quickly and the effect is seen rapidly. The degree of solubility of an agent in blood is indicated by its blood gas solubility coefficient (see table 1).

The comparative potency of volatile agents depends on the minimum alveolar concentration (MAC). This has been defined as the concentration of the anaesthetic required in the alveoli to produce in surgical anaesthesia in 50% of patients. Although this is largely a theoretic value it allows us to compare the potencies of different anaesthetic agents. An agent with a low MAC value is more potent than one with a high MAC value. Trichloroethylene, like ether, is very soluble yet it produces its effects at a fraction of the concentration of that needed for ether. This is because the MAC for trichloroethylene is 0.17% and for ether 1.92%. The physical properties of the different agents are listed in table 1.

The inhalation agents that are commonly used in Africa and other places where resources are limited are ether and halothane. When it is available, trichloroethylene is also used.

Patterns of anaesthesia differ from country to country. Halothane, once the mainstay of modern inhalation anaesthesia and an agent that we in Malawi still regard as a "luxury" anaesthetic is now hardly used in the USA. In adult anaesthesia in the UK it has been largely displaced by two newer agents, enflurane ("Ethrane") and Isoflurane ("Forane") both of which are far more costly than halothane. Ether, of course, is never used in the western world and trichloroethylene has a diminishing number of users worldwide as production has now virtually ceased.

ETHER (Diethyl ether)

This is an inexpensive agent made from sugar cane (ethanol). Ether has been known since the 16th century as "sweet vitriol" but only when W.T.G.Morton demonstrated its effects in Boston in 1846 did its anaesthetic properties become known worldwide. This "first anaesthetic" took place on 16th October 1846.

Ether is stored in dark bottles with corks/caps as light may decompose it. If it is taken to high altitude its boiling point is lowered (for example where atmospheric pressure is 425 mmHg ether will boil at 20^{o}C).

Advantages: Ether stimulates respiration and blood flow due to its sympathomimetic effect mediated by adrenaline release. When too much ether is given respiration becomes depressed before the heart. These effects make ether a "safe" anaesthetic agent. It is a bronchodilator and produces analgesia. It may be used as the sole anaesthetic agent and is capable of producing good abdominal muscle relaxation. Ether causes little uterine relaxation.

Disadvantages: Ether is associated with a slow onset and a slow recovery. It stimulates salivation

Table 1. Physical characteristics of the volatile agents.

Characteristics	Ether	Halothane	Trichloroethylene	Nitrous oxide
MAC	1.92%	0.77%	0.17%	101%
Blood/gas coefficients	12.1	2.3	9.15	0.47
Oil/gas coefficient	65	224	960	1.4
Boiling point	35^{o}C	50^{o}C	87^{o}C	-89^{o}C

and is best used with atropine premedication. The vapour is unpleasant to breathe initially and causes irritation of the bronchial tree which may slow down the induction of anaesthesia. The incidence of nausea and vomiting is higher with ether than with other agents. The frequency is related to the concentration of ether used and is lower when ether is given via an endotracheal tube during relaxant anaesthesia.

Indications: Any general anaesthetic. It is especially useful for caesarean section (because the baby tolerates it and the uterus contracts well), major operations requiring intubation and poor risk cases (using a low dose). It is the volatile agent of choice when general anaesthesia is needed but no oxygen is available.

Contraindications: There are no absolute contra-indications to ether. It is better avoided in moderate or severe pre-eclampsia because of its sympathomimetic activity. Likewise, liver or renal failure and phaeochromocytoma are relative contra-indications.

Ether is explosive when mixed with oxygen and is inflammable in air. It may be ignited by a flame or an electrical spark such as those produced by diathermy or static electricity. The ether vapour is inflammable within the patient (lungs, airway or stomach full of vapour) or outside the patient within 25cm of the anaesthetic circuit. Scavenging must always be carried out (where possible) to avoid contact between heavy inflammable ether vapour and diathermy apparatus or other electrical devices that may spark. If the end of the scavenging tube is placed on the floor (away from any possible sources of ignition) then the heavy ether vapour will remain at floor level and the smell of the agent to the surgical and anaesthetic team reduced.

If in doubt about the safety of ether with diathermy, don't use them together.

Dosage and technique: The easiest method is to give ether to the patient after they have been intubated following atropine, thiopentone and suxamethonium. IPPV is commenced initially with 10 - 15% ether and then according to the patient's requirements, the ether is cut back after 2-8 minutes to 4-8% (usually 6-8%). Poor risk, septic or shocked patients can be kept just insensible with 2% ether. Discontinue the ether well before the end of the operation to avoid a prolonged recovery. With skill you can have your patients almost awake as they are moved off the table. If the patient is given a long acting muscle relaxant and the ventilation controlled, then the ether may be reduced to around 3 - 4%.

Vaporisers: The small Ether TEC vaporiser will deliver a lower dose than the EMO for the same setting. When using the EMO vaporiser do not give 15-20% ether for more than a few minutes because it is a very efficient vaporiser and will rapidly overdose the patient.

Practical points: If you are not used to giving ether, you will be surprised at its slow onset of action. Don't let the surgeon start until the patient is really deep. People working in theatre may complain about the smell of ether and nurses may claim that it gives them headaches. Symptoms improve after a while and may be reduced by efficient scavenging.

HALOTHANE (Fluothane)

Halothane contains thymol as a stabilizing agent and is stored in dark bottles as it is decomposed by ultraviolet light. It is more expensive than ether (about US $25.00 per bottle compared to US $3.00 for three times the volume of ether).

Advantages: Halothane is a well tolerated, non-irritant potent agent giving rapid induction, low dose maintenance and rapid recovery. There is a predictable, dose-related depression of respiration and cardiac function.

Disadvantages: As halothane is a very potent agent it is not suitable for use by untrained anaesthetic staff. Its poor analgesic properties necessitate deep planes of anaesthesia before surgery can be tolerated. For this reason it is generally not suitable as a sole agent without an analgesic supplement, eg nitrous oxide, trichloroethylene, local anaesthetic block or other analgesic, especially during spontaneous respiration. It provides no post-operative analgesia, and causes uterine relaxation and haemorrhage particularly if greater than 0.5% halothane is used. The depression of the cardiovascular system may cause bradycardia, hypotension and a reduction in cardiac output. These

effects may be marked in children who should receive atropine either as premedication or i.v. at induction if halothane anaesthesia is planned. The combined depressant effects on the circulation and respiration mean that during anaesthesia and the early recovery phase supplemental oxygen should always be given.

Halothane sensitises the heart to adrenaline and predisposes the patient to developing arrhythmias. These arrhythmias occur most commonly in patients who are retaining CO_2 or who have an inadequate analgesic component in their anaesthetic. They can usually be managed by supporting the ventilation, reducing the amount of halothane in the inspired gases and supplementing with another analgesic e.g. a small dose of i.v. opiate (be sure to support respiration due to the extra respiratory depression that may result). If this is not effective i.v. lignocaine or propranolol (avoid in asthma) are generally effective. Injection of adrenaline by the surgeon during anaesthesia with halothane may be dangerous and the doses need to be carefully monitored. The surgeon should never inject more than 10mls of 1:100,000 in any ten minute period and never exceed 30mls of 1:100,000 in an hour. If possible avoid adrenaline altogether; if it has to be used monitor the pulse closely and support ventilation.

Postoperative shivering may occur which is usually shortlived. "Halothane hepatitis" occurs on very rare occasions, and is almost unheard of in children. In common with all agents being considered in this article halothane causes an increase in intracranial blood flow and therefore pressure.

Indications: almost all general anaesthesia. Inhalation induction especially in upper airway problems such as partial obstruction.

Contraindications: These include simultaneous administration with adrenaline, especially during spontaneous breathing, or a history of hepatitis following a previous anaesthetic. Avoid high doses during caesarean section or evacuation of retained products of conception or placentae as uterine bleeding may result.

Dosage: Induction requires inspired concentrations of up to 3%. Maintenance dose is 1-2% for spontaneously breathing patients and 0.5 - 1% during controlled ventilation. Great care should be taken to avoid an overdose which may occur easily with higher doses.

Vaporisers: Halothane should always be given through a calibrated vaporiser. When using an Oxford Miniature Vaporiser (OMV) the thymol in halothane may cause the pointer to stick. This can be remedied either by washing the vaporiser through with ether or stripping and cleaning if you have the facilities.

Practical points: Halothane alone is not ideal: because it has no analgesic properties and high concentrations are needed to abolish reflex activity. This becomes expensive and may also be unsafe. The combination of high concentrations of halothane, oxygen and air, high levels of carbon dioxide (from respiratory depression) and heart disease is potentially very hazardous for the patient, especially if the pulse is not adequately monitored for arrhythmias. I would never use this method, though many do and get away with it, probably because heart disease is so uncommon in Africa.

Inhalation induction starting with 3% seems to be well tolerated by all patients and stage two effects are minimised with this dose.

A common arrangement is to have two draw-over vaporisers in series containing halothane and trichloroethylene, with the halothane nearest the patient.

Supplementary oxygen is mandatory when using halothane to avoid hypoxia.

In a study recently conducted in Malawi, halothane accounted for one quarter of the entire anaesthetic department budget.

TRICHLOROETHYLENE (Trilene)

Advantages: A non-irritant, safe agent. It provides good analgesia during and after surgery. It maintains cardiac output and is inexpensive.

Disadvantages: Trichloroethylene takes effect slowly. It has weak anaesthetic properties and may

result in a rapid respiratory rate in spontaneously breathing patients. Arrhythmias may occur and adrenaline administration is contraindicated. If high doses are used a prolonged recovery will occur - particularly in elderly patients. Trichloroethylene is stabilised in solution by the addition of 0.01% thymol and should be protected from light.

Indications: Trichloroethylene is mainly used as an analgesic supplement to halothane or used on its own for minor procedures such as fracture manipulation. It has been used as the sole agent for tonsillectomy without intubation and for analgesia in labour.

Contraindications: Never use trichloroethylene in a circle with soda-lime as the toxic compounds phosgene and carbon monoxide are produced.

Dosage: 0.5 - 1% initially, reducing to 0.2 - 0.5%.

Vaporisers: A variety of vaporisers have been used with trichloroethylene, the Oxford Miniature Vaporiser (OMV) is recommended.

Practical points: A very easy agent to give but remember to turn it off well before the end of the operation to avoid prolonged sedative effects. It is most commonly used to give background analgesia for long cases or combined with halothane for short cases using inhalation induction. When combining vaporisers in this way always place the halothane vaporiser closer to the patient. ICI has ceased production of their blue "Trilene", though laboratory grade trichloroethylene from Germany can still be obtained.

NITROUS OXIDE

Advantages: Nitrous oxide has a rapid onset and recovery. It is a good analgesic supplement for halothane and reduces the incidence of awareness. It produces minimal cardiovascular and respiratory effects.

Cylinders containing a 50% mixture of nitrous oxide in oxygen (named Entonox) are produced in some countries. The contents may be breathed by patients via a demand valve for analgesia following trauma, changes of dressings or childbirth.

Disadvantages: In developing countries nitrous oxide is expensive to produce and transport. It is delivered to the patient through a rotameter and is mixed with oxygen to produce an inspired mixture of not less than 30% oxygen. If the rotameters are set incorrectly a hypoxic gas mixture may be given to the patient. (This may be a particular problem if nitrous oxide is mixed with "oxygen" from an oxygen concentrator).

During anaesthesia nitrous oxide diffuses into any body cavity which contains gas. This includes air spaces in the gut, middle ear, endotracheal tube cuff and pneumothorax.

Diffusion hypoxia (Fink principle) may occur at the end of anaesthesia when nitrous oxide rapidly leaves the blood and tissues and passes out through the lungs. This may result in a dilution of the oxygen in the lungs for a few minutes and is prevented by administering extra oxygen at the end of anaesthesia.

In the developing world where resources are scarce and transport costs high, the use of nitrous oxide is an unnecessary extravagance. Before its use was discontinued at the Queen Elizabeth Hospital in 1988 nitrous oxide accounted for a quarter of the total pharmacy budget for the whole hospital (which included 3,000 outpatients a day)!

Contraindications: Nitrous oxide is not used in drawover circuits. It should never be given to a patient with an untreated pneumothorax or a patient who has been scuba diving within the previous 24 hours due to the potential for decompression sickness. Less than 50% nitrous oxide is largely ineffective.

How should volatile agents be used?

One method is to use them for both induction and maintenance of anaesthesia. The patient inhales the agent via a close-fitting facemask and provided the smell is accepted and the stage two excitement effects are not excessive, this is a very satisfactory method of inducing general anaesthesia for short, minor cases.

Another method is to induce anaesthesia intravenously and use the volatile agent for maintenance of anaesthesia. Often the intravenous induction will be followed by tracheal intubation. Most general

anaesthesia for major cases may be done this way. When muscle relaxants are used the concentration of anaesthetic agents may be reduced but care should be taken to avoid the patient becoming too light whilst paralysed.

PHARMACOLOGY OF NON-DEPOLARISING MUSCLE RELAXANTS

L.K.G. Vimal and O.O. Oladapo, Abmadu Bello Teaching Hospital, Zaria, Nigeria

Non-depolarising muscle relaxants are commonly used during anaesthesia to provide relaxation for surgery, to allow mechanical ventilation and they are also regularly used in intensive care. This article describes the mechanisms by which the drugs work and also the differences between specific drugs.

Mechanism of action

Non-depolarising muscle relaxant drugs (NDMRD) compete with acetyl choline (ACh) molecules released at the neuromuscular junction to bind with the ACh receptors on the post synaptic membrane of the motor endplate. They therefore block the action of ACh and prevent depolarisation (or activation) of the muscle contraction process. Muscle groups differ in their sensitivity to muscle relaxants; ocular muscles responsible for opening and moving the eyes are the most sensitive followed by the muscles of the jaw, neck, limbs, intercostals and abdomen. The diaphragm is the least sensitive muscle, which is why patients undergoing surgery sometimes hiccup or breathe as an early sign that the relaxants are wearing off.

Non-depolarising muscle relaxant drugs also act on presynaptic receptors interfering with the entry of calcium which causes an inhibition in the release of ACh. Other drugs such as the aminoglycoside antibiotics (eg gentamicin) and volatile agents may also effect this mechanism and increase sensitivity to relaxants.

A variety of relaxant drugs are in use in different parts of the world. All produce profound muscle paralysis but have varying effects on the autonomic nervous system. None of the drugs cross the blood brain barrier as they are water soluble, polar molecules and therefore have no effect on the central nervous system. All non depolarising drugs should be used with care in patients suspected to be suffering with myasthenia gravis or myasthenic syndrome as patients with these conditions are extremely sensitive to their effects.

The commonly used drugs are summarised below.

Tubocurarine (Curare, d-tubocurarine) is a naturally occurring drug which takes about 3 minutes to act when given intravenously and lasts for 30-40 minutes.

Cardiovascular effects: Curare has no direct action on the heart but there is often a slight fall in the blood pressure secondary to a vasodilating effect via the sympathetic ganglia. In the presence of volatile agents the blood pressure fall may be greater. Care should be taken with this combination in hypotensive patients.

Respiratory effects: Curare has occasionally been associated with bronchospasm due to the release of histamine. It should be used with caution in asthmatic patients.

Histamine release may occur following the administration of curare and frequently presents as a red weal in the line of the vein which has been used for the injection. Problems associated with this reaction are very rare.

Placental transfer is not a feature of curare and the drug may be safely used in obstetrics.

Effect of metabolic abnormalities: Curare is potentiated by the presence of respiratory acidosis and hypokalaemia.

Distribution, metabolism and excretion: Thirty to forty percent is excreted unchanged in the urine and most of the remainder in the bile. In renal failure the drug is excreted effectively by the biliary route provided large or repeated doses are avoided.

Dose, administration and use: The initial dose

should be 0.3 - 0.6mg/kg followed by supplementary doses of 5mg when required (usually after 20-30 minutes). Neonates (less than 1 month old) are sensitive to curare and an initial dose of 0.3mg/kg is recommended.

Storage: Curare does not need to be refrigerated.

Gallamine (Flaxedil) is a synthetic (manufactured) drug which acts 1 -2 minutes after i.v. injection and lasts 20-30 minutes.

Cardiovascular effects: Gallamine produces an increase in heart rate, usually by 20-30 beats/minute due to an inhibitory action on the vagal supply to the heart. Blood pressure is usually unaltered unless bradycardia was previously present.

Histamine release is very rare.

Placental transfer: Gallamine is thought to cross the placenta more than other relaxants although it has been used successfully for Caearean section.

Effect of metabolic abnormalities: Gallamine is potentiated by alkalosis and antagonised by acidosis.

Distribution, metabolism and excretion : Gallamine is excreted almost entirely by the kidneys and should be avoided in patients with renal impairment.

Dose, administration and use : A dose of 1.5 - 2mg/kg is effective in 1-2 minutes and lasts for 15-30 minutes. Supplementary doses are usually 20 mg.

Storage: Gallamine does not require refrigeration.

Alcuronium (Alloferin) is a semi-synthetic muscle relaxant which has many similarities with curare. It is slightly shorter acting than curare.

Cardiovascular effects: After an i.v. dose there is frequently a slight fall in blood pressure due to vasodilation secondary to a degree of sympathetic blockade. This is occasionally accompanied by a tachycardia. Alcuronium is associated with a slightly higher incidence of anaphylactoid reactions than other non-depolarising muscle relaxants.

*Placental transfer***:** Alcuronium does not cross the placenta in appreciable amounts and has been widely used in obstetrics.

Distribution, metabolism and excretion: Most of the drug is excreted unchanged in the urine although some is also excreted in the bile. When used in patients with renal or hepatic impairment the dose should be reduced.

Effect of metabolic abnormalities: Mild acidosis or alkalosis does not alter the duration of action of alcuronium.

Dose, administration and use: Although there is some variation in requirements between patients an initial bolus of 0.2-0.3mg/kg is usually sufficient to provide relaxation for 20-40 minutes. Further increments should be with 15-25% of the original dose. It is potentiated by halothane. In children some anaesthetists recommend using doses of 0.125-0.25mg/kg. Always allow at least 20 minutes following the last dose before attempting to reverse the patient.

Storage: Alcuronium should be stored below 25 degrees centigrade and be protected from light.

Pancuronium (Pavulon) is a synthetic non-depolarising neuromuscular blocking agent.

Cardiovascular effects: There is a mild vagal blocking effect on the heart and an inhibition of the re-uptake of noradrenaline by the cardiac sympathetic nerves. These result in a rise in pulse rate of about 20% and an increase in the blood pressure of 10-20%.

Respiratory effects: Pancuronium can be safely used in patients with asthma.
Histamine release is not a problem.

Placental transfer is not a problem and pancuronium may be used in obstetric anaesthesia.

Distribution, metabolism and excretion: Sixty to eighty percent of pancuronium is excreted through the kidneys and the remainder is metabolised in the liver and excreted in the bile. It should be avoided if possible, in patients with renal impairment.

Effect of metabolic abnormalities: Acidosis potentiates pancuronium.

Dose administration and use: An initial dose of 0.1mg/kg will last 20-40 minutes. Increments of 1-2mg should be given as required. Always allow at least 20 minutes following the last dose before attempting to reverse the patient. Infants, children, elderly and obese patients may be more sensitive to pancuronium.

Storage: Pancuronium should be kept in a refrigerator.

Atracurium (Tracrium) is a short acting relaxant which is rapidly broken down by the body. This makes atracurium very predictable as it wears off rapidly compared to the longer acting relaxants.

Cardiovascular effects: Although atracurium produces few direct circulatory effects the absence of vagal blocking activity makes the patient vulnerable to bradycardias during anaesthesia. These episodes are commonest during ophthalmic (traction on the ocular muscles), ENT or abdominal surgery, particularly laparoscopy. The patient should be monitored closely and any bradycardias treated with atropine. Some anaesthetists give atropine or glycopyrrolate routinely at induction to prevent this problem.

Histamine release may occur with doses of atracurium greater than 0.6mg/kg. Histamine may also be released if atracurium precipitates in the syringe or vein. This may occur if atracurium is injected immediately after thiopentone.

Respiratory effects: In standard doses atracurium rarely causes problems with bronchospasm.

Placental transfer is insignificant and the drug is widely used in obstetrics.

Distribution, metabolism and excretion: Atracurium is broken down to inactive metabolites by ester hydrolysis and spontaneous Hoffman degradation. There is little change in its effects in patients with renal or liver failure. When used for long operations it is very predictable.

Dose, administration and use: A dose of 0.3-0.6mg/kg will provide relaxation for 20-40 minutes. Supplemental doses should be 5-10mg.

Contraindications: Atracurium precipitates (comes out of solution) in an alkaline pH and it should never be mixed with thiopentone. Always flush the vein with saline if using the two drugs at induction.

Storage should be in a refrigerator at 4 degrees centigrade as the drug deteriorates at room temperature.

Vecuronium (Norcuron) is a short acting relaxant prepared as a powder which is dissolved in sterile water immediately prior to use.

Cardiovascular effects are minimal although the potential problems listed under atracurium apply.

Histamine release is not a feature.

Placental transfer is minimal.

Distribution, metabolism and excretion : Vecuronium is excreted both in bile and urine. Its action is slightly prolonged in renal impairment.

Dose administration and use: An i.v. dose of 0.08 - 0.1mg/kg will produce relaxation for 15-30 minutes and supplemental doses should be 1-2mg.

Storage: Vecuronium powder does not need to be refrigerated.

General considerations in the use of muscle relaxants

Muscle relaxants are principally used to provide good muscular relaxation for surgery. When they are used respiration must be controlled via an endotracheal tube. A few general guidelines for the use of relaxants are listed below:

Always be certain that you will able to ventilate the patient by face mask before paralysing them.

If a rapid onset of action is required then suxamethonium should be used as it acts more quickly than any of the non-depolarising drugs. If a short duration of paralysis is required suxamethonium is

most suitable and may be given in repeated doses provided atropine is administered prior to the second dose of suxamethonium to avoid bradycardia.

Non-depolarising muscle relaxants take about one and a half to two minutes to act and you should allow time for relaxation to develop before attempting intubation.

The supplemental dose should be about 25% of the initial dose. Never attempt to reverse the relaxation until at least 15-20 minutes after the last dose of relaxant was given.

Never extubate a patient until you are certain that the paralysis has been reversed and they have adequate muscle strength to protect their airway and breathe. One way of testing this is to assess whether they are able to lift their head off the pillow for 5 seconds. Ensure that breathing is of adequate depth and frequency.

It takes some time before the larynx is able to protect the airway and so the patient is best placed in the lateral position for recovery.

If a nerve stimulator is available it can be used to monitor the degree of relaxation. However it is not essential and relaxants can be safely be used without a nerve stimulator by careful observation of clinical signs.

When muscle relxants are administered awareness is always a danger since a paralysed patient cannot move in response to pain. It is therefore essential to ensure that the depth of anaesthesia is adequate.

Relative costs of muscle relaxants (UK prices)

Drug	Ampoule size	Cost
Curare	15mg	71p
Gallamine	80mg	72p
Alcuronium	25mg	£1.86
Pancuronium	4mg	66p
Atracurium	50mg	£3.38
Vecuronium	10mg	£4.23
Suxamethonium	100mg	31-71p

VENOUS CUTDOWN AND INTRAOSSEOUS INFUSION

Brian W Davies, Registrar in Paediatric Surgery, St James' University Hospital, Leeds, UK

Gaining intravenous access is a common procedure but may be difficult in hypovolaemic patients or those with difficult veins. When direct cannulation of a vein cannot be performed or is taking too long, a venous cutdown or intraosseous infusion are alternative methods of access to the circulation. These two techniques are described below. In this article "proximal" means the part of the vein or bone closer to the chest, and the word "distal" the part of the vein or bone furthest from the chest.

Venous cutdown

This procedure exposes the vein surgically and then a cannula is inserted into the vein under direct vision. If no cannulae are available the sterile end of the drip tubing may be used in adults after cutting off the Luer (cannula) connection. The procedure must be performed under sterile conditions to avoid sepsis developing which will not only shorten the life of the infusion but may have serious consequences for the patient.

During the procedure 2 ligatures (sutures) are placed around the vein. The distal ligature is used to tie off the vein distally and the proximal ligature holds the cannula in the vein While the vein is incised the ligatures help to hold it.

Equipment

1. Sterile gloves
2. Swabs and sterile drapes
3. Skin disinfectant
4. Local anaesthetic (5ml of 0.5% lignocaine is sufficient)
5. Scalpel
6. Two small curved artery forceps
7. Sharp pointed scissors (use scalpel if scissors blunt/unavailable)

8. Ligatures (2/0 catgut / vicryl are best, but silk is adequate)
9. Skin closing sutures
10. Cannula

Sites. In adults use the upper limb at the medial aspect of the antecubital fossa. Try to avoid the leg veins as they are thicker and more prone to thrombosis, phlebitis and infection. In children a cutdown may be performed using either the brachial or long saphenous veins.

Technique. Clean the skin and use the drapes to create a sterile area around the chosen vein.

1. Infiltrate the skin with local anaesthetic.

2. Make a 1.5 - 2cm transverse incision over the vein (a).

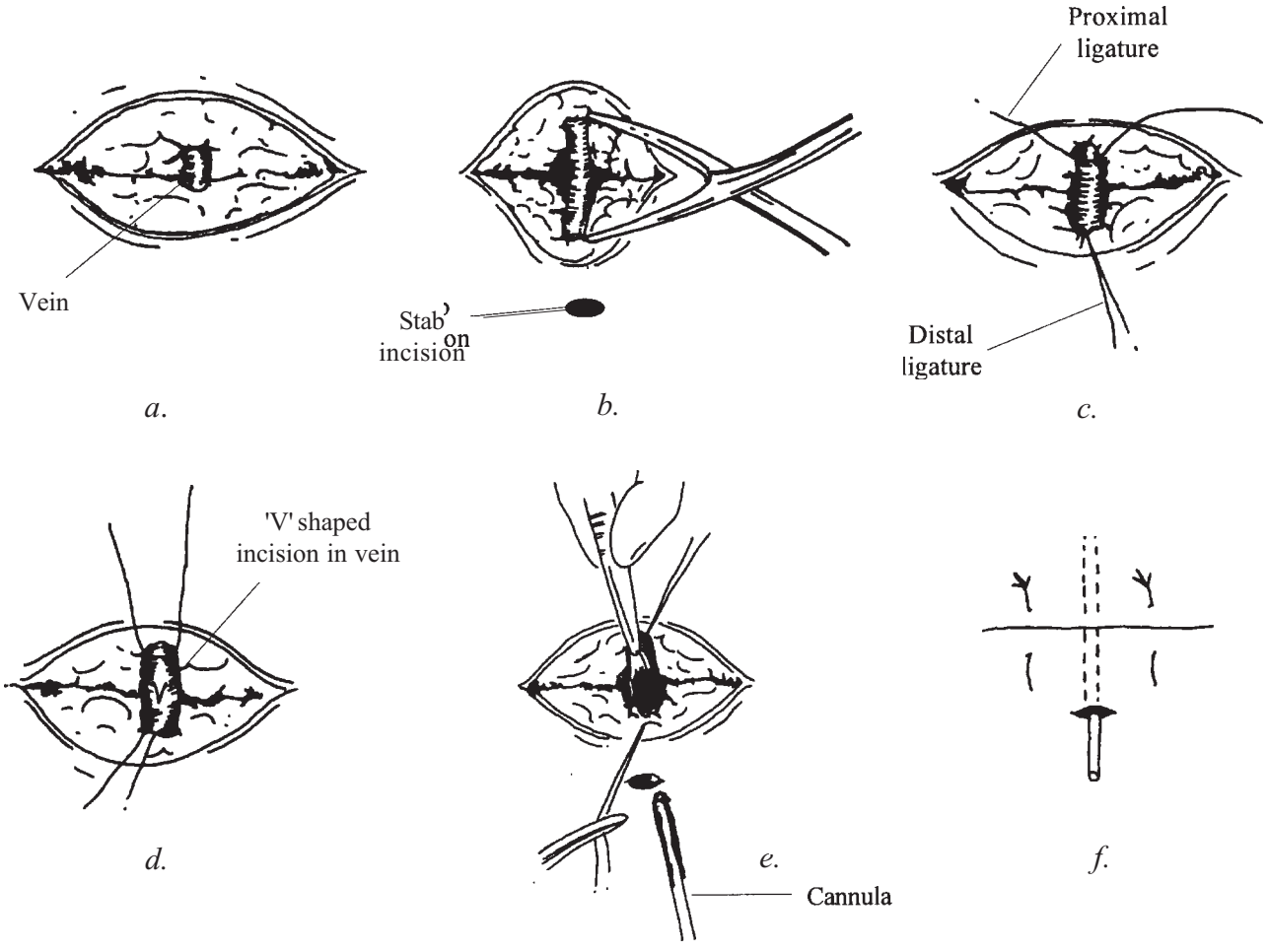
3. Bluntly dissect out the vein by opening the forceps in the line of the vein (b).

4. Make a small stab skin incision 1cm distal to the incision in the line of the vein. Pass two ligatures around the vein. Tie the distal one, but leave the ends uncut. Hold the ends of the ligatures with the artery forceps (c).

5. Whilst holding the ligatures tight, make a "V" shaped incision in the anterior surface of the vein with the scissors or scalpel (d & e).

6. Pass the cannula through the inferior stab incision and the through the "V" shaped incision into the vein. Tie the proximal ligature tightly over the cannulated vein and, if there is no bleeding, now cut the ends of the ligatures. If bleeding occurs place a further ligature around the vein. Connect the cannula to the giving set and commence the infusion.

7. Close the skin with sutures (f).



After the infusion is finished the cannula can be removed by a firm steady pull followed by direct pressure over the site of the incision for 5 minutes.

Intraosseous infusion

The marrow cavity can be used for the administration of fluids as it is in continuity with the venous circulation. Blood can be taken for crossmatch and electrolyte estimation and fluid or drugs may be given provided they are gently syringed in. The procedure must be performed under sterile conditions to avoid causing osteomyelitis. The infusion is best limited to a few hours until intravenous access is achieved.

Indication: The technique is used for vascular access in life threatening situations in babies, infants and children. It is indicated when other attempts at venous access fail.

Equipment

1. Skin disinfectant
2. Intraosseous or bone marrow needle
3. Local anaesthetic
4. 5ml syringe
5. 50ml syringe

Site. Use either the anterior aspect of the tibia or femur. Avoid bones with osteomyelitis or fractures and do not use the tibia if the femur is fractured on the same side.

Technique

1. Clean the skin and inject a small amount of local anaesthetic in the skin and continue to infiltrate down to the periosteum
2. Insert the intraosseus needle at 90 degrees to the skin (perpendicular).

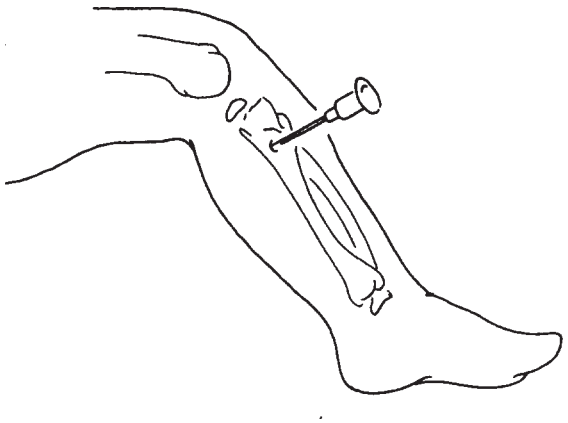


Figure 2. Tibial technique for intraossesous infusion

3. Advance the needle until a "give" is felt; this occurs when the needle penetrates the cortex of the bone.
4. Remove the trochar. Confirm correct position by aspirating blood using the 5ml syringe.
5. Secure the needle in place with sterile gauze and strapping.
6. Give boluses of fluid (infusion volume depends on clinical situation) using the 50ml syringe to push the fluid in gently.
7. The intraosseous route should be replaced as soon as a normal vein can be cannulated. The longer the period of use the greater the risk of sepsis.

MAKE YOUR OWN ENDOTRACHEAL TUBE INTRODUCERS.

Mr M Yeats, Derriford Hospital, Plymouth

An endotracheal tube introducer made of thick copper wire can be used to stiffen the tube during intubation. This may be of help during difficult intubations. The introducer has a loop at one end to hold it with and a rounded silver tip at the other end which prevents tissue damage during use. The loop also prevents the introducer from accidentally disappearing down the endotracheal tube. An introducer can be easily made as described below.

A piece of copper wire of a suitable diameter should be obtained from your hospital maintenance department or a local electrical repair shop. Use a thicker wire for adults and a thinner piece for children.

Cut a piece of the wire 5 centimeters longer than your longest endotracheal tube and bend one end of the wire into a loop big enough to pass your finger through. Using a piece of sandpaper round off the other end of the wire to make it smooth.

Although the introducer may be used in this basic form it is preferable if the tip is rounded off by putting on a silver atraumatic tip. This is done by holding the introducer with the loop facing upwards in a vice. Put some solder flux paste on the tip and heat it with a hot flame (for example a propane torch) until the flux turns to a clear colour. Apply the silver solder (use the type that jewelers use) to the tip of the wire so that it melts on to it. Allow it to form a tear drop shape then stop heating. Allow it to cool and then clean off the crusted flux and polish the silver tip until it is smooth. It is also advisable to solder the loop closed in the correct position. When this type of introducer is used the tip should reach almost to the end of the endotracheal tube but never protrude beyond it. If the introducer is longer than the tube being used, bend the stem of the introducer to prevent it from going down too far.

An improved version of the introducer can be made which will fit most tubes. A tapered brass cylindrical block about 2.5 cm long is drilled so that the wire can slide through the middle. A thread is tapped for a small thumb screw at a right angle to the direction of the wire. When tightened the small thumb screw holds the block in position and makes the introducer adjustable to fit all lengths of endotracheal tubes. The block is tapered to make a good fit inside the endotracheal tube.

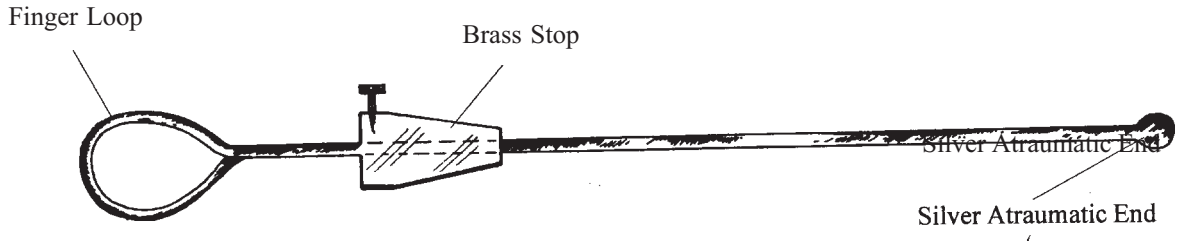


Figure 1. Endotracheal Introducer

CORRESPONDENCE

What do you do when the laryngoscope bulb fails during intubation?

I enjoyed the excellent article on maintaining a laryngoscope written by Mike Yeats in Update in Anaesthesia No 4. One problem I have come across is failure of the laryngoscope during the intubation sequence. I suggest the easiest solution is to either perform a blind nasal intubation (if you have the necessary skills) or use the operating light shining in the mouth whilst exposing the larynx with the blade of the laryngoscope. This is usually successful and may be quicker than trying to fix the bulb.

Mr C K Essien
PO Box 505
Obya Adansi
Ghana

Editors' note:

This is useful advice but remember that if intubation is delayed care must be taken to ensure that the patient does not become hypoxic by using facemask ventilation. Often a quick check that the bulb is screwed in tightly and then reopening the blade to improve contact may be enough to remedy the situation. Always test the laryngoscope prior to inducing anaesthesia and, if possible, have a spare one at hand.

GENERAL ANAESTHESIA FOR CAESAREAN SECTION

I would like to make some additional comments on the recent article by Dr Patricia Coyle from Ethiopia. General anaesthesia for Caesarean section in Malawi Government hospitals is via the Danish Malawi anaesthetic machine which is composed of a drawover circuit and an oxygen concentrator. The oxygen concentrator can deliver 95% oxygen at 2 litres per minute or about 80% oxygen at 4 litres a minute. The remaining part of the minute volume of the patient is room air. Before general anaesthesia for Caesarean section pre-oxygenation should be attempted. During this phase the patient generally breathes with a minute volume of some 10-15 litres a minute. Unfortunately in a drawover circuit it is not possible to provide an adequate fresh gas flow using 100% oxygen. Therefore when I perform a rapid sequence induction I preoxygenate the patient using 2 litres of 95% oxygen through the drawover circuit and then induce anaesthesia using thiopentone or ketamine followed by sux-amethonium. Cricoid pressure is applied in the usual fashion and is maintained until the airway is secured with an endotracheal tube. The lungs are gently ventilated by face mask with cricoid pressure in place whilst waiting for the suxamethonium to act. In our situation the gentle hand ventilation prevents hypoxia developing and is important if an inadequate flow of oxygen is being delivered to allow full pre-oxygenation. I have given anaesthesia for between six and seven hundred cases of Caesarean section with the technique described and have not encountered any problems of regurgitation or aspiration.

Mr S Phiri
Anaesthetic Department
Kamuzu Central Hospital
PO Box 149
Lilongwe
Malawi

Editors' comments:

The problem of preoxygenation via a drawover circuit has been addressed in two ways.

The first by using the technique that Mr Phiri describes. Figure 1 illustrates how the inspired oxygen concentration during pre-oxygenation varies according to the percentage of oxygen delivered by the oxygen concentrator and the patients minute volume. Most small oxygen concentrators have an output between 1 and 4 litres/minute. At 1-2 litres a minute the concentrator will typically produce 95% oxygen, at 4 litres a minute 75-85% oxygen. Figure 1 illustrates how a flow of 4 litres a minute with 80% oxygen produces the best form of pre-oxygenation in this situation. The circuit will always entrain some air and preoxygenation will therefore never be complete.

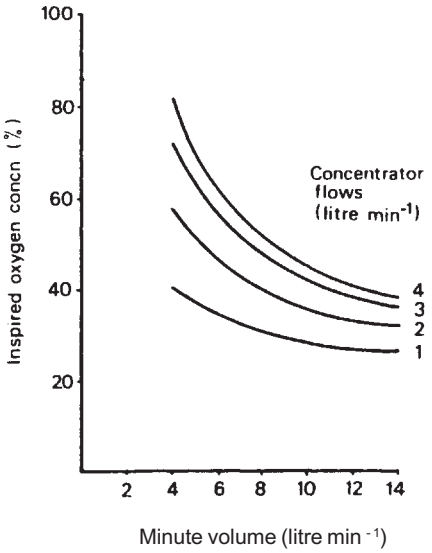


Figure 1. Predicted inspired oxygen concentrations.

In the second method a disposable plastic bag (such as a bin bag) is prefilled with oxygen from the oxygen concentrator and is then temporarily attached to the drawover circuit in such a way that the patient breathes from the bag during the pre-oxygenation phase. Using this technique the patient breathes 95% oxygen during the time it takes to empty the bag and preoxygenation is much more efficient. Remember to disconnect the bag as soon as it is empty or the circuit will become obstructed.

QUESTIONNAIRE

Update in Anaesthesia is designed to provide a source of practical advice for anyone administering anaesthesia in developing countries or difficult circumstances. We know that a variety of people perform this role from fully trained physician anaesthetists, clinical officer anaesthetists, nurse anaesthetists, surgeons, general doctors, medical students and in some places untrained personnel. We hope that the articles are useful and that the mixture of subjects is fairly balanced. We are keen to receive comments about Update and are grateful for any letters sent to us. A short questionnaire has been printed below to discover a few details about our readers. This will help us in the choice of articles we publish, and will assist our sponsors to assess the suitability of our project for further funding. If you read our journal please help us by completing the answers on a separate sheet of paper and sending them to Dr I H Wilson, Department of Anaesthetics, Royal Devon and Exeter Hospital, Barrack Rd, Exeter EX2 5DW, U.K.

1. What is your name, and what is the address of your hospital?

2. What kind of hospital do you work in?

3. How many beds are in your hospital?

4. How many doctors work in your hospital

5. What specialities are available in your hospital?

6. What operations are done in your hospital (if you can supply the types of operations and approximate numbers over a 3 month period it would be helpful)?

7. How many anaesthetists are in your department and what grades are they?

8. What is your position and role in your hospital.

9. What anaesthetic training have you received?

10. In the last 6 months how many cases did you anaesthetise using (please give approximate numbers):
    a. A Boyles (continuous flow or plenum) machine?
    b. A drawover system using
       EMO ether?
       OMV halothane?
       OMV trichloroethylene?
       PAC or TEC ether?
       PAC or TEC halothane?
    c. Spinal or Epidural anaesthesia?
    d. Brachial plexus block
    e. Local infiltration anaesthesia?
    f. Intravenous regional anaesthesia (Bier's block)?
    g. Ketamine?
    h. Open drop ether?

11. Are there any drugs or equipment which are in sort supply at times in your hospital? If yes what are they?

12. How do you receive our journal?

13. How many people read your copy of Update?

14. Which editions have you received?

15. Which articles do you think were most relevant to you?

16. Are the articles easy to understand? If not, why not?

17. Are any sections more worthwhile than others?

18. Have there been any articles which were not useful?

19. What subjects would you like to see covered in the future?

20. Has Update changed your anaesthetic practice in any way?

21. Do you have access to anaesthetic textbooks in your hospital? Which books do you refer to most and when were they written?

22. When was the last time that you received any training in anaesthesia (of any type - please explain)?

23. Do you have access to a fax machine ?

24. Do you have access to a computer / word processor. If yes, which type ?

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