Update in
Anaesthesia

Pentazocine

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INTRODUCTION

In 1967 pentazocine became the first opioid agonist-antagonist to be introduced into clinical practice as an analgesic. It was hoped that pentazocine would prove to be a powerful analgesic, free of the side-effects of opioid narcotics, particularly avoiding drug dependency. In practice pentazocine has proved to be less effective than hoped, but it is still used widely in resource-poor countries.

CHEMISTRY

Pentazocine is a benzmorphan which is chemically related to morphine. It is a white or cream, odourless, crystalline powder. It consists of a racemic mixture of dextro- (d) and laevo- (l) isomers which is soluble in acidic aqueous solutions. Pentazocine hydrochloride is used for oral use and the lactate form is used for parenteral and rectal administration.

Molecular weight (free base)	321.9
pKa	8.7
Solubility in water	1 in 30
Chemical structure	$C_{19}H_{27}NO.HCl$

PHARMACODYNAMICS

The action of pentazocine is mainly due to its l-isomer and it is a potent analgesic with both an agonist and antagonist action at opioid receptors. It differs from morphine in that it is a weak antagonist at OP3 (μ) opioid receptors, with its analgesic action due to an agonist action on OP2 (κ) receptors which interrupts pain pathways in the spinal cord. It also has some agonist action at other receptors which may result in dysphoric side-effects. It has no anti-inflammarory or anti-pyretic function.

Given intravenously, estimates of its potency vary from one third to one quarter the strength of morphine. This ratio is similar with intramuscular use with 30 - 40 mg of pentazocine equivalent to 10 mg morphine. When given by mouth, the analgesic action of pentazocine is much weaker than morphine and is thought to lie somewhere between that of peripherally acting analgesics such as paracetamol and weak opioids such as codeine.

Other actions of pentazocine mirror those of other opioids including respiratory depression, cough suppression, miosis, decreased gastric emptying and constipation and increased smooth muscle tone in the uterus and bladder. However in normal use these effects are usually of little clinical significance.

In contrast to other strong opioid analgesics however, there is a dose-related systemic and pulmonary hypertension, increased left ventricular end-diastolic pressure and a rise in central venous pressure, probably as a result of a rise in plasma catecholamine concentrations. Pentazocine also increases renal plasma flow but no change in glomerular filtration rate is seen.

PHARMACOKINETICS

Absorption

Pentazocine is completely absorbed after oral administration with peak plasma concentration at about 1-3 hours and a mean plasma half-life of about 2 hours. However blood levels show considerable variation both within and between subjects due to extensive but variable pre-systemic (hepatic) elimination.

Oral bioavailability ranges from 11 to 32% in subjects with normal hepatic function. Regardless of route of administration, pain relief lasts 2-3 hours as a maximum.

Animal studies show rapid and widespread distribution in the liver, lungs, kidneys, muscle and brain following IV or IM administration.

Summary

This article describes the pharmacology and clinical uses of the opioid agonist-antagonist pentazocine. Pentazocine is widely used around the world despite its considerable side-effects and tendency to cause dependency. It still has a useful function to play in the management of moderate to severe pain when used intravenously or intramuscularly, but should not replace morphine or pethidine if available.

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Low levels are detected in organs other than the liver after oral administration due to pre-systemic elimination.

The apparent volume of distribution varies but indicates significant drug accumulation in some tissues. Plasma protein binding is also variable, but up to 50 % may be present in red blood cells. Placental transfer occurs, with mean cord blood levels in the region of 60-70 % of those in maternal blood.

Table 1. *Peak plasma concentrations*

Route of administration	Time to peak plasma concentration (minutes)
Intravenous	2-3
Intramuscular/ subcutaneous	15-30
Oral	60-90

As a result of extensive hepatic metabolism, less than 10 % of an oral dose appears unchanged in the urine, although in patients with cirrhosis a significant reduction in body clearance and a marked increase in bioavailability are seen.

Metabolism and elimination

Up to 10 % is excreted unchanged in the urine with 1-2 % in the faeces as a result of enterohepatic circulation. Both are independent of the route of administration. The remainder undergoes extensive hepatic metabolism, including conjugation with glucuronic acid and oxidation of the terminal methyl groups of the dimethylallyl side chain. The two principle metabolites found in the urine are the cis-alcohol metabolite (11 %) and the trans-carboxylic acid metabolite (40 %). Both are inactive.

An increased rate of metabolism due to enzyme induction has been reported in smokers and following nitrous oxide administration.

PHARMACEUTICAL PREPARATIONS

Trade names of pentazocine include Fortral, Talwin, Fortralgesic, Fortralin, Sosegon, Sosenyl, Pentgin and Liticon. Pentazocine is also available in combination with aspirin and paracetamol (acetaminophen).

DOSE AND PATIENT RESTRICTIONS

As with analgesics in general there is no consistent relationship between analgesic activity and plasma concentrations of pentazocine. However normal doses are set out in Table 3.

Adults

Oral administration is usually started at 50-100 mg every 3-4 hours; titrating dose and frequency of administration to pain relief with the total daily dose not exceeding 600 mg.

Starting treatment at night, and using frequent smaller doses in preference to less frequent large doses, helps to reduce the incidence of side effects. Rectal administration may give more prolonged analgesia than equivalent oral doses.

Table 2. *Pharmaceutical preparations of pentazocine*

Parenteral forms:	
1. Talwin injection (USA):	30mg/ml in 1 or 2ml ampoules, sterile cartridge needle units, 10ml multiple dose vials. Mixed in an aqueous solution of pH4-5 as pentazocine lactate. For IV, IM and subcutaneous injection.
2. Fortral injection (UK):	30mg/ml in 1 or 2 ml ampoules for IV, IM or subcutaneous use
Oral forms:	
1. Talwin-Nx (USA)	50mg pentazocine with 500mcg naloxone
2. Fortral tablets (UK)	25mg and 50mg pentazocine hydrochloride tablets
3. Talwin Compound	12.5mg pentazocine with 325mg aspirin (2 tablets three-four times daily)
4. Talacen (acetaminophen)	25mg pentazocine with 650mg paracetamol (1 tablet four hourly)
Rectal forms:	
1. Fortral suppositories	50mg pentazocine lactate
Generally preparations should be stored at room temperature and protected from light and freezing	

Table 3. *Dosing of pentazocine*

Route of administration	Dose	Interval	Total dose in 24 hour
Intravenous - inject undiluted by slow bolus	0.5 mg/kg or 30-40mg	3-4 hourly	not to exceed 360mg
Intramuscular - inject deep into well developed tissue	1.0 mg/kg or 30-60mg	3-4 hourly	not to exceed 360mg
Oral	50-100mg	3-4 hourly	not to exceed 600mg
Use subcutaneous injection only when necessary: severe tissue damage is possible at injection sites			

Children
Oral administration under the age of 6 years is not recommended. Some sources recommended avoiding pentazocine for children of 6-12 years, although other studies suggest 25 mg every 3-4 hours as a suitable dose. Children over 12 years may receive an adult dose.

Use in pregnancy
Safety has not been unequivocally established and penatzocine should be used with caution. Neonatal dependency has been reported in women who have taken 50-300 mg daily throughout pregnancy.

Pentazocine has not been demonstrated to pass into breast milk although monitoring is recommended if high doses are prescribed.

Use in the elderly
Although no specific problems have been identified, care is required with impairment of hepatic or renal function as it may predispose to increased toxicity.

THERAPEUTIC USE

Postoperative pain
Pentazocine is a controlled drug in the UK and is prescribed parenterally for moderate to severe acute postoperative pain. In these circumstances 30-60 mg by IM or SC injection has a similar analgesic action to 10 mg morphine or 100 mg pethidine (meperidine).

In comparison to morphine or pethidine, the duration of action of pentazocine is slightly shorter. It has been claimed that pentazocine produces a lower incidence of side-effects in postoperative patients compared with morphine and pethidine, in particular nausea and vomiting, sedation and hypotension. However the side-effects of pentazocine are dose-related and overall the incidence and severity of side-effects at equivalent analgesic doses are similar with all three drugs.

Oral pentazocine is not a strong analgesic. While some comparisons show it to be as effective as codeine or dihydrocodeine, other studies have shown aspirin to have a greater analgesic effect than pentazocine. It is clear that the duration of analgesia produced by pentazocine is about 3 hours.

Chronic Pain
The usefulness of oral pentazocine in chronic pain is limited by its weak and unpredictable analgesic activity, dose-related (particularly psychomimetic) side-effects and its ability to antagonise the effects of pure opioid agonists if used concurrently.

Obstetrics
Pentazocine appears to be an effective analgesic during labour. There is some evidence that uterine activity may be increased and, compared with pethidine, the second stage of labour may be shortened. However there is no advantage with regard to side-effects; respiratory depression is comparable to pethidine in both the mother and the neonate.

Renal and biliary colic
Pentazocine may be used for the relief of acute pain of renal or biliary colic and has been shown to cause less smooth muscle contraction in renal and biliary tracts that morphine. However another study has shown a significant rise in intrabiliary pressure so that its use may be best avoided if other drugs are available.

Myocardial infarction
Pentazocine is an effective analgesic post myocardial infarction and may cause a rise in systolic blood pressure in contrast to the hypotensive effect of other opioids. However the associated rise in pulmonary artery pressure, left ventricular end-diastolic pressure and left ventricular minute work is potentially hazardous as it may lead to an increase in myocardial oxygen demand and extension of the infracted area. An alternative opioid analgesic is generally preferred.

Intravenous anaesthesia and premedication
There have been attempts to use pentazocine for intravenous anaesthesia and as a premedicant prior to general anaesthesia. However it confers no advantage over standard drugs and is generally only used when other drugs are not available.

CONTRAINDICATIONS

1. Respiratory depression
2. Raised intracranial pressure
3. Arterial or pulmonary hypertension
4. Pre-existing opioid dependency
5. Porphyria

Respiratory depression

This is mainly found with the l-isomer and in equipotent doses to that seen with morphine. Care is required in patients with impaired respiratory drive. Transient apnoea may occur in the neonate following its use in labour.

Raised intracranial pressure

Pentazocine increases intracranial fluid pressure in patients with an acute brain injury or when ICP is already increased due to a space-occupying lesion. However these changes are not seen in normal patients or in patients ventilated after a head injury, suggesting that mild respiratory depression with an associated intracranial vasodilatation from a rise in $PaCO_2$ may be the underlying cause.

Arterial or pulmonary hypertension

Pentazocine increases both heart rate and systolic blood pressure to a variable and unpredictable degree and should be avoided in patients with hypertension. IV doses of 30-60mg may cause an increase in pulmonary artery pressure with an associated increase in left ventricular end-diastolic pressure and their use should also be avoided. An increase in central venous pressure has also been reported but little consistent effect on systemic vascular resistance, cardiac output, stroke volume or coronary perfusion has been found.

Pre-existing opioid dependency

Withdrawal effects may occur in patients with opioid dependency due to its opioid antagonistic action.

Porphyria

Pentazocine has been found to be porphyrogenic in rats and on this basis is not recommended for use in patients with porphyria.

ADVERSE REACTIONS

Despite hopes that pentazocine would have less severe side-effects than pure μ-receptor agonists, this has not been the case in practice.

Life threatening adverse effects

Respiratory depression - when used as an adjuvant to anaesthesia in patients with chronic respiratory insufficiency.

Agranulocytosis - latency of 4-24 weeks from exposure to the drug. Although fatalities have been reported most cases are reversible on withdrawing the drug.

Severe or irreversible adverse effects

Epileptic seizures - rare and most often associated with high dose IV use during anaesthesia, or if there is an underlying intracranial pathology.

Addiction - although not considered addictive when first introduced, its potential for abuse has been increasingly recognised, particularly for the IV formulation which has resulted in greater control over its prescribing. As a consequence pentazocine-only tablets have been withdrawn in the USA in favour of one in combination with naloxone. Although naloxone is ineffective when given orally it is effective when injected IV.

Withdrawal symptoms are usually mild and include anxiety, dysphoria, tremor, sweating and musculoskeletal pains.

Pruritis - stinging, flushing when given IV. Repeated IM injections may cause soft tissue induration, fibrosis or ulceration, hyperpigmentation and a myopathy which, if severe, may impair movement resulting in contractures (more often associated with drug abuse rather than therapeutic use.

Symptomatic adverse effects

They are often dose-related, mild and self-limiting but can be severe:

- Psychotomimentic effects in 20% of patients. Includes disturbed dreams, auditory or visual hallucinations, euphoria and depersonalisation. Naloxone may be effective.
- Sedation, light headedness, vertigo
- Nausea and vomiting
- Other opioid side-effects include sweating, hot flushes, dry mouth, urinary retention
- Blurred vision, nystagmus, diplopia, miosis
- Headaches, chills and fever.

ACUTE OVERDOSE

Deaths due to pentazocine alone are rare. The main clinical features are respiratory depression, tachycardia and hypertension. Status epilepticus, coma, acidosis, respiratory depression, profound hypotension and ventricular arrhythmias can also be found.

Treatment is IV naloxone, a competitive antagonist at the opioid receptors, mediating the respiratory depression and maintaining respiration. Dose given is 0.1 mg repeated at 2-minute intervals according to the clinical state of the patient. 0.4-2 mg is sufficient in most cases although higher doses of 15-20 mg have been known to be required. An infusion of naloxone may be required.

OTHER EFFECTS

No significant biochemical effects have been recognised. No interference with clinical pathology tests. IM injections may increase creatine kinase levels.

DRUG INTERACTIONS

- **Halothane** - increases respiratory depression and hypotensive effects.
- **Anticoagulants** - may increase anticoagulant effect of both heparin and oral coumarins (e.g. warfarin).
- **Lignocaine** - prior administration enhances respiratory depressant effect of pentazocine.
- **CNS depressants** - increased sedation.
- **Opioid analgesics** - withdrawal effects if opioid dependency.
- **Monoamine oxidase inhibitors** - increased toxicity in mice although not demonstrated in humans.
- **Barbiturates** - do not mix in the same syringe as precipitation will occur.

CONCLUSION

Pentazocine was introduced in the hope that it would provide pain relief for moderate to severe pain without the side-effects of morphine. Unfortunately the reality has been disappointing. Although 30 mg of IM or IV pentazocine is comparable to 10 mg morphine, it has a more variable efficacy than morphine and its side-effects are still considerable, particularly the potential for dependency. This almost certainly accounts for the decrease in its use in the United Kingdom. Despite its removal from the WHO guide of essential drugs, it is still widely used in some poorly-resourced countries.

In summary, pentazocine still has a useful function to play in the management of moderate to severe pain when used intravenously or intramuscularly but should not replace morphine or indeed pethidine if available.

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Update in
Anaesthesia

Malignant hyperthermia

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Summary

Malignant hyperthermia (MH) is a rare but potentially fatal condition triggered by suxamethonium or an anaesthetic vapour. Early recognition of signs and prompt treatment are essential. The pathophysiology, clinical features and treatment of MH are described, with an emphasis on management and prevention in poorly-resourced settings.

INTRODUCTION

Malignant hyperthermia (MH) is a rare condition that was first recognised in the 1960s when a young man presenting for repair of a fractured tibia and fibula was more concerned about the anaesthesia than the operation. It transpired that 10 of his relatives had died following anaesthesia with ether. He subsequently went on to have a mild reaction to halothane anaesthesia.[1] Analysis of the family tree of this patient indicated an abnormal response to anaesthesia that was inherited via a dominant gene with incomplete penetrance. Much has been elucidated about MH since that time. There is good understanding of the cellular basis for MH, there is a specific treatment agent (dantrolene), an in vitro test for susceptibility to the condition has been developed and the specific gene defect has been identified. However, MH remains a condition that has the potential to be rapidly fatal in an otherwise fit and healthy individual. Anaesthetists should be aware of the diagnosis and treatment of this condition, the need to avoid specific trigger agents, and they should be able to provide safe anaesthesia for a patient who is susceptible to MH.

The actual incidence of MH cases has been reported to be between 1:5,000 and 1:50,000-1:100,000 general anaesthetics, but the prevalence of susceptibility to MH is felt to be much higher at about 1:3,000.[2] Due to its genetic basis, the prevalence of MH susceptibility varies around the world, and case reports or series have been reported for most ethnic groups including black Africans,[3,4,5] Thai,[6] Chinese,[7,8] Japanese[9] and Brazilians.[10]

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PATHOPHYSIOLOGY OF MALIGNANT HYPERTHERMIA

The primary problem in MH is an inherited disorder of calcium handling in the sarcoplasmic reticulum of skeletal muscle. The genetic defect is found on the gene encoding the intracellular ryanodine receptor, responsible for calcium release in skeletal muscle cells. In response to potent volatile anaesthetics and depolarising muscle relaxants the uncontrolled release of intracellular calcium leads to muscle rigidity, a hypermetabolic state and a build up of the breakdown products of skeletal muscle.

When suxamethonium is used, the first sign may be masseter spasm, where the jaw is clenched tightly after administration, preventing intubation or airway manoeuvres. Not all cases of masseter spasm will go on to develop MH, but all should be treated with a high degree of suspicion. If possible, further investigation for MH in all cases of masseter spasm is advised, since some will prove to have susceptibility despite the lack of a subsequent reaction.

Without treatment, MH can lead to multi-organ failure and death. Muscle breakdown products accumulate (rhabdomyolysis), leading to hyperkalaemia and myoglobinuria. Enzymes from skeletal muscle can cause renal failure, cardiac failure and disseminated intravascular coagulation. Renal failure exacerbates the hyperkalaemia and acidosis from rhabdomyolysis. The combination of hyperkalaemia, acidosis and hyperthermia lead to a high risk of fatal myocardial arrhythmias. Otherwise, renal failure as a consequence of rhabdomyolysis may prove to be fatal, particularly where there are no facilities for renal replacement therapy.

Initially mortality from MH was about 80%. Improved recognition and improvements in anaesthetic monitoring have helped to reduce mortality. Outcomes have improved significantly since the introduction of dantrolene, a specific treatment for MH, with no deaths reported in a series of New Zealand cases since dantrolene became available in 1981.[11] Mortality in countries

with limited access to dantrolene may still be significant, with a mortality of 25.8% in Taiwan between 1994 and 2004.[12]

MH is linked to some rare myopathies. Central core disease (CCD), a rare non-progressive autosomal dominantly inherited myopathy has been shown to be linked to RYR-1 mutations in 93% of Japanese patients.[2] Patients typically present in infancy with hypotonia and proximal muscle weakness. CCD is closely linked to MH susceptibility by in-vitro contracture testing. However, the link is variable, and there are other rare mutations associated with central core disease.

CASE SCENARIO

A 24-year-old male of Maori origin presented for open appendicectomy as an emergency. The patient had no past medical history of note, no previous anaesthetics and denied any family history of anaesthetic complications.

A rapid sequence induction was performed with alfentanil, thiopentone and suxamethonium. Intubation was awkward with some muscle tension noted, but not overt masseter spasm.

Mechanical ventilation was instituted, but relatively high pressures were required for ventilation and the end-tidal (ET) CO_2 rose to 9-10.5kPa (70-80mmHg). The patient's pulse was 70-80bpm and the blood pressure was stable at 100-120mmHg systolic. Nasopharyngeal temperature was 36.5°C.

Ventilation was increased but $ETCO_2$ increased to 12kPa (90mmHg). His temperature increased to 37.5°C over half an hour. The surgeon commented on a degree of muscle tension despite neuromuscular blockade with rocuronium. Anaesthesia was completed with intravenous propofol, the soda lime and circuit were changed, surgery was expedited and cold packs were applied to groin and axillae. The MH trolley was brought into theatre and senior help was summoned. The mixing of the first dose of dantrolene was commenced.

There was no further rise in $ETCO_2$ or temperature. An initial arterial blood gas showed supranormal oxygenation, respiratory acidosis with metabolic compensation and a lactate less than 2.0. The $ETCO_2$ at this point had fallen to 60mmHg (9kPa). The decision was therefore taken not to administer dantrolene and to wake the patient. He was extubated awake and kept in recovery for about 1 hour, where he complained of generalised muscle pains. He was then transferred to the High Dependency Unit. Initial bloods showed normal renal function and full blood count, with a creatine kinase (CK) of 25,000iu.l^{-1}. Urinary myoglobin was positive and he was treated with hydration and alkalinisation of urine. By the morning CK had risen to 30,000iu.l^{-1}, but settled over the following three days, after which he was discharged to the ward. He was given verbal and written instructions regarding malignant hyperthermia and referred for testing at a regional centre. Testing at 6 months post-event showed a strongly positive contracture test to both halothane and caffeine.

Also associated with MH are myotonia fluctuans, Multiminicore disease, Multiminicore myopathy, King Denborough syndrome and hypokalaemic periodic paralysis.[2]

CLINICAL FEATURES

Immediate changes

The presentation of MH is variable, but if a patient develops the signs of a hypermetabolic state or abnormal muscle rigidity under anaesthesia, then this should lead the anaesthetist to have a high index of suspicion.

Masseter spasm may be a herald of MH in patients administered suxamethonium, but not all patients with masseter spasm develop MH. For those who do not have masseter spasm, a rise in end-tidal CO_2 ($ETCO_2$) is normally the first sign. Tachycardia or tachyarrhythmia may be the first sign in the absence of $ETCO_2$ monitoring.

A rise in temperature typically occurs later, but at least two patterns are demonstrated, either an early rapid rise in temperature over a period of minutes, or alternatively a slow rising temperature which becomes apparent after about an hour. The temperature may rise more than 2°C per hour in fulminant MH.[2]

Other features of MH include:

- *Muscle rigidity* unaffected by neuromuscular blockade.
- *Cyanosis* develops with an increased oxygen extraction ratio. Oxygen consumption may increase up to threefold leading to cellular hypoxia despite a supranormal oxygen delivery. Increased end-tidal (i.e. alveolar) CO_2 leads to displacement of oxygen in the alveolus, as described by the alveolar gas equation (PaO_2 = PiO_2 - [$PaCO_2$/R]), reducing alveolar O_2 despite an adequate FiO_2.
- *Arrhythmias* – predominantly ventricular.
- Hypoxia, hyperkalaemia, metabolic acidosis and hypocalcaemia.

Late complications

Later complications are a result of rhabdomyolysis. Patients can go on to develop multi-organ failure as a result of a combination of rhabdomyolysis, electrolyte abnormalities and hyperthermia, leading to death.

Malignant hyperthermia may not be identified during a first anaesthetic and commonly presents during a second or third anaesthetic.

DIFFERENTIAL DIAGNOSES OF MALIGNANT HYPERTHERMIA

Other causes of a hypermetabolic state should be considered in the differential diagnosis:

- **Sepsis**.
- **Thyroid storm**.
- **Neuroleptic malignant syndrome** presents with hyperthermia and rigidity, typically developing hours to days after introduction of a neuroleptic drug. The pathogenesis is related to central dopamine handling rather than peripheral calcium channel effects. Treatment consists of a dopamine agonist (L-dopa or bromocriptine), but dantrolene may be required.

TREATMENT

A high index of suspicion for the diagnosis is important, as the management of malignant hyperthermia is dependant on early detection. On suspicion of MH, treatment should be instituted immediately, prior to confirmatory tests.

Effective treatment requires the immediate withdrawal of trigger agents (volatile agents), the administration of dantrolene, and the quick and effective actions of a well-trained team. Assistance is required for the management of such a rapidly deteriorating patient, including for the mixing of dantrolene, which is time-consuming.

Treatment falls into **immediate management** to halt the process and treat the immediate metabolic effects of MH, **ICU management** to continue resuscitation, manage the effects of rhabdomyolisis and observe for complications and **further management** aimed at investigation and advice for both patient and family.

Immediate management

Removal of trigger agents

Volatile anaesthesia should be discontinued and no further doses of suxamethonium should be used (particularly when masseter spasm is encountered at rapid sequence induction). A high fresh gas flow, preferably 100% O_2 should be used to flush the anaesthetic machine and ideally the breathing circuit should be replaced with a clean circuit. Anaesthesia will need to be maintained with intravenous agents (ketamine, thiopentone, propofol and opiates are all safe to use). The end of surgery should be expedited in order to focus on the management of MH.

Muscle relaxation

Non-depolarising muscle relaxants such as pancuronium or vecuronium are ineffective in reducing muscle contraction in MH as the pathology is intracellular. Dantrolene has an inhibitory effect on the ryanodine receptors on the sarcoplasmic reticulum of skeletal muscle, inhibiting calcium ion release. It is a specific antagonist to the MH process. Powder for reconstitution contains mannitol and sodium hydroxide to enhance solubility and increase pH. The Association of Anaesthetists of Great Britain and Ireland (AAGBI) guidelines suggest a dose of 2-3mg/kg initially, followed by 1mg/kg up to every 10 minutes as required to reduce muscle contraction.[13] It is time-consuming to mix dantrolene so it is advisable that this role is delegated.

In the absence of dantrolene, treatment will be predominantly supportive, removing trigger agents and concentrating on the cooling measures described below.

Cooling

Active cooling is likely to be necessary, particularly where dantrolene is not available, but vasoconstriction by excessive cooling of the peripheries should be avoided. Active warming devices such as hot air mattresses can be converted to cooling mode, cold intravenous fluids should be used and consideration should be given to cold bladder irrigation and cold peritoneal lavage, particularly if the abdomen is already open as part of the surgery. Cold packs can be used, but should only be used in areas of high blood flow where tissue damage is less likely (not around the peripheral limbs). Sites where cold packs are placed should be inspected and duration of application should be limited.

Hypoxia and acidosis

The patient should be hyperventilated to, as close as possible, a normal pH. An adequate FiO_2 to maintain good oxygenation, despite the high metabolic demands, is needed – 100% oxygen is advisable initially. Sodium bicarbonate helps treat the acidosis and enhances the solubility of myoglobin by forced alkaline diuresis.

Rhabdomyolysis

Adequate hydration and alkalinisation of the urine help to solubilise myoglobin released from skeletal muscle, reducing the risk of renal failure. Aim for a urine output >3ml.kg^{-1}.h^{-1} and a urine pH>7.0.[13]

Hyperkalaemia

Hyperkalaemia should be managed if serum potassium exceeds 6.5mmol/l or is felt to be contributing to arrhythmias.

- Polystyrene sulphonate resins (e.g. **calcium resonium**), 15g orally or 30g rectally, 6-8 hourly bind and remove potassium, but are relatively slow-acting in acute episodes.
- **Insulin** 15 units in 100ml of 20% **glucose** IV over 30-60 minutes drives potassium into the cells. (Roughly equivalent regimens using more or less concentrated glucose eg. 50ml of 50% glucose or 200ml of 10% glucose will have the same effect).
- **Bicarbonate** 50mmol IV leads to exchange of potassium for hydrogen ions across cell membranes and is particularly effective at reducing hyperkalaemia in the presence of acidosis.

- **Salbutamol** (a β_2-agonist) 5mg nebulised, 50mcg IV bolus or 5-10mcg/min IVI increases cellular uptake of potassium.
- **Calcium** 5-10ml of 10% calcium gluconate, or 3-5ml of 10% calcium chloride has a rapid onset, but a short duration of action, acting as a physiological antagonist to potassium.
- **Haemodialysis**, where available, may be required to clear potassium and for management of renal failure in some cases.

Cardiac arrhythmias

Sinus tachycardia with or without ventricular ectopic beats is common in MH, and may be the presenting feature. More serious arrhythmias can occur, especially VT and VF.

- **Procainamide** is useful for both ventricular and supraventricular arrhythmias and can be given IV at 25-50mg/min, up to a maximum of 1g. The ECG should be monitored for widened QRS and prolonged PR interval.
- **Magnesium sulphate** is useful for ventricular arrhythmias in a dose of 2g IV over 10 minutes.
- **Amiodarone** can be useful for both ventricular and supraventricular arrhythmias with a loading dose of 5mg/kg over 1 hour (roughly 300mg), followed by an infusion not exceeding 1.2g in 24h.

Calcium channel antagonists should be avoided in a MH crisis.

Disseminated intravascular coagulation

Conventional treatment with clotting factors (fresh frozen plasma, cryoprecipitate and platelets) as dictated by blood tests is used.

Management in ICU

Continuation of the treatments above is likely to be necessary, potentially including further doses of dantrolene for up to 24h. Care should be in a high dependency setting, where frequent monitoring can be continued, as well as intensive therapy. Reactivation can occur for up to 24h and active monitoring to detect this is necessary.

In addition to the above specific treatment, it is necessary to offer general supportive therapy and particularly to observe for and treat *renal failure* and *compartment syndrome*.

Compartment syndrome is a result of tissue damage in a limiting fascial sheath, with swelling and oedema leading to compression of the tissues and structures. High tissue pressures lead to further tissue necrosis and can obstruct blood flow to distal tissue.

- Common sites are calves and forearms.
- Presentation is often with intractable pain from tissue ischaemia (masked in unconscious patients).
- Other signs include
  - Tight tissues
  - Distal limb ischaemia
  - Absence of distal pulses
- Compartment pressures can be measured with a manometer, via a needle.
- Treatment consists of surgical fasciectomy

Further management

The patient should be referred to a specialist centre for MH testing if possible. If MH testing is not possible, the MH grading scale can be used to assess the likelihood of this having been a genuine episode of MH.[14] There is no strict cut-off to define the diagnosis, but, coupled with clinical judgement, the MH grading scale has been found to be useful. The scale is based on clinical and common biochemical markers and provides a likelihood range from almost never (0 points) to almost certain (50+ points). Marks are scored for rigidity, evidence of muscle breakdown, respiratory acidosis, temperature increase, cardiac involvement, family history and a miscellaneous section (acidosis and reversal with dantrolene). The validity of the scale has been questioned since the introduction of in vitro contracture testing into common use,[15] but no other clinical scale has been suggested as a replacement.

The patient and family should be counselled regarding the implications of MH. It is important that this is explained prior to discharge from hospital and this is the responsibility of the anaesthetic team.

With a diagnosis of MH there is a 50% chance of any parent, child or sibling having MH, and they should be appropriately counselled, and regarded as MH positive until negative testing is available. Aunts and uncles have a 25% chance and first cousins have a 12.5% chance of having MH. Accurate mapping in the absence of genetic testing can be difficult, but a detailed family history may help to elucidate the affected family. In the absence of testing, where there is a high suspicion of risk, the use of a volatile and suxamethonium-free anaesthetic should be seriously considered, and it would be wise to have a very high clinical suspicion for MH.

The gold standard test for MH is still the in vitro contractility test to halothane and caffeine. This requires a muscle biopsy to be taken under local anaesthesia (or using a trigger-free anaesthetic), six months after the event. Genetic testing is possible once the specific genotype is known from a family member. However, in order to definitively exclude MH susceptibility in a subject, in vitro contracture testing is still required.

ANAESTHESIA IN PATIENTS SUSCEPTIBLE TO MH

The key to anaesthesia in susceptible patients is to avoid the possible MH trigger agents, suxamethonium and volatile anaesthetics.

Due to the widespread use of volatile agents in theatres, it is important to ensure decontamination of the anaesthetic machine. A volatile-free machine can be kept in the theatre complex for such an occasion (and can also be used for critically ill patients in recovery), however, this is expensive and unnecessary. Alternatively, it is possible to ensure that the machine to be used is adequately cleared of volatile anaesthetic agents by flushing it through with 100% oxygen. The period required to flush the machine will depend on the machine used as well as the amount of componentry and circuitry changed. While the time required at 10 l/min may be as little as 5 minutes with some machines, after changing a large portion of the components, a minimum of 30 minutes seems wise, with 60 minutes of flushing appearing safe in almost all circumstances. If using a circle system, it is advisable to change the patient circuit and soda lime.

Black rubber absorbs anaesthetic vapours and should be changed to a fresh circuit prior to use for patients with known susceptibility to MH when possible, but a 20 minute flush with at least 8 l/min oxygen gives outflow concentrations of less than 5 ppm halothane in previously contaminated rubber circuits.[16] High fresh gas flows are required not only to flush the machine, but also to ensure volatile concentration doesn't subsequently rise. It should be noted that a recovery area with previously anaesthetised patients has been shown to have concentrations as high as 1 part-per-million halothane,[17] so complete exclusion of volatile is likely to be impossible.

Anaesthesia can be safely administered with intravenous agents and a non-depolarising agents such as vecuronium, pancuronium or rocuronium.

Where feasible for the planned surgery, spinal or regional anaesthesia are safe and appropriate techniques in patients who are MH susceptible, but safe emergency anaesthetic circuits and drugs should always be available for such a patient should general anaesthesia become necessary.

In addition to a trigger-free anaesthetic, it is important to monitor the patient carefully to detect possible activation of MH during anaesthesia or recovery. Monitors should include $ETCO_2$ during anaesthesia and temperature monitoring during anaesthesia and recovery.

A pre-prepared MH trolley (a station with the necessary equipment for dealing with MH, including dantrolene, sterile water, sodium bicarbonate and cooling packs) should be immediately available throughout the procedure and recovery. The duration of postoperative monitoring is controversial, however 1 hour in recovery appears to be a safe period, and for day surgery patients, a further one and a half hours prior to hospital discharge.

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Update in
Anaesthesia

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Anaesthesia for paediatric ear, nose, and throat surgery

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Summary

Children account for approximately one-third of all patients undergoing ear, nose, and throat (ENT) surgery. Procedures range from simple day-case operations, such as myringotomy, to complex airway reconstruction surgery undertaken in specialist centres. This article describes the anaesthetic management of some of the commonly performed paediatric ENT procedures, including adenotonsillectomy, oesophagoscopy, and middle ear surgery. Paediatric bronchoscopy has been dealt with in detail in an earlier article in this journal.[1]

ADENOTONSILLECTOMY

Tonsillectomy is one of the most frequently performed surgical operations in children. According to the Department of Health Hospital Episode Statistics (http://www.hesonline.nhs.uk), 25 000 tonsillectomies and 6500 adenoidectomies were performed in children under 15 years of age in England in 2003.[4] The tonsils and adenoids are lymphoid tissues forming part of the Waldeyer's ring encircling the pharynx. They appear in the second year of life, are largest between 4 and 7 years of age and then regress. Children with adenotonsillar hypertrophy can present with nasal obstruction, recurrent infections, secretory otitis media and deafness (secondary to Eustachian tube dysfunction), and obstructive sleep apnoea (OSA). Tonsillectomy is indicated in children with recurrent tonsillitis if they have had five or more episodes of sore throat per year because of tonsillitis, or if symptoms have persisted for at least 1 year and are disabling, that is, interfering with normal functioning (SIGN publication no. 34, available from http://www.sign.ac.uk). Other indications for tonsillectomy include chronic tonsillitis, peritonsillar abscess, and OSA. Adenoidectomy is indicated when there is evidence of enlarged adenoids causing nasal obstruction, OSA, or hearing loss. In the presence of OSA, adenotonsillectomy eliminates obstruction in 85 – 95% of children, yielding improvement of symptoms and quality of life.

Preoperative assessment

Preoperative assessment should elicit features of OSA, especially in the younger child, in whom obstructive symptoms rather than recurrent infections are commonly the indication for surgery (prevalence of OSA 1 – 3%).

Symptoms of OSA include heavy snoring, apnoeas, restless sleep, extended neck position during sleep, and daytime hypersomnolence. Over time, this can lead to neurocognitive impairment, behaviour problems, failure to thrive, and rarely cor pulmonale.

Children with severe OSA have a higher incidence of perioperative complications and may need postoperative HDU/ICU care. Specifically, they are at an increased risk of desaturation, laryngospasm, and developing airway obstruction during induction of anaesthesia.[2] They have increased sensitivity to the respiratory depressant effects of sedatives and opioids and a diminished ventilatory response to CO_2 compared with normal.[2,3] The overall incidence of postoperative respiratory complications in children with severe OSA is 16 – 27% compared with an incidence of 1% in children without OSA. Other risk factors for respiratory complications include age >3 years, craniofacial abnormalities, neuromuscular disorders, failure to thrive, and obesity.[3]

Preoperative investigations are not routinely indicated for patients undergoing adenotonsillectomy (NICE Guideline on Preoperative Tests, available from http://www.nice.org.uk). It is difficult to confirm the diagnosis and quantify the severity of OSA. The gold standard for diagnosis is nocturnal polysomnography, but there is a great deal of variability in scoring methods between different sleep laboratories, and the test is expensive to perform.

Recent studies suggest that overnight oximetry to score the frequency and depth of desaturation events may be useful in identifying patients with severe OSA.[4] In children with long-standing OSA, a full blood count will reveal polycythaemia and an ECG may show a right ventricular strain pattern.

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Anaesthetic considerations

The main areas of anaesthetic concern are airway management, provision of analgesia, and prevention of postoperative nausea and vomiting (PONV).

Airway management

Sharing the airway with the surgeon, remote access, and the need to prevent soiling of the respiratory tract are factors that need to be taken into consideration in airway management. Two techniques are commonly used: the tracheal tube and the reinforced laryngeal mask airway (LMA).[5,6] The advantages and disadvantages of these techniques are compared in Table1.

The tracheal tube provides a definitive airway, and a 'south-facing' RAE tube positioned in the midline provides good surgical access. The disadvantages of intubation are that muscle paralysis or a deep plane of anaesthesia are required, bronchial intubation or accidental extubation can occur with surgical movement of the neck, and there is variable protection against airway soiling. The dilemma of whether to extubate the patient when fully awake and able to protect their airway or still deeply anaesthetized to avoid a stormy emergence and bleeding always exists. The reinforced LMA offers a good airway with no soiling of the respiratory tract, avoidance of the use of neuromuscular blocking agents, smooth emergence, and airway protection until awake. To avoid soiling the laryngeal inlet, the LMA should be removed with the cuff still inflated. To ensure best surgical access, the smallest LMA for size should be used, and when positioned correctly, the cuff should not be visible once the Boyle-Davis gag has been opened to its fullest extent. An incorrectly sized LMA, or too large a blade on the mouth gag, can cause obstruction.

The main disadvantages of the LMA are that it does not offer the definitive airway provided by a tracheal tube and it may restrict surgical access in younger patients. However, with both the tracheal tube and the LMA, dislodgement or compression can occur during positioning of the mouth gag, and airway patency must be re-confirmed before surgery proceeds.

A postal survey of anaesthetic techniques used in paediatric tonsillectomy in the UK in 1996 – 7 suggested that only 16% of anaesthetists used the reinforced LMA routinely.[7] I.V. induction with propofol, tracheal intubation with succinylcholine, and spontaneous ventilation with isoflurane were the commonest anaesthetic techniques.[7] Concern about the danger of succinylcholine-induced hyperkalaemic cardiac arrest in children with undiagnosed muscle disease has led to a decline in the use of this drug for elective intubation. Alternative techniques for intubation include deep inhalation anaesthesia, combinations of propofol with a short-acting opioid, or the use of a short-acting non-depolarizing neuromuscular blocking agent during light anaesthesia.

Analgesia

Adequate postoperative analgesia is best provided with a combination of simple analgesics and small doses of opioids. Paracetamol[8] and NSAIDs have a morphine-sparing effect. The concerns around the potential for increased perioperative bleeding with NSAIDs have largely been discounted, with the exception of ketorolac, which should be avoided. Administering the simple oral analgesics before operation is safe and ensures effectiveness by the end of surgery.

Alternatively, the rectal route can be used after induction of anaesthesia. However, this route is less acceptable to many

Table 1. *Comparison of the LMA and the tracheal tube for tonsillectomy*

	LMA	**Tracheal tube**
Advantages	Straightforward airway	More secure airway
	No soiling of airway with blood	Good surgical access
	Smooth emergence	
	Paralysis not required	
	Airway protection until awake	
	Minimizes trauma to the airway	
Disadvantages	Less secure airway	Risk of airway trauma
	May impair surgical access	Oesophageal/bronchial intubation
		Requires paralysis
		Soiling of airway with blood
		Problems associated with extubation

patients and will not achieve therapeutic levels by the end of surgery in most cases. A single dose of dexamethasone 0.1 – 0.5 $mg.kg^{-1}$ has also been shown to reduce postoperative analgesic requirements, whereas local anaesthetic infiltration of the tonsillar bed has not been found to be superior to placebo. Regular doses of paracetamol and an NSAID after operation provide good analgesia.

Prevention of postoperative nausea and vomiting (PONV)

The incidence of PONV can be as high as 70% after adenotonsillectomy and a multimodal approach is indicated to combat this.

Minimizing starvation, avoiding the use of nitrous oxide (N_2O), and balanced analgesia with prophylactic administration of antiemetics reduce the incidence of PONV. A combination of ondansetron 0.1 - 0.2$mg.kg^{-1}$ and dexamethasone 0.1 - 0.5$mg.kg^{-1}$ (maximum 8mg) intraoperatively has been shown to greatly reduce the incidence of PONV.[9] Intraoperative fluid administration has also been shown to decrease the incidence of postoperative nausea. Rescue antiemesis can be provided by further doses of ondansetron with or without cyclizine 0.5 – 1$mg.kg^{-1}$ (up to 50mg).

Special considerations

Severe OSA

In general, sedative premedication and long-acting opioids are best avoided in patients with severe OSA. Inhalation induction is preferred, as airway obstruction commonly occurs during induction, and children with associated craniofacial anomalies may prove to be difficult to intubate.[2] Consideration should be given to the use of a small dose of fentanyl to supplement simple analgesia, as this is associated with less postoperative respiratory depression.

The incidence of complications varies with the time of day that the procedure is performed. Children undergoing surgery in the morning have fewer desaturations than those undergoing the same procedure in the afternoon. Close postoperative monitoring and the availability of an ICU bed is required.

Day-case tonsillectomy

Successful and safe implementation of day-case tonsillectomy requires careful patient selection. Exclusion criteria include age >3 years, significant co-morbidity, OSA, and living further than a one hour drive from the hospital or having no private transport. Thought also needs to be given to the risk of early haemorrhage and the management of postoperative pain and PONV.

The incidence of early postoperative bleeding is <1% and the majority of these occur within the first 4h after surgery. An extended observation period of 4 – 6h before discharge is therefore recommended; this limits surgery to morning lists. A multimodal analgesic and antiemetic regimen as previously discussed is very important, as the main reasons for overnight admission are PONV, pain, and poor oral intake.

Bleeding tonsil

Haemorrhage is the most serious complication after tonsillectomy and can occur within the first 24h (primary haemorrhage) or up to 28 days after surgery (secondary haemorrhage). In the National Prospective Tonsillectomy Audit (July 2003 – September 2004), the incidence of post-tonsillectomy haemorrhage patients was 3.5% and the overall rate of return to theatre was 0.9%. The incidence of primary haemorrhage was 0.6% and the majority of these occurred within the first 6h after operation. Factors influencing haemorrhage rates were age (lower rates in children than adults), indication for surgery (highest rates with quinsy and recurrent tonsillitis, lowest with obstructive symptoms), and surgical technique (higher rates with use of diathermy and disposable equipment, lowest with blunt dissection).

The anaesthetic considerations in bleeding tonsil include hypovolaemia, the risk of pulmonary aspiration (swallowed blood with or without oral intake), potential for a difficult intubation because of excessive bleeding obscuring the view with or without oedema after earlier airway instrumentation, a second general anaesthetic, and the stress to both child and parents. Blood loss is because of venous or capillary ooze from the tonsillar bed and is difficult to measure, as it occurs over several hours and is partly swallowed.

Excessive blood loss may lead to the child spitting blood. In these cases, the child is likely to be seriously hypovolaemic, anaemic, and potentially difficult to intubate because of poor visualization of the larynx. Tachycardia, tachypnoea, delayed capillary refill, and decreased urine output are early indicators of hypovolaemia, whereas hypotension and altered sensorium are indicators of advanced volume depletion. Preoperative resuscitation (guided by trends in monitoring) is essential, even if this requires the insertion of an interosseous needle. Induction of anaesthesia in a hypovolaemic child can precipitate cardiovascular collapse. Haemoglobin and coagulation variables should be checked. Blood and blood products should be immediately available and transfused as necessary. Before induction, in addition to the standard equipment, a selection of laryngoscope blades, smaller than expected tracheal tubes, and two suction catheters should be immediately available. Anaesthesia is induced once the child is haemodynamically stable. Preoxygenation and rapid sequence induction with slight head-down positioning of the patient ensures rapid control of the airway and protection from pulmonary aspiration. Consideration should be given to adopting the left

lateral position if bleeding is excessive. Controlled ventilation provides good conditions for haemostasis.

Fluid resuscitation and transfusion of blood and blood products should continue intraoperatively as necessary. Once haemostasis is achieved, a large-bore stomach tube is passed under direct vision and the stomach emptied. Neuromuscular block is antagonized and the trachea is extubated, with the child fully awake in the recovery position. After operation, the child should be monitored closely for any recurrence of bleeding.

OESOPHAGOSCOPY

Rigid oesophagoscopy is performed for the removal of an ingested foreign body. History of ingestion, dysphagia, and odynophagia are the usual presenting symptoms, whereas a previous stricture is a predisposing factor for obstruction. The commonest site of impaction of the foreign body is at the level of the cricopharyngeus muscle. Oesophagoscopy should be performed in all cases of suspected impacted foreign body to prevent complications of perforation, mediastinitis, and fistula formation.

Anaesthetic considerations include management of the shared airway and the risk of pulmonary aspiration or oesophageal perforation during the procedure. A rapid sequence induction protects against pulmonary aspiration and ensures rapid control of the airway. The tracheal tube should be secured on the left side to allow easier access for the endoscopy. Adequate depth of anaesthesia and muscle relaxation during the procedure are essential to reduce the risk of oesophageal perforation. Analgesia is provided by a combination of intravenously or rectally administered simple analgesics and a small dose of opioid. The patient is extubated when fully awake. If oesophageal perforation is suspected, oral intake should be withheld, IV antibiotics commenced, and the patient closely observed for features of mediastinitis, such as severe chest pain, pyrexia, and subcutaneous emphysema.

EAR SURGERY

The most common surgical procedures on the ear are those performed to treat otitis media and its complications. Otitis media is the second most prevalent illness of childhood. This is because of a combination of factors including Eustachian tube dysfunction and an increased susceptibility to upper respiratory tract infection (URTI) in early childhood. The short Eustachian tube in young children predisposes to reflux of nasopharyngeal secretions into the middle ear space and thus to recurrent infections. Oedema of the Eustachian tube mucosa secondary to recurrent URTI, and mechanical obstruction of the Eustachian tube orifice by enlarged adenoids, lead to a negative pressure in the middle ear and a transudative effusion (secretory otitis media). Children with otitis media present with deafness and complications such as perforation, ossicular chain damage, and cholesteatoma. Surgery is performed to improve hearing and to eradicate middle-ear disease.

MYRINGOTOMY

Myringotomy and insertion of pressure-equalizing tubes are used to improve middle-ear aeration and hearing in chronic otitis media. It is a short procedure performed as a day-case. The preoperative assessment should elicit features of URTI, as otitis media is associated with recurrent URTI and these children can consequently have increased airway irritability. A small percentage of this population may also display symptoms of OSA secondary to adenoidal hypertrophy. The anaesthetic technique usually involves the patient breathing spontaneously via a facemask or LMA, with the head positioned to one side. Mild postoperative pain can occur in up to 75% of patients, but this can be avoided with the preoperative administration of paracetamol, NSAIDs, or both.[10]

MYRINGOPLASTY, TYMPANOPLASTY, AND MASTOIDECTOMY

Children with complications of chronic otitis media need more complex ear surgery. Myringoplasty involves repair of a tympanic membrane perforation in a dry ear. Tympanoplasty is performed when there is extensive middle-ear damage and involves reconstruction of the tympanic membrane and the ossicular chain. The approach to the ear can be permeatal or postaural, the latter providing better surgical access. Two surgical techniques of tympanic membrane grafting are used, the underlay and the overlay. The underlay technique involves elevation of a tympanomeatal flap and placing the graft material underneath (or medial to) the eardrum.

The overlay technique involves stripping the lateral epithelium off the eardrum and placing the graft material on the outer side of (or distal to) the eardrum. Various graft materials may be used, the most common being temporalis fascia, tragal perichondrium, and fat.

Mastoidectomy is performed to eradicate chronic suppurative middle-ear disease. The anaesthetic considerations associated with these three procedures are similar; therefore, we shall described their anaesthetic management collectively.

Anaesthetic considerations

Typically, these procedures are performed in the older child or teenager and can be of prolonged duration. The main factors that have a bearing on anaesthetic management are the effect of N_2O on the middle ear, the need for a bloodless operative field, the use of facial nerve monitoring by the surgeon, and the high associated incidence of PONV.

As the relative solubility of N_2O in blood is 34 times that of nitrogen, it diffuses across into the non-compliant middle-ear

cavity much more rapidly than nitrogen can leave. This can lead to pressures as high as 350 mm H_2O within 30 min of commencing N_2O, especially in the presence of Eustachian tube dysfunction.[11]

Displacement of tympanoplasty grafts, worsening of deafness, rupture of the tympanic membrane, and increased PONV have all been associated with elevated middle-ear pressures. In addition, after discontinuation of N_2O, rapid re-absorption of the gas leads to negative pressures in the middle ear and this can lead to 'lifting off ' of the underlay tympanic membrane graft. As the middle ear remains open until the surgeon places the graft over the tympanic membrane, N_2O can be used up to 10 – 15min before graft placement and then discontinued. However, it may be best to avoid its use in middle-ear surgery completely.

Any bleeding during middle-ear surgery distorts the view through the operating microscope and can make the procedure difficult. Venous ooze can be minimized by a head-up tilt of 10° – 15° and ensuring unimpeded venous drainage. Epinephrine infiltration by the surgeon, relative hypotension (mean arterial pressure 10 – 20%, normal), and avoidance of tachycardia minimize arterial bleeding.

In its course through the temporal bone, the facial nerve runs through the middle ear in close relation to the ossicles and through the mastoid before emerging from the stylomastoid foramen. Therefore, it is vulnerable to damage during middle-ear surgery, especially as the disease process can distort the anatomical relationship of the nerve to the ear structures and make identification difficult. Intraoperative facial nerve monitoring is useful for identification and preservation of the nerve during ear surgery. A single dose of a short-intermediate acting relaxant can be used to aid tracheal intubation, its effects should have worn off sufficiently before the stage in the operation when facial nerve monitoring is required. However, it may be prudent to avoid the use of relaxants altogether by using other agents to facilitate intubation or by avoiding intubation. Whether using a tracheal tube or an LMA, the patient requires controlled ventilation for this procedure. Much of the surgery is performed using an operating microscope; therefore, if paralysis is to be avoided, a deep plane of anaesthesia is required to guarantee immobility. Controlled ventilation also allows control of the end-tidal CO_2, which helps to minimize bleeding.

The options for airway management are a tracheal tube or a reinforced LMA. The advantages of a tracheal tube over an LMA are a secure airway and ease of controlled ventilation, though a stormy emergence contributing to graft displacement is a potential problem. Smoother emergence can be ensured by tracheal extubation in a deep plane of anaesthesia. A reinforced LMA has the potential advantages of less airway stimulation and smooth emergence, but care must be taken to limit airway inflation pressures in order to prevent gastric distension during controlled ventilation.

For either technique and where available, maintenance of anaesthesia with propofol and remifentanil, or sevoflurane and remifentanil, offers many advantages. They allow controlled ventilation without neuromuscular blocking agents, thus permitting unimpeded facial nerve monitoring. Remifentanil provides a titratable degree of hypotension while maintaining a stable heart rate and provides excellent operating conditions. The use of TIVA is also associated with a lower incidence of PONV.[12]

Analgesia and antiemesis

A multimodal approach provides good analgesia and minimizes opioid-induced PONV. Oral paracetamol and NSAIDs given before operation are better accepted by the older child; alternatively, these can be given rectally or intravenously during surgery. As remifentanil has no residual analgesic effect after termination of the infusion, a small dose of morphine should be given 30 – 40 min before the end of the procedure to ensure adequate analgesia on awakening. A greater auricular nerve block has been shown to reduce postoperative opioid requirement. Postoperative analgesia is provided by regular, simple analgesics and small doses of opioids if necessary.

Routine prophylactic ondansetron and dexamethasone are indicated because of the emetogenic potential of middle-ear surgery. Avoiding prolonged starvation, adequate hydration, avoiding N_2O, use of TIVA, and balanced analgesia also help decrease PONV.

BONE-ANCHORED HEARING AID

The bone-anchored hearing aid (BAHA) is a surgically implantable system for the treatment of conductive deafness in children with chronic ear infections or congenital external auditory canal atresia who cannot benefit from conventional hearing aids. It allows sound to be conducted through the bone rather than via the middle ear, a process known as direct bone conduction. The procedure involves two short operations. Firstly, a titanium fixture is implanted into the mastoid bone and this over time integrates with the bone of the skull. Around 6 months later, at a second operation, an external abutment is placed over the fixture and this allows a sound processor to be connected.

The majority of children presenting for BAHA implant have associated congenital anomalies, the commonest being Goldenhar's syndrome (26%) and Treacher Collins syndrome (21%).[13] There is also a high incidence of congenital heart disease (19%) and craniofacial anomalies. The main anaesthetic

concern is an increased incidence of difficult intubation. In most instances, after inhalation induction, the airway can be safely and easily maintained using a reinforced LMA. However, equipment for fibreoptic intubation and appropriately trained staff should be available in the event of a need for intubation. Analgesia is provided with a combination of paracetamol, NSAID, and a small dose of opioid. Routine antiemetics are indicated, as PONV is common.

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Update in
Anaesthesia

The Transversus Abdominis Plane (TAP) block: Abdominal plane regional anaesthesia

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Summary

TAP block involves deposition of local anaesthetic agent into the fascial plane superficial to the transversus abdominis muscle. This technique can be used for any surgery involving the lower abdominal wall, including bowel surgery, caesarean section, appendicectomy, hernia repair, umbilical surgery and gynaecological surgery. The relevant anatomy is described, followed by detailed descriptions of the landmark and ultrasound-guided techniques.

Regional anaesthesia is a rapidly evolving subspecialty area. Over recent years there has been growing interest in abdominal plane blocks, with promising data emerging on efficacy. The TAP block allows sensory blockade of the lower abdominal wall via local anaesthetic deposition above the transversus abdominis muscle.

HISTORY OF THE TAP BLOCK

Abdominal field blocks and costo-iliac block have been used in anaesthesia for surgery involving the anterior abdominal wall for several decades. A technique involving multiple injections of local anaesthetic in the abdominal wall was used in the 1980s.[1] This technique was improved with a blind landmark technique, via the 'lumbar triangle of Petit'.[2] The clinical efficacy of the landmark technique and, more recently, ultrasound guided techniques have been investigated in several centres around the world.[3,4]

ANATOMY

Innervation of the anterolateral abdominal wall arises from the anterior rami of spinal nerves T7 to L1. Branches from the anterior rami include the intercostal nerves (T7-T11), the subcostal nerve (T12), and the iliohypogastric and ilioinguinal nerves (L1). These give rise to lateral cutaneous and anterior cutaneous branches as they become more superficial.

The intercostal nerves T7 to T11 exit the intercostal spaces and run in the neurovascular plane between the internal oblique and the transversus abdominis muscles. The subcostal nerve (T12) and the ilioinguinal and iliohypogastric nerves (L1) also travel in the

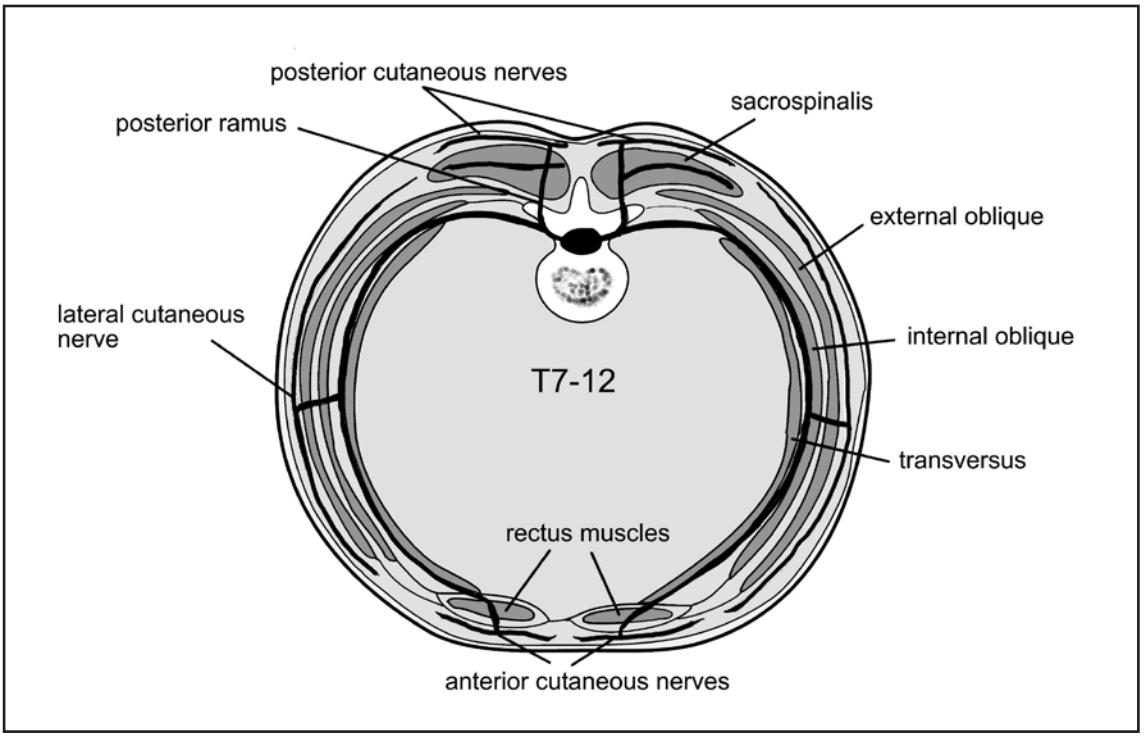

Figure 1. *Transverse section of the abdominal wall showing the path of nerves T7-T12 (left) and L1 (right) within the transversus plane*

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plane between the transversus abdominis and internal oblique, innervating both of these muscles. T7-T12 continue anteriorly from the transversus plane to pierce the rectus sheath and end as anterior cutaneous nerves. The thoracic nerves, T7 to T12, provide motor innervation to pyramidalis and the rectus muscle. These nerves have cutaneous branches laterally in the abdomen. T7-T11 provide sensory innervation to the skin, costal parts of diaphragm, related parietal pleura and the peritoneum. T7 gives sensory innervation at the epigastrium, T10 at the umbilicus, and L1 at the groin.[5,6]

CLINICAL APPLICATIONS

TAP block can be used for any surgery involving the lower abdominal wall. This includes bowel surgery, caesarean section, appendicectomy, hernia repair, umbilical surgery and gynaecological surgery. A single injection can achieve sensory block over a wide area of the abdominal wall. The block has been shown to be useful in upper abdominal surgery,[7] but the upper extent of the block and its use in upper abdominal surgery are

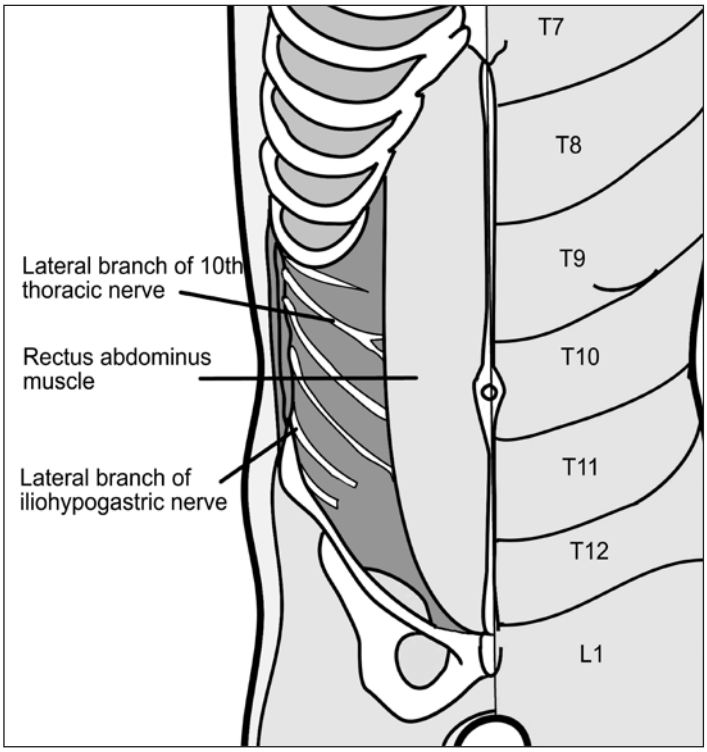

Figure 2. *Cutaneous nerve distribution and dermatomes of the abdominal wall*

controversial.[8,9,10,11] TAP block is particularly useful for cases when an epidural is contraindicated or refused.[3] The block can be performed unilaterally (eg. appendicectomy), or bilaterally when the incision crosses the midline (eg. Pfannenstiel incision). A single injection can be used, or a catheter inserted for several days of analgesic benefit. TAP block also has a role as rescue analgesia on awake postoperative patients who did not receive blocks prior to abdominal surgery.[12]

PERFORMING THE BLOCK

The principal of the block is to deposit local anaesthetic into the tissue plane between the internal oblique and the transversus abdominis. The two methods used include a blind technique, based on surface anatomy landmarks, and an ultrasound guided technique performed under direct vision. These methods are described below. The block takes up to 30 minutes to be effective so should be performed after induction and prior to surgery where possible. Intravenous opioid is required for skin incision and the early operative period as the block becomes established. TAP block for caesarean section is performed at the end of surgery and hence intravenous opioid will be required in the immediate post operative period while the block is becoming established.

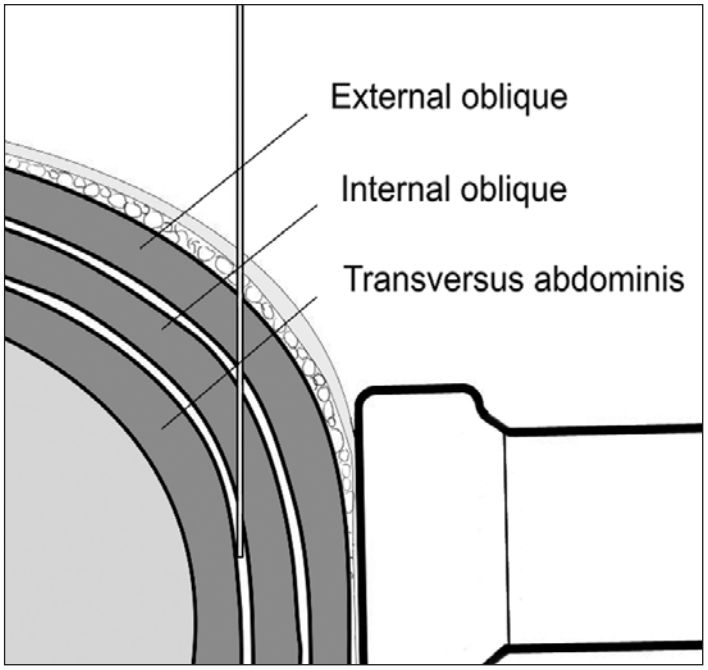

Figure 3. *Transverse section of the abdomen showing injectate deposition in the transversus plane*

Anaesthetic Agent

The volume of injectate is critical to success of TAP block. In an average sized adult 30ml of local anaesthetic should be used for unilateral block and 25-30ml used each side for bilateral block. Lignocaine, bupivacaine and ropivacaine have each been used for this block with success. Adequate volume is more important than using strong concentrations of local anaesthetic. The maximal safe dose of the chosen agent must be strictly adhered to. Examples of possible doses are shown in Table 1.

Landmark Technique

The landmark for palpation is the 'triangle of Petit' which lies above the pelvic rim in the midaxillary line (see Figure 4). The

Table 1. *Examples of appropriate drug selection for unilateral and bilateral TAP block based on patient weight*

	30kg	50kg	60-80kg
Unilateral (eg. Appendicectomy, hemicolectomy)	15ml: 0.5% ropivacaine or 0.375% bupivacaine	25ml: 0.5% ropivacaine or 0.375% bupivacaine	30ml: 0.5% ropivacaine or 0.375% bupivacaine
Bilateral - dose to each side (eg. LSCS, abdominal hysterectomy)	15ml: 0.25% ropivacaine or 0.25% bupivacaine	25ml: 0.25% ropivacaine or 0.25% bupivacaine	30ml: 0.25% ropivacaine or 0.25% bupivacaine

inferior border of the triangle is the iliac crest. The anterior border of the triangle is formed by the lateral edge of the external oblique muscle. The posterior border of the triangle is formed by the lateral edge of the latissimus dorsi muscle.[2,7,13] The triangle is tender to deep palpation in conscious patients.

The puncture site is just above the iliac crest and just posterior to the midaxillary line within the triangle of petit. A 24G blunt-tipped 50mm needle is inserted perpendicular to the skin, and a give or 'pop' is felt when the needle passes through the fascial extensions of the internal oblique muscle. The needle tip is therefore between the fascial layers of the external and internal oblique. Further advancement with a second 'pop' indicates that the needle has advanced into the fascial plane above transversus abdominis and, after aspiration, 25-30ml of local anaesthetic is injected.[2,7] There has been some controversy about seeking one or two 'pops' during the landmark technique of TAP block. Use of a 'two pop' technique is generally advocated and is supported by the cadaveric and imaging studies published to date.[14,15]

The triangle of Petit can be difficult to palpate, especially in obese patients. Rafi suggests a needle insertion point 2.5cm behind the highest point of the iliac crest when the triangle is not clearly palpable.[2] Requesting the patient to lift his head and shoulders from the supine position will contract the abdominal muscles and can assist palpation of the triangle.

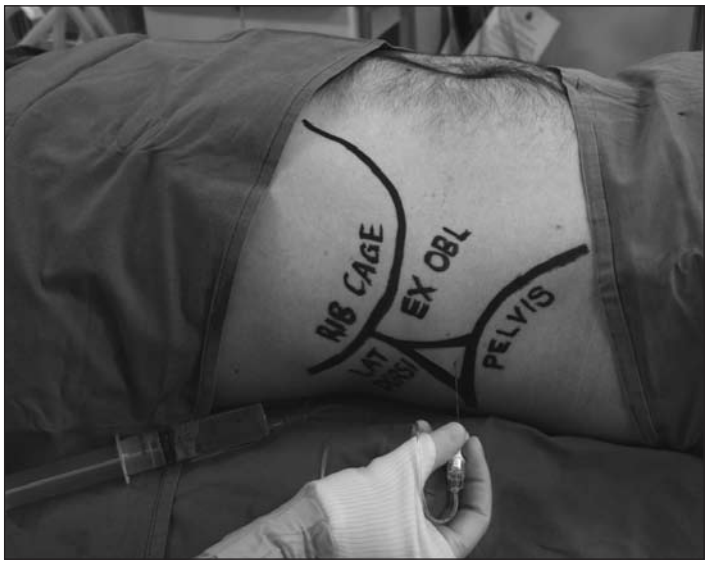

Figure 4. *Surface anatomy labelled for landmark insertion of TAP block in an adult male in the supine position*

Ultrasound Technique

The TAP block can be performed relatively easily with the use of ultrasound. A broadband linear array probe is used, with an imaging depth of 4-6cm. The ultrasound probe is placed transverse to the abdomen (horizontal plane) in the midaxillary line between the costal margin and the iliac crest. Three muscle layers are clearly seen in the image. A 100mm short bevel needle is used. The needle is inserted in a sagittal plane approximately 3-4 cm medial to the ultrasound probe. The point of needle insertion is closer to the probe in children and further from the probe for obese adults. For optimal imaging of the needle it should be held parallel to the long

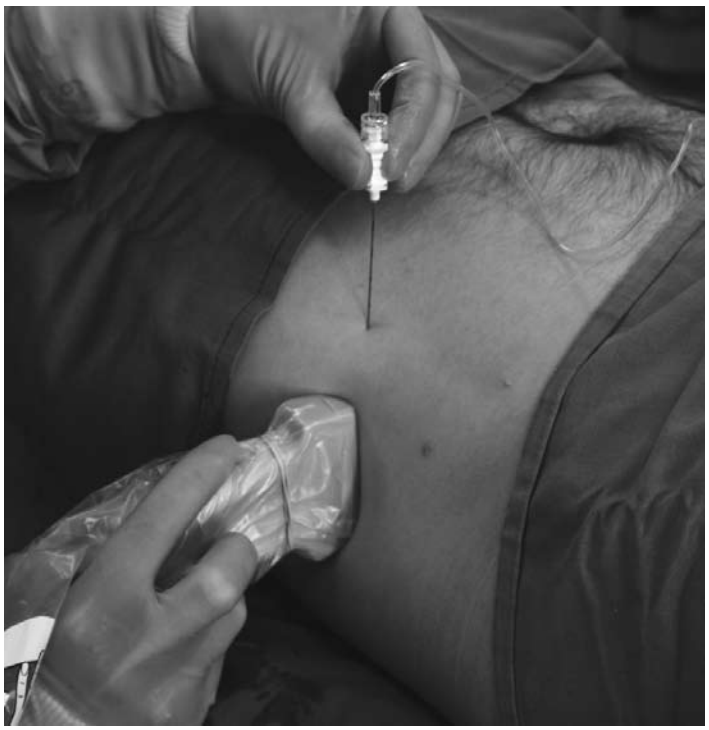

Figure 5. *Needle and probe position for ultrasound guided TAP block in an adult male in the supine position*

axis of the ultrasound probe (in plane technique). The probe is moved slightly anteriorly to image the skin puncture and superficial course, then gradually posteriorly to the midaxillary line position (shown in Figure 5), following the needle to the correct position in the transverse abdominis plane.

Real time ultrasound imaging allows observation of the needle passage through the skin and subcutaneous tissue, then through the external and internal oblique muscles. The needle tip is directed into the plane below the internal oblique and above the transversus abdominis muscle. A small volume of local anaesthetic (1ml) will be seen to open the plane between the two muscles and can be followed by insertion of the full dose of local anaesthetic. If the 1ml dose appears to be

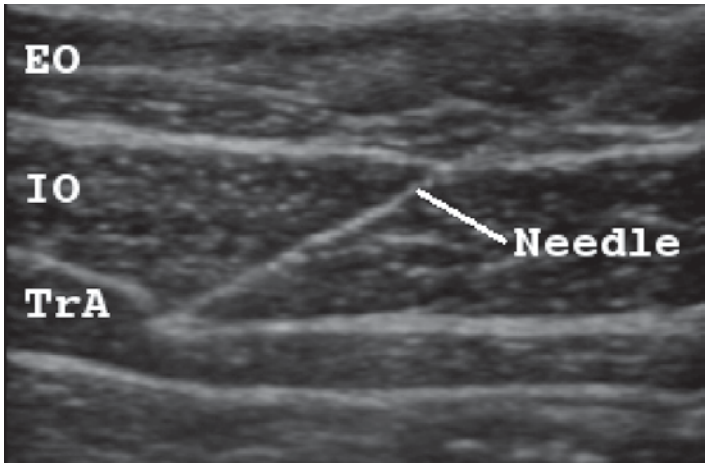

Figure 6. *Ultrasound image showing the muscle layers of the lateral abdominal wall with the needle seen positioned above the transversus abdominis muscle. EO: external oblique, IO: internal oblique, TrA: transversus abdominis*

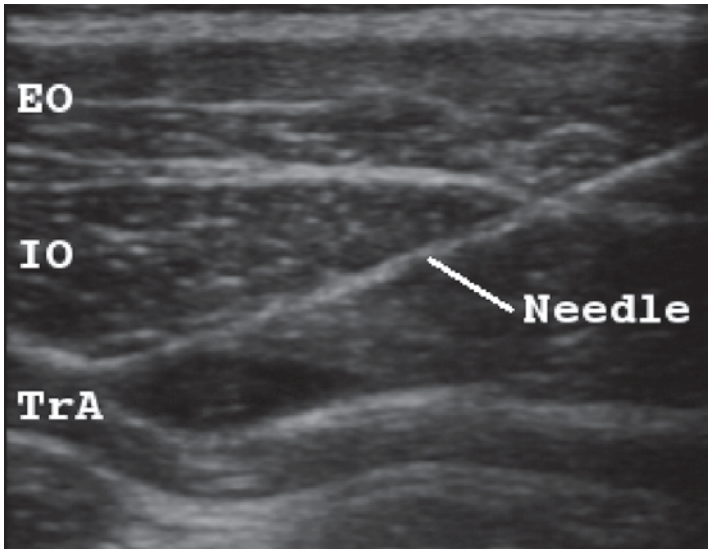

Figure 7. *Ultrasound image during initial injection of 5ml local anaesthetic. Injectate is in the transversus plane and alters the muscle layer appearance. EO: external oblique, IO: internal oblique, TrA: transversus abdominis*

within muscle rather than between them, needle adjustment is required. The local anaesthetic injectate appears hypoechoic (black compared to the muscle layers) on ultrasound imaging. When the needle tip is positioned correctly the injectate will be seen on ultrasound to spread out in the plane between the two muscles.[3] Ultrasound can also allow direct visualisation of structures in this region such as the deep iliac circumflex vessels and the iliohypogastric/ilioinguinal nerves.[16,17]

AREAS OF CONTROVERSY

There is debate in the literature regarding the extent of the sensory block achieved. Published investigators agree there is reliable block spread between L1 and T10 dermatomes. Initial publications found a block height from L1 to T7 could be achieved and hence the block was suitable for use in midline laparatomy.[9,11] Other investigators have found that the block does not reliably rise above the umbilicus and is therefore better suited to lower abdominal surgery only.[8,10] McDonnell and Laffey state that examining extent of the block prior to full spread could be misleading and measurement will be most accurate, when full block height has been achieved several hours after insertion of the block.[9] Some investigators have found the block height does not continue to extend over hours.[10] It may be that a different distribution of anaesthetic (and hence sensory blockade) occurs with the landmark technique compared to ultrasound-guided technique. Further anatomic studies are in progress to examine this issue.

EVOLVING DEVELOPMENTS IN TECHNIQUE

An alternative approach called the oblique subcostal TAP block has recently been described.[10] In this variation the ultrasound probe is held below and parallel to the costal margin, oblique to the sagittal plane. A 100-150mm needle is inserted at a position close to the xiphoid process and in plane to the ultrasound probe. The local anaesthetic is deposited between the transversus abdominis and rectus abdominis muscles, or between the rectus muscle and posterior rectus sheath (if there is no transversus at that level). The advantage of this approach is reliable spread of sensory block above the umbilicus (eg. for cholecystectomy).

LITERATURE REVIEW

In 2004, a report was published detailing a trial of the landmark TAP block procedure performed on both cadaveric specimens (methylene blue dye was used) and on healthy volunteers (radio-opaque dye with 0.5% lignocaine was used).[14] Cadaveric dissection revealed dye deposition in the transversus abdominis plane. CT imaging of live volunteers identified dye in the transversus abdominis neurovascular plane and pinprick testing indicated sensory block from L1-T8 dermatome.

Another cadaveric study was published by the same team of investigators in 2007.[15] This examined spread of methylene

blue dye injected via the angle of Petit using the landmark technique. The cadaveric dissection revealed reliable deposition of injectate into the transversus abdominis plane. In addition three healthy male volunteers were given a TAP block with radio-opaque dye and lignocaine to a final concentration of 0.5% and final volume of 20ml. 20 minutes after the block CT imaging demonstrated spread throughout the transversus abdominis plane. Sensory block assessment revealed a block from L1 to T7 which receded over 4 to 6 hours. A further three healthy male volunteers were given a TAP block of 1% lignocaine and radiopaque dye then MRI was performed at 1, 2 and 4 hours after the block. A gradual reduction in deposition of the injectate was demonstrated over time.

A small trial of TAP blocks performed on 12 open retropubic prostatectomy patients was reported in 2006.[18] The blocks were performed with 20ml of 0.375% bupivacaine to each side pre-operatively. Minimal morphine consumption was demonstrated (mean of 6.33mg at 48 hours with a range of 0-15mg). There were no adverse effects reported from the block.

In 2007, TAP block efficacy was examined in a randomised clinical trial of 32 patients undergoing large bowel resection via midline abdominal incision.[7] The patients were randomised to receive standard care (PCA, regular non-steroidal antiinflammatory drugs and paracetamol) or TAP block with the landmark technique (20ml 0.375% levobupivacaine). They found the TAP group had decreased visual analogue scale pain scores at emergence and at all times measured postoperatively up to 24 hours. There were no complications from the blocks and a high reported patient satisfaction level in the TAP group.

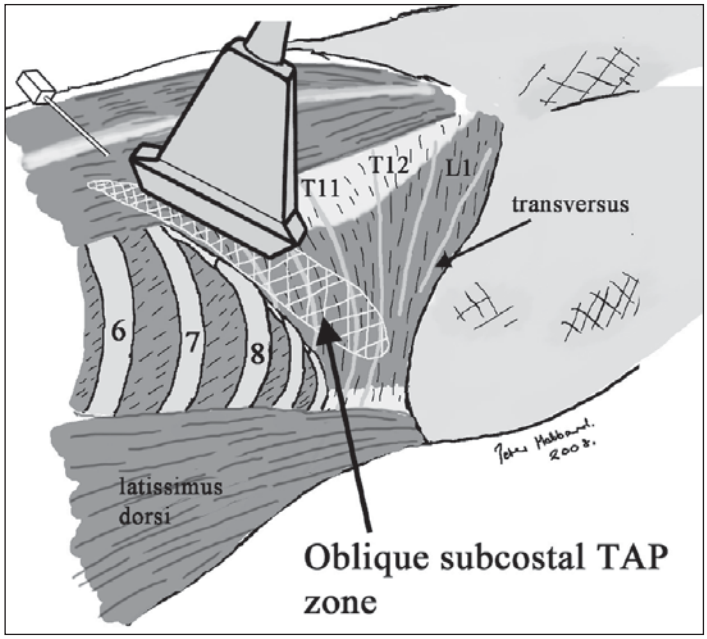

Figure 8. *Needle and probe position for ultrasound guided oblique subcostal TAP block (reproduced with kind permission from Dr P Hebbard)*

Another randomised clinical trial from the McDonnell team in 2008 examined TAP block efficacy after caesarean section delivery.[19] Fifty elective patients for caesarean section (via spinal anaesthetic and Pfannenstiel incision) were randomised to receive TAP block (landmark method) versus placebo in addition to standard anaesthesia (paracetamol, diclofenac and intravenous morphine). The TAP block was performed at the end of surgery using 1.5mg/kg ropivacaine (to a maximal dose of 150mg). A blinded investigator assessed patients at specific time intervals between 2 to 48 hours postoperatively. Results showed TAP block reduced visual analogue pain scores and mean total morphine requirements in the first 48 hours (18mg versus 66mg in the placebo group). There were no complications from the blocks.

A second publication on use of TAP blocks after caesarean section describes placement of TAP catheters under ultrasound guidance in three case reports.[16] Continuous infusions of 0.2% ropivacaine at 4ml/h was used for 72 hours. The reported benefits of the block included low pain scores, minimal use of supplemental opioid and absence of nausea and vomiting.

A recent case report describes a complication relating to the blind landmark technique for TAP insertion.[20] A TAP block was performed on a woman for abdominal hysterectomy (50kg in weight and 160cm tall). At laparotomy, approximately 50ml of fresh blood was found in the abdomen, due to needle perforation of the liver. The liver was found to be enlarged and reached the right iliac crest. Authors of the report recommend palpation of the liver edge prior to block insertion, especially in people of small stature.

DANGERS AND LIMITATIONS

Regional anaesthesia in general has a very low rate of serious complications. The risk in regional anaesthesia varies with the type and location of the block. The head and neck for example, are sites of higher complication rates compared to the abdomen. General risks of regional blockade include: needle trauma, intraneural injection, neural ischaemia, inadvertent intravascular injection, local anaesthetic toxicity, infection, and poor/failed block.[17] The general risks for regional blockade are applicable to the TAP block, however the site of injection for the TAP block is relatively low risk. This review found only one published report of complication from the TAP landmark technique (as already described)[20] and could not identify any published cases of complications from the ultrasound-guided TAP block.

The landmark technique relies on the 'pop' sensation which some clinicians believe is an imprecise sign.[3] The identification

of the landmarks is more challenging in the obese hence the risk of peritoneal perforation is probably higher. If anatomy is abnormal, such as hepatomegaly, there is risk of damage from the needle puncture. Ultrasound techniques are likely to improve the safety of this block as the needle passage and injection can be followed in real time, however this has not been scientifically tested to date. Some authors argue that peritoneal perforation with a small gauge sterile needle is not likely to be significant.[21]

FUTURE DEVELOPMENTS

Ultrasound guidance for performance of this block has become the method of choice where available. We can look forward to further data being published on effects of the block and likely a wider range of possible techniques, particularly for targeting the upper abdominal wall. The ease with which this block can be performed, an excellent safety profile to date, and outstanding clinical utility, will no doubt lead to increasing popularity and use of the transversus abdominis plane block.

ACKNOWLEDGEMENTS

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Update in
Anaesthesia

Management of acute cervical spine injury

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Summary

This article covers the aspects of acute cervical spine injury that are relevant to anaesthetists. The anaesthetist's involvement will range from participating in the resuscitation of patients with polytrauma to the provision of safe anaesthesia to allow surgical treatment for cervical spine or other injuries. The importance of early immobilisation is emphasised and strategies used to 'clear' the cervical spine are described.

INTRODUCTION

Between 2 and 5% of patients suffering from blunt polytrauma have a cervical spine injury. Cervical spine injuries tend to occur between 15 and 45 years and are seen more commonly in males (7:3). The most common level of fracture is C2 whereas dislocations occur most commonly at the C5/6 and C6/7 levels.[1]

The initial management of the polytrauma patient follows the Advanced Trauma Life Support (ATLS) practice of airway and cervical spine control, breathing and circulation. Assessment of injuries takes place initially in the form of a primary survey, during which time life-threatening injuries are excluded. This is followed by a secondary survey when a more detailed assessment of injuries is carried out, including spinal injuries. All polytrauma patients should be assumed to have a cervical spinal injury until proven otherwise; precautionary cervical spine immobilisation should be instigated for all patients at the scene of the injury by pre-hospital staff. By immobilising the spine immediately, major injuries can be treated at the scene, or on arrival at hospital, without the risk of disrupting an unstable cervical spine injury and causing secondary neurological injury.[2]

IMMOBILISATION OF THE SPINE

Until spinal injuries can be excluded or 'cleared' the spine must be immobilised and this can be achieved in a number of ways. However, all methods continue to allow varying degrees of movement. Soft cervical collars are the most inefficient and provide very little stability and therefore should not be used. Whereas the application of Gardner-Wells forceps can be considered the most effective technique it is rarely a practical solution in the acute setting. Two methods are in common use, compromising between simplicity of application and effectiveness: these are semi-rigid collars and manual in-line stabilisation (MILS). In the prehospital setting, MILS should be applied as an initial manoeuvre as the patient's airway is assessed and then, when available, a semi-rigid collar should be applied. Further stability is achieved by using sandbags or blocks on either side of the head, with two non-elastic self adhesive tapes strapped across the head and on to a rigid spinal board. Users should be aware of the disadvantages of semi-rigid collars (Table 1).

Laryngoscopy is more difficult with a semi-rigid collar in place. If laryngoscopy and intubation is urgently indicated the collar should be removed and MILS applied instead (Figure 1). During laryngoscopy MILS reduces cervical spine movement by up to 60%. An assistant squatting behind the patient applies MILS by placing his or her fingers on the mastoid processes and the thumbs on the temporoparietal area of the skull. The hands are then pressed against the spinal board and act to oppose movements of the head caused by the anaesthetist. Axial traction should not be applied because of the risk of exacerbating cervical spinal injuries. Until the spine is 'cleared' a log roll should be performed for any movement or transfer of the patient.[3, 4]

Table 1. *Disadvantages of semi-rigid collars*

Total immobilisation is not achieved
Increases the chance of difficult laryngoscopy
Can exacerbate cervical spinal injuries
Can cause airway obstruction
Can increase intracranial pressure (ICP)
Increases risk of aspiration
Increases risk of deep vein thrombosis (DVT)
May cause significant decubitus ulcers

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Clearing the cervical spine

The exclusion of spinal injuries or 'clearance' requires the exclusion of both bony and ligamentous injuries, and ideally requires a combination of clinical assessment and radiological investigation. Clinical clearance of cervical spine injury is difficult or impossible in patients who are unconscious (due to sedation, anaesthesia or head injury) or have distracting injuries to other parts of the body. Anaesthetists should understand the principles of clearing the cervical spine, since a proportion of patients cannot be clinically cleared for several days and prolonged cervical spine immobilisation (with its inherent risks) may be necessary.

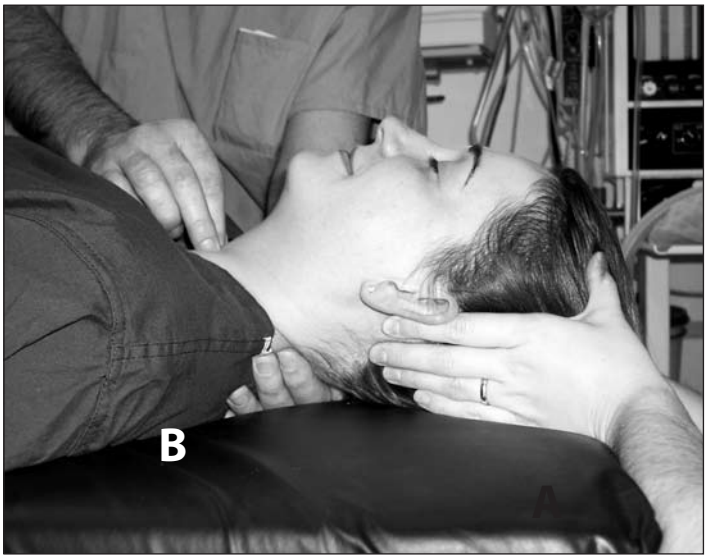

Figure 1. *(A) Application of manual in-line stabilisation (MILS). (B) Bimanual application of cricoid pressure*

Two sets of screening clinical criteria have been proposed prior to imaging the cervical spine, in an attempt to reduce the number of unnecessary X-rays. These are the Canadian C-spine rule and the National Emergency X-radiography Utilisation Study (NEXUS) criteria. Both are sensitive tools.[1] The NEXUS criteria include 'no evidence of posterior cervical tenderness', 'no history of intoxication', 'an alert patient', 'no focal neurological deficit' and 'no painful distracting injuries'. If all the criteria are fulfilled then the cervical spine can be cleared without the need for imaging.

If these screening tests indicate that radiological imaging is required, the strategy needed to clear the cervical spine differs depending on whether the patient is awake or unconscious. In the alert patient it is generally agreed that clearing the spine requires a 3-view plain X-ray series (lateral and AP cervical spine views with a 'peg view'), with a computerised tomogram (CT) for areas that cannot be visualised or are suspicious. If these are normal, but the patient is complaining of neck pain, a lateral cervical spine X-ray should then be performed in flexion and extension.

In the unconscious, since ligamentous injuries are difficult to exclude with accuracy using radiography, there is less agreement on the best method. Three options are available:

1. The cervical spine is left uncleared and the spine kept immobilised until the patient is fully conscious. Inherent with this method are the complications of immobilisation for any long duration, particularly decubitus ulcers.
2. Alternatively the patient has a combination of plain X-rays and CT scans to exclude bony injuries and, where available, this should be followed by magnetic resonance imaging (MRI) or fluoroscopy to exclude ligamentous injuries.
3. MRI may not be available and there are considerable practical difficulties associated with its use in unconscious critically-ill patients. A thin-cut CT scan is an alternative, including coronal and sagittal reconstruction of the entire cervical spine. Although less sensitive than MRI for the detection of ligamentous injury, CT is more practical and the number of unstable ligamentous injuries missed is extremely small.[1,3,5] It is worth remembering that the incidence of ligamentous injury without bony injury in blunt trauma is 0.02%.

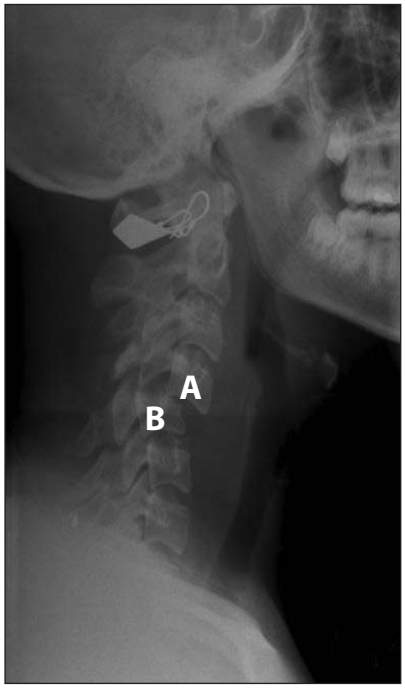

Figure 2. *Lateral cervical spine X-ray, showing fracture-dislocation of C4 (A) on C5 (B)*

AIRWAY MANAGEMENT

Patients may require airway instrumentation as an emergency (for airway obstruction, respiratory failure or as part of the management of a severe head injury) or later in their management as part of anaesthesia for surgical management of other injuries.

The extent to which the injured cervical spine can be safely moved is unknown. Therefore the main aim during management of the airway, in patients with potential cervical spine injuries, is to cause the least amount of movement possible. All airway manoeuvres will produce some degree of movement of the cervical spine, including jaw thrust, chin lift

and insertion of oral pharyngeal airways. Mask ventilation is known to produce more movement than direct laryngoscopy.

Most anaesthetists are comfortable with direct laryngoscopy and oral intubation and it is therefore the obvious first choice in establishing a definitive airway in the polytrauma setting. During direct laryngoscopy, significant movement occurs at the occipito-atlanto-axial joint. Manual in-line stabilisation (MILS) is used to minimise this movement. Previous anecdotal reports of the spinal cord being damaged following direct laryngoscopy in patients with unstable cervical spine injuries were based on weak coincidental evidence.[6] Therefore the technique of direct laryngoscopy with MILS is now an accepted safe technique for managing the airway in patients with potential cervical spine injuries. In addition the gum elastic bougie is a useful adjunct during direct laryngoscopy. It allows the anaesthetist to accept inferior views of the vocal cords thereby limiting the forces transmitted to the cervical spine and therefore movement. No particular laryngoscope blade has shown a superior benefit except the McCoy levering laryngoscope which will improve the view at laryngoscopy by up to 50% in simulated cervical spinal injuries. The McCoy is therefore an alternative to the Macintosh for those experienced in its use (Figure 4).

The laryngeal mask airway (LMA) or intubating laryngeal mask airway are both extremely useful in the failed or difficult intubation. The forces applied during insertion can cause posterior displacement of the cervical spine but the movement is less than that seen in direct laryngoscopy. In the 'can't intubate, can't ventilate' scenario there should be early consideration of the surgical airway or cricothyroidotomy. These techniques can produce posterior displacement of the cervical spine but this should not prevent the use of this life-saving procedure.

Nasal intubation has formerly been included in the Advanced Trauma Life Support course airway algorithm. However, the low success rate and high incidence of epistaxis and laryngospasm has resulted in this technique been superseded. Awake fibreoptic intubation has consistently produced the least amount of movement of the cervical spine in comparative studies. However in the acute trauma setting, blood or vomit in the airway may make the technique impossible. Further disadvantages include a relatively prolonged time to intubation,

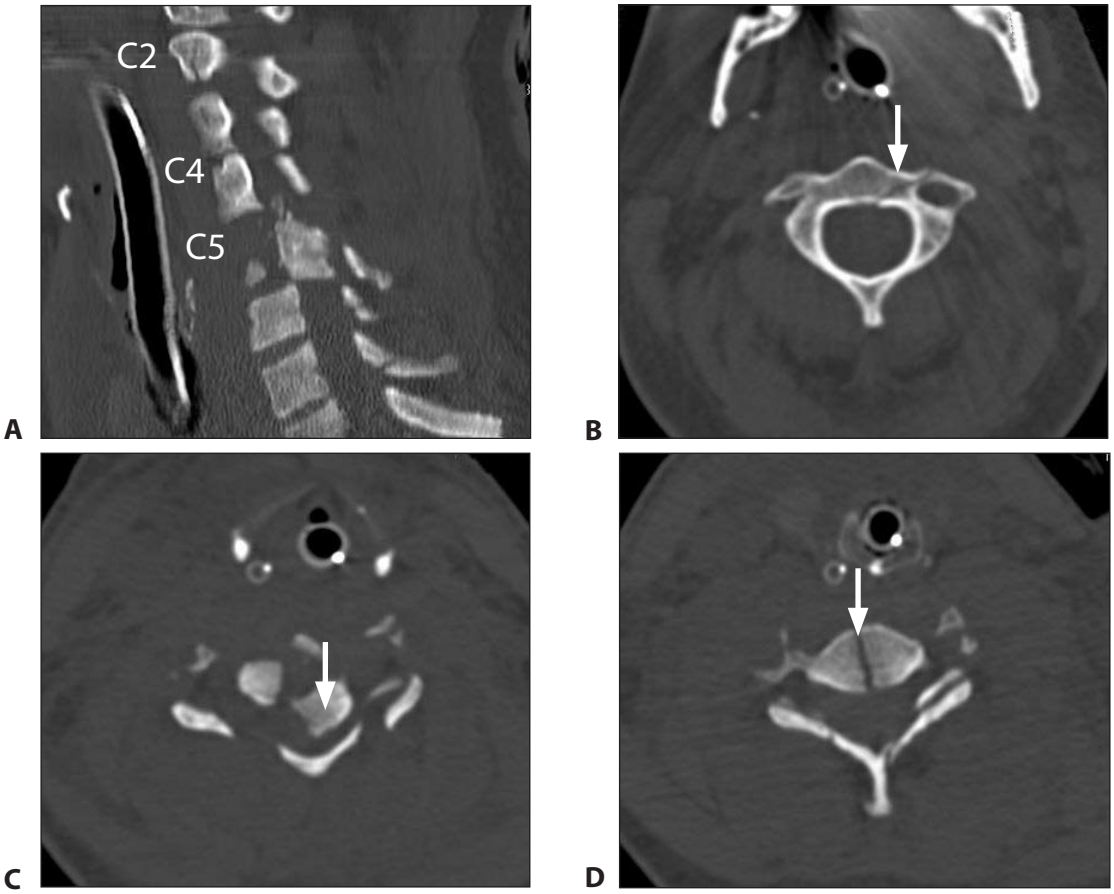

Figure 3. *Computed Tomography (CT) of the cervical spine. (A) sagittal reconstruction showing fractures at multiple levels; (B) transverse section fracture through the vertebral body of C2 to the left of the dens (arrowed); (C) transverse section - comminuted fracture with displacement of the left hemi-body of C5 into the spinal canal (arrow), presumably compressing the cord; (D) transverse section - midline fracture through the vertebral body of C6 (arrow), with bilateral fractures of the laminae of the vertebral arch*

risk of aspiration and, if gagging or coughing occur, an increase in the intracranial pressure (ICP). Despite theses concerns, for those anaesthetists with sufficient expertise and in the appropriately chosen patient, awake fibreoptic intubation is an option.[1,4]

Suxamethonium is safe to use in the first 72 hours and after 9 months following the injury. In the intervening period there is a risk of suxamethonium-induced hyperkalaemia due to denervation hypersensitivity and therefore should be avoided.

Spinal cord injury results in important pathophysiological consequences in various systems of the body that require appropriate treatment.

RESPIRATORY MANAGEMENT

Respiratory failure is common and pulmonary complications are the leading cause of death. The diaphragm (C3-C5) and intercostals (T1-T11) are the main inspiratory muscles. The accessory inspiratory muscles consist of sternocleidomastoid, trapezius (both 11th cranial nerve), and the scalene muscles (C3-C8). Expiration is a passive process but forced expiration requires the abdominal musculature (T6-T12). The abdominal muscles are therefore important for coughing and clearing respiratory secretions.

The severity of respiratory failure depends on the level and completeness of the injury. Complete dissection of the spinal cord above C3 will cause apnoea and death unless the patient receives immediate ventilatory support. For lesions between C3 to C5 the degree of respiratory failure is variable and the vital capacity can be reduced to 15% of normal. These patients are at risk of increasing diaphragmatic fatigue due to slowly progressive ascending injury resulting from cord oedema. This commonly results in retention of secretions and decompensation around day 4 post-injury, and intubation and ventilation is required. Where facilities are available some would electively intubate and ventilate patients in this group.

Figure 4. *The McCoy levering laryngoscope*

Initially the intercostal muscles are flaccid, allowing in-drawing of the chest during inspiration with a consequential compromise in respiratory function. This gives the characteristic appearance of 'paradoxical breathing' – on inspiration the diaphragm moves down, pushing the abdominal wall out and drawing the chest wall inwards. As the muscles become spastic, respiratory function improves, allowing potential weaning of the patient from the ventilator. It is important to remember that paralysis of the abdominal musculature means that in the upright position the diaphragm works in a lower and less effective position and so a supine position is preferred. Abdominal binders are an alterative. Patients with high cervical spine lesions have increased bronchial secretions, possibly due to altered neuronal control of mucous glands.

In general, the decision to intubate depends on several factors, including:[7,8]

- loss of innervation of the diaphragm,
- fatigue of innervated muscles of respiration,
- failure to clear secretions,
- history of aspiration,
- presence of other injuries e.g. head and chest injuries,
- premorbid conditions, especially respiratory disease.

CARDIOVASCULAR MANAGEMENT

Cardiovascular instability is particularly seen with high cervical cord injuries. At the time of injury there is an initial brief period of increased sympathetic activity resulting in hypertension, an increased risk of subendocardial infarction and arrhythmias. This is followed by a more sustained period of neurogenic shock, resulting from loss of sympathetic outflow from the spinal cord, which may last up to eight weeks. This is characterised by vasodilatation and bradycardia and tends to be seen only in lesions above T6. Bradycardia is caused by loss of cardiac sympathetic afferents and unopposed vagal activity and may lead to asystole. This can be treated with atropine.

Hypotension is due to the loss of peripheral vasoconstriction. The loss of sympathetic innervation to the heart means that increases in cardiac output are primarily achieved by increases in stroke volume. The initial treatment of hypotension involves intravenous fluid administration. Once preload responsiveness is lost, (i.e. the stroke volume cannot be increased further), then vasopressors will need to be commenced using either dopamine or norepinephrine, which are both α- and β_2-receptor agonists, thereby providing vasoconstriction, chronotropic and inotropic support to the heart.[7, 8]

The end-point of resuscitation is controversial. There is evidence that ongoing ischaemia and secondary spinal cord damage is successfully treated by raising the mean arterial pressure to 85mmHg for up to seven days.[9]

AUTONOMIC DYSREFLEXIA

This complication does not occur during the acute phase of spinal injury but is mentioned here for completeness. The condition can be triggered by various stimuli including surgery, bladder distension, bowel distension and cutaneous stimuli. Severe signs are seen with higher lesions, and it is rarely seen in patients with cord lesions below T10. The symptoms may start weeks to years following the spinal injury and include paroxysmal hypertension, headaches and bradycardia. Below the lesion cutaneous vasoconstriction, piloerection and bladder spasm may be seen. Above the lesion there may be flushing, sweating, nasal congestion and conjunctival congestion. The patient may complain of blurred vision and nausea.

If left untreated, complications include stroke, encephalopathy, seizures, myocardial infarction, arrhythmias and death. Management options include removal and avoidance of triggers such as the insertion of a urinary catheter. If surgery is planned, consider the use of spinal anaesthesia as this reliably prevents the symptom complex. Other options include increased depth of anaesthesia and vasodilators for the treatment of hypertension.[8]

VENOUS THROMBOSIS

The incidence is 40 to 100% in untreated patients with a spinal injury and pulmonary embolism is one of the leading causes of death in this group of patients. Prophylaxis must be started as soon as possible although there is no consensus as to exactly when or how this should be initiated. Treatment can be divided into two clear groups, pharmacological and non-pharmacological. Low-molecular-weight heparin is effective in preventing deep vein thrombosis (DVT), but is associated with an increased risk of haemorrhage within the injured spinal cord if given acutely. Therefore mechanical compression devices and graduated elastic stockings are often applied for the first 72 hours when the risk of DVT is low and anticoagulants considered thereafter. Prophylaxis should be continued for at least eight weeks.[7]

GASTROINTESTINAL MANAGEMENT

Bleeding due to stress ulceration should be prevented with an H_2 receptor antagonist such as ranitidine. Ileus and gastric distention can be treated with nasogastric suctioning and prokinetic drugs, e.g. metoclopramide or erythromycin.[8]

SPECIFIC TREATMENT

Different therapies have been tried, attempting to reduce the secondary neuronal injury due to cord ischaemia and inflammation. Although some have shown potential in animal studies most have not shown significant benefit in clinical studies. Only methylprednislone has shown any promise. There have been four randomised, controlled trials involving high dose methylprednisolone. The most discussed are the three National Acute Spinal Injury Studies (NASCIS), showing that administration of methylprednisolone in the acute phase showed a slight but significant benefit. However, this was at the cost of increases in the incidence of pneumonia and sepsis and the trials were criticised on several levels. Therefore methylprednislone is only a treatment option and cannot be considered a standard of care.

Early surgical decompression has been shown to be benefit in animal models of spinal cord injury. To date the evidence in humans is lacking, and the timing of surgical decompression remains a topic of debate and ongoing research.[10]

SUMMARY

The initial management of patients involved in blunt trauma follows the ATLS principle of airway and cervical spine control, breathing and circulation. The spine is immobilised as soon as possible to prevent secondary neurological injury. However, extrication collars should be removed and MILS applied prior to establishing a definitive airway, where this is indicated. Despite movement at the occipito-atlanto-axial joint, direct laryngoscopy with MILS is an accepted safe method to manage the airway in patients with potential cervical spine injuries. The gum elastic bougie and the McCoy laryngoscope are useful tools in this context. A cervical spine injury is likely to result in respiratory failure and cardiovascular instability, which may require ventilatory and/or inotropic support.

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Update in
Anaesthesia

Anaesthesia for foot and ankle surgery - general and regional techniques

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Summary

Regional and general techniques to provide anaesthesia and postoperative analgesia for foot and ankle surgery are discussed. Spinal anaesthesia is appropriate for shorter procedures, whereas general anaesthesia in combination with a regional technique is generally used for procedures over two hours. Ankle block and sciatic nerve block at the knee provide effective perioperative analgesia and both are described in detail.

INTRODUCTION

Anaesthesia for foot and ankle surgery can be provided by general or loco-regional anaesthesia and, given the peripheral site of surgery, a combination of both is generally well-tolerated. Regional anaesthesia confers excellent analgesia postoperatively, reducing the requirements for systemic analgesics. Anaesthetic techniques are usefully divided into those appropriate for surgery to the foot and those for surgery to the ankle.

If you are unsure about your choice of an appropriate regional technique, talk to the surgeon to clarify the site of surgery and incision, and the anticipated postoperative pain.

ANATOMY

The sensory supply to the foot and ankle is shared between branches of the femoral and sciatic nerves. The motor supply is almost exclusively from the posterior tibial nerve (a branch of the sciatic nerve).

Femoral nerve (L2-4)

The terminal branches form the saphenous nerve (L3-4), which supplies the skin over the medial malleolus, the medial aspect of the foot, with variable innervation to the head of the first metatarsal.

Sciatic nerve (L4-S3)

The sciatic nerve divides into the tibial and common peroneal nerves at a variable level between the buttock and popliteal fossa. Commonly this is about 6-10 cm proximal to the posterior knee skin crease, but may occur more proximally in up to 30% of patients.

The tibial nerve supplies motor nerves to the flexor muscles of the calf and foot, and divides into the posterior tibial and sural nerves. The posterior tibial nerve passes posteriorly to the medial malleolus, running just posterior to the tibial artery. It then divides into the medial and lateral plantar nerves in the foot, which supply motor innervation to the foot and sensory nerves to the internal structures of the foot and skin over the sole of the foot. The sural nerve supplies sensation to the lateral aspect of the heel and foot, with the calcaneal branch of the tibial nerve supplying the remaining parts of the heel.

The common peroneal nerve winds around the head of the fibula and then divides into

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Table 1. *Foot and ankle surgery*

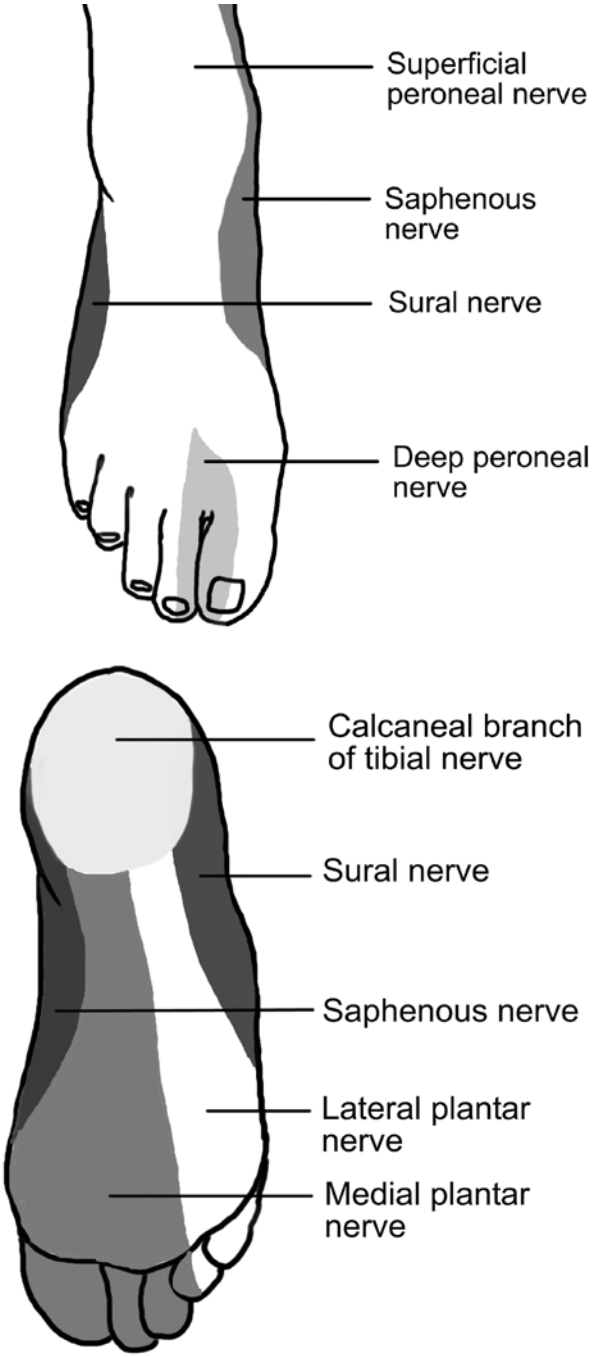
Site of surgery	Examples	Anaesthesic technique
Foot (i.e. midfoot, forefoot and toes)	• Scarf osteotomy (bunionectomy) • Weil osteotomy • Calcaneal fracture repair	General with ***ankle block*** or Spinal if surgery less than 1½ - 2 hours
Ankle	• Ankle arthroscopy • Arthrodesis (e.g. tibio-talar fusion) • Ankle replacement	General with more proximal block (e.g. ***popliteal block***) or Spinal if surgery less than 1½ - 2 hours

superficial and deep branches, supplying the dorsum of the foot and ankle, and the first web space respectively.

ANAESTHESIA FOR FOOT SURGERY

General points

- General anaesthesia (GA) with an ankle block, or spinal anaesthesia is usually appropriate. Where patient choice or medical co-morbidities dictate, spinal anaesthesia may be used for procedures lasting less than 1½ to 2 hours. Ankle block is usually combined with general anaesthesia, but may be used as the sole anaesthetic in patients who are unfit for GA. Be aware that the onset time is usually in excess of 30–40 minutes and that performing the block is painful and some form of sedation is recommended.
- Longer-acting local anaesthetic agents are preferred and generally provide analgesia for 12 hours or greater.
- The maximal safe dose for bupivacaine is 2mg.kg^{-1}. Where only lignocaine is available 3mg.kg^{-1} can be used; this can be increased to 7mg.kg^{-1} if epinephrine is added, but this is not advisable for ankle block due to the risk of causing ischaemia of the foot. Analgesia may be prolonged by the addition of clonidine to the local anaesthetic.
- Where available local anaesthetic catheters may be placed for longer term use in the postoperative period.
- Check the block prior to surgery, testing sensation to pinprick.
- If anaesthesia is inadequate, identify which nerve supplies the relevant area and repeat the infiltration of that nerve.
- Within the limits of maximal local anaesthetic doses, advise the surgeon to infiltrate locally during surgery to augment the block.



Figures 1 and 2. *Sensory innervation of the foot and ankle*

ANKLE BLOCK

Preparation

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

Technique - general

- Performing the block is painful so remember to inject the local anaesthetic (LA) slowly. Heating the local anaesthetic to body temperature may also help to reduce pain. Sedation is usually required.
- All five nerves can be blocked with the patient supine and the foot on a padded support. Some prefer to block the **posterior tibial** and **sural nerves** with the patient prone. To block the posterior tibial nerve in a supine position, externally rotate the leg, with the

knee slightly flexed – this allows the foot to be externally rotated.

- When a nerve stimulator is not available, a 23G needle, 3-4cm in length is appropriate for all injections. It is important always to aspirate prior to injection of local anaesthetic, to exclude intravascular injection.
- The authors feel that more use of a higher concentration of local anaesthetic (e.g. 0.5% bupivacaine) improves the success of the block.
- Most foot and ankle procedures require block of the **posterior tibial** nerve, since it also provides sensory innervation to most of the internal tissues of the foot. However, it is not always essential to block all four of the remaining nerves and your choice of injections should be tailored to suit the surgical procedure. If in doubt, ask the surgeon where his incisions will be and aim to cover these areas.
- Optimise analgesia with paracetamol and, where appropriate, a non steroidal antiinflammatory agent.
- Most surgeons use a thigh or calf tourniquet for these procedures and so additional intraoperative analgesia is often required to cover this. Tourniquet pressure is usually 100mmHg above the systolic blood pressure (generally 250mmHg is chosen). The risk of ischaemic damage is reduced if the tourniquet time is limited to 2 hours. The physiological response to tourniquet pain may make it difficult to assess whether the block is working and it is unwise to wake a patient relying totally on a block to provide effective analgesia.
- Most patients are positioned supine, with a wedge under the buttock on the operative side.

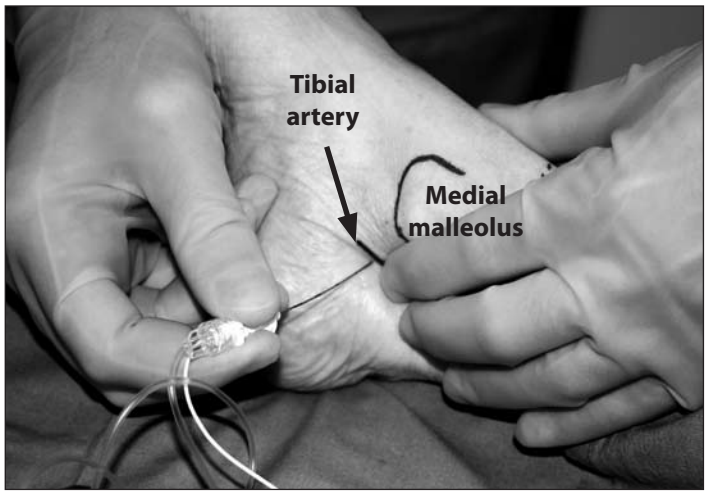


Figure 3. *The posterior tibial nerve is located immediately posterior to the tibial artery behind the medial malleolous*

Technique – specific nerve blocks

Always aspirate to exclude for vascular puncture before injecting local anaesthetic.

Posterior tibial nerve (this nerve can be located with a nerve stimulator – see below)

- Palpate the tibial artery just posterior and inferior to the medial malleolous. Insert the needle to pass 2-3 mm posterior to the artery (Figure 3).
- If paraesthesia is felt, inject 3-5ml LA. If not, advance to contact the tibia, withdraw 0.5cm and then inject 7-8ml LA.

Use of a peripheral nerve stimulator to locate the posterior tibial nerve.

- Of the five nerves supplying the operative field, only the **posterior tibial nerve** has a major motor supply. Where available, use of a peripheral nerve stimulator to locate this nerve behind the medial malleolus improves the success of the block (see Figure 3).
- Using a 50mm stimulator needle, look for flexion of the great toe or, less commonly, flexion of the other toes (Figure 4).
- Be aware that the threshold current for stimulation is usually higher than that achieved for other nerves and a higher value should be accepted. In practice any sort of stimulation indicates that the needle tip is close to the nerve, but it is worthwhile checking that the threshold is above 0.3mA (implying that the needle tip is not within the nerve). Be aware that conditions causing peripheral neuropathy (e.g. diabetes) may cause an abnormal or absent response to nerve stimulation.

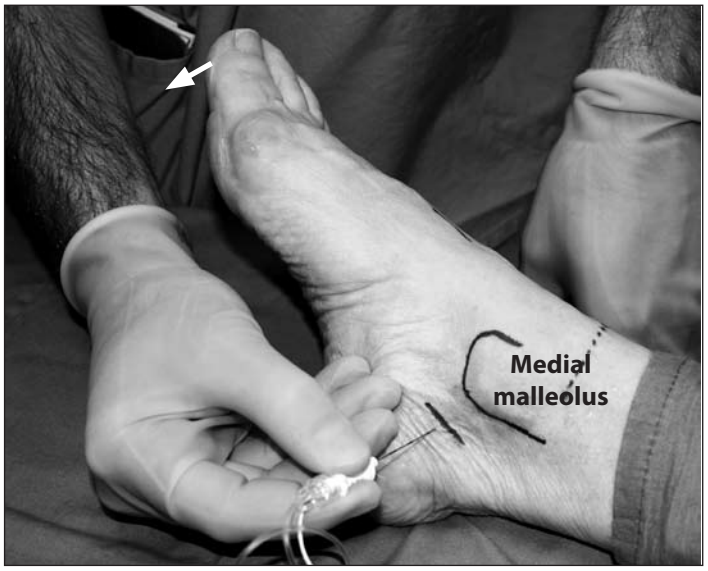


Figure 4. *Successful location of the posterior tibial nerve is indicated by flexion of the hallux (arrow)*

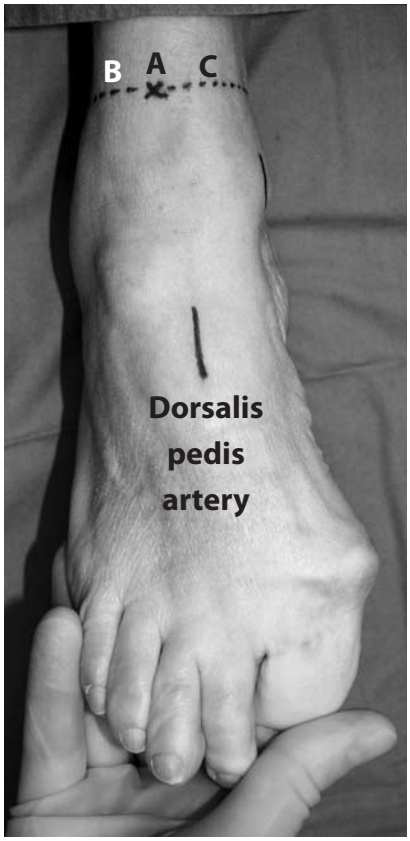


Figure 5. *(A) Needle insertion point for block of superficial peroneal nerve laterally (B) and saphenous nerve medially (C)*

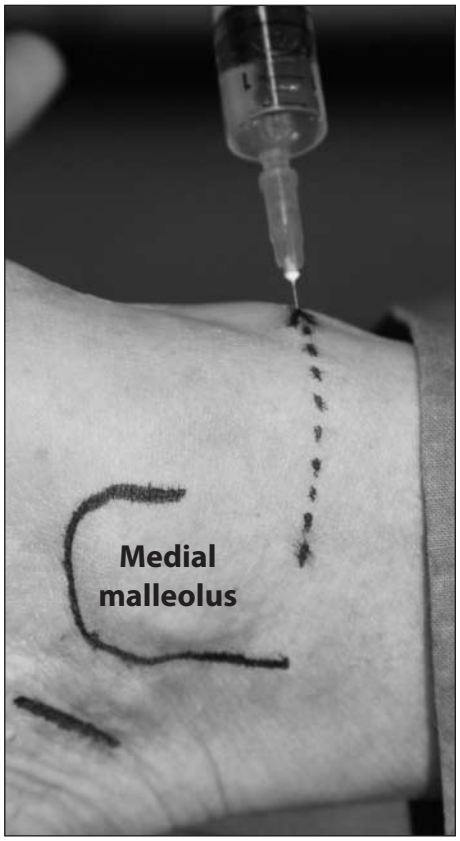


Figure 6. *Block of the saphenous nerve by infiltration in a band between the needle entry point and the superior border of the medial malleolus (shown by dotted line)*

- Use of a peripheral nerve stimulator reduces the volume of local anaesthetic agent required (usually 5ml for the posterior tibial nerve) and, in the authors' opinion, improves the success rate of the block (although some authors estimate the success rate at 90% without use of nerve stimulation). Block failure is overcome by supplementation with local infiltration provided by the surgeon.

Sural nerve

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

Superficial peroneal nerve

- Infiltration around the superficial peroneal and saphenous nerves can be performed from a single site. The needle is inserted subcutaneously at the most anterior point of the lower leg at the level of the cephalic borders of the malleoli (Figure 5).
- Turn the needle towards the lateral malleolus and inject 3ml LA in a subcutaneous band between the lateral malleolus and the anterior border of the tibia (Figure 5). This should reach all the branches of this nerve.

Saphenous nerve

- Withdraw the needle to just stay in the skin and turn the needle to point towards the medial malleolus (Figures 5 and 6).
- Infiltrate 3ml LA subcutaneously as the needle is advanced towards the medial malleolus (Figure 6). The great saphenous vein lies in this area, just antero-medial to the medial malleolus - in order to infiltrate around the vein without causing damage, it may be necessary to make a further skin puncture lateral to the vein.

Deep peroneal nerve

- Palpate the dorsalis pedis (anterior tibial) artery. Insert your needle superficial to the artery and pass it posteriorly to the left and then right of the artery, injecting 2ml LA deep to the fascia on each side (Figure 7).
- If the artery cannot be felt, insert the needle between the tendons of extensor hallucis longis (medially) and extensor digitorum (laterally), about one third of the way down the foot from the ankle to the toes. The extensor hallucis longis tendon is prominent on the dorsum of the foot during extension of the big toe.

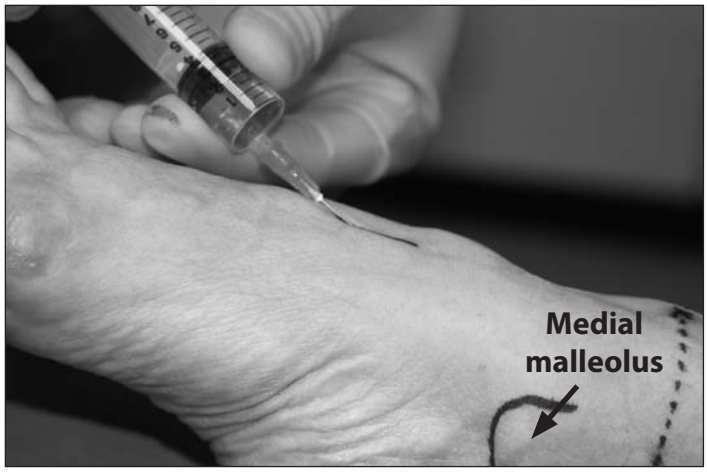


Figure 7. *Block of the deep peroneal nerve using injections on either side of the dorsalis pedis artery*

Cautions

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

Notes for specific surgical procedures

Scarf osteotomy

- Realignment osteotomy of the first metatarsal ('bunionectomy').
- Postoperative pain is considerable.
- The surgeon makes an incision along the medial aspect of the first matetarso-phalangeal joint (posterior tibial nerve, saphenous nerve and superficial peroneal nerve). Some surgeons make a second incision in the first web space (deep peroneal nerve) to release the sesamoid bones from the lateral ligaments. The sural nerve does not need be blocked.

Weil osteotomy

- Correction of claw toe with osteotomy of the metatarsal – often multiple.
- Block the same four nerves as above and, if the fourth and fifth metacarpal bones are involved, then a sural nerve block should also be performed.
- Injection of local anaesthetic into the webspaces disrupts the surgical field and should be avoided.

Metatarso-phalangeal fusion

- Usually the first metatarsal, for severe hallux valgus or pain due to osteoarthritis.
- Depends on the joints involved. Generally will require posterior tibial, saphenous, superficial and deep peroneal blocks. Sural block should be added if the 5th digit is involved.

Zadecks procedure

- Partial nail-bed excision.
- A simple ring block of the digit can be used. Vasoconstrictors should be avoided.

Excision of Morton's neuroma

- A neuroma in the webspace of the toe – often multiple.
- Although an ankle block will cover the incision(s), infiltration of local anaesthetic by the surgeon will be sufficient. Web space blocks by the anaesthetist are inappropriate since they disrupt the surgical field.

Note that for revision surgery, some surgeons prefer to make an incision in the sole of the foot – this painful approach would benefit from an ankle block.

ANAESTHESIA FOR ANKLE SURGERY

An ankle block is unlikely to provide complete analgesia for more proximal surgery. In addition, injection sites for an ankle block are likely to be at the site of surgical incision. If unsure whether to use an ankle or more proximal block, discuss the choice with the surgeon. Techniques to anaesthetise the femoral and sciatic nerves more proximally are appropriate (see Nerve blocks for anaesthesia and analgesia of the lower limb in *Update 11*, 2000, available at www.worldanaesthesia.org), however much of the motor weakness caused by a proximal sciatic nerve block can be avoided by blocking the tibial and common peroneal nerves in the popliteal fossa. This is a useful alternative for ankle (and some more proximal midfoot) surgery. These nerves can be blocked using a lateral or posterior approach. If surgery includes the medial side of the ankle, the saphenous nerve can be blocked just below the knee (see below).

SCIATIC NERVE BLOCK AT THE POPLITEAL FOSSA

Popliteal Nerve Block - Lateral approach

Indications

- Ankle and foot surgery.
- Provides anaesthesia for a calf tourniquet.

Anatomy

- The sciatic nerve lies lateral to the popliteal artery and vein (see Figure 8) and divides into the tibial and common peroneal nerves between 6 and 10cm above the politeal crease. In 70% of individuals, this division occurs within 10cm of the popliteal crease.

Preparation
As for ankle block.

Technique

- This technique requires use of a peripheral nerve stimulator and an appropriate (usually 100mm) short bevelled needle.
- The patient is positioned supine with the leg straight and the whole leg and foot exposed. The patient is usually mildly sedated.
- The groove between vastus lateralis and biceps femoris is palpated and a position in this groove, 8 cm proximal to the popliteal crease in identified (see Figure 9). The landmarks can be accentuated by asking the patient to perform a straight leg raise. After injecting a small amount of local anaesthetic subcutaneously, insert a 100mm stimulating needle in a horizontal plane, between your two fingers pressed into the groove. Aim to hit the femur within 2-3cm of the skin ('A' in Figure 8 and Figure 10). When you have identified the femur, withdraw the needle to the skin and redirect 30 to 45° posteriorly (towards the bed) – 'B' in Figure 8 and Figure 11. Advance slowly towards the sciatic nerve stimulating with a current of 1.5mA. Look for movement of the foot or toes:

Tibial nerve	Common peroneal nerve
Plantar flexion of ankle or toes	Dorsiflexion of ankle or toes
Inversion of foot	Eversion of foot

- The depth of the nerve is usually 5-7cm. Stimulation of the common peroneal nerve is usually encountered first, since it lies more laterally. Stimulation of either nerve is acceptable, since injection of a large volume of local anaesthetic should be sufficient to block both nerves, which lie in close proximity at this level. Aim to achieve a threshold for stimulation of 0.3 to 0.5mA. Inject 30-35ml local anaesthetic (e.g. 0.375% bupivacaine).
- Do not accept isolated twitches of the calf muscle.
- If no stimulation is achieved, first check all of your electrical connections. Then withdraw the needle and reinsert aiming a further 5-10° posteriorly. If this is still unsuccessful, repeat the process, realigning a further 5-10° posteriorly. Do not re-align anteriorly, since there is a high risk of hitting the popliteal artery or vein (see Figure 8).

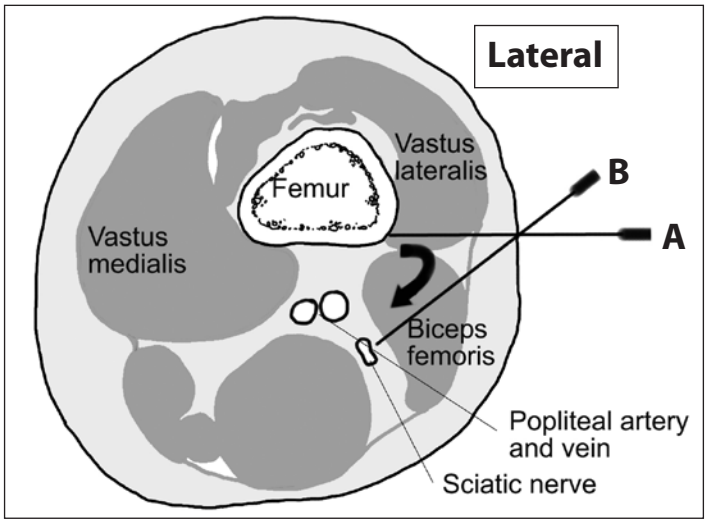


Figure 8. *Cross-section through the left thigh 8cm above the popliteal crease, showing relative positions of the sciatic nerve and the popliteal artery and vein*

- Remember that the skin over the medial malleolus is not covered by this block and is innervated by the saphenous nerve which can be blocked separately by a fan of local anaesthetic anterior and proximal to the medial malleolus (as described above) or at the knee (see below).

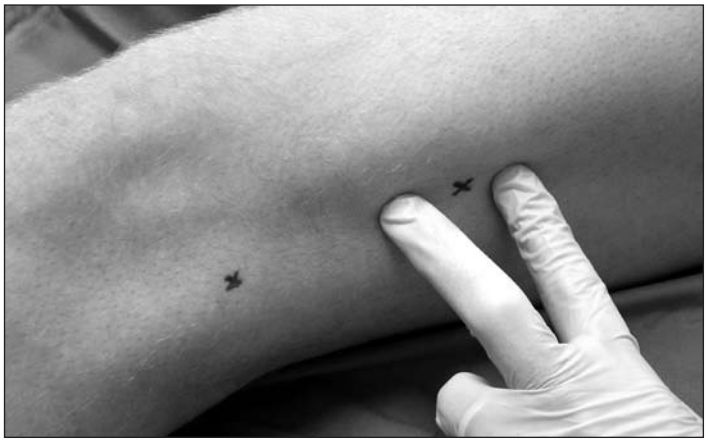


Figure 9. *Palpation of the groove between vastus lateralis and biceps femoris*

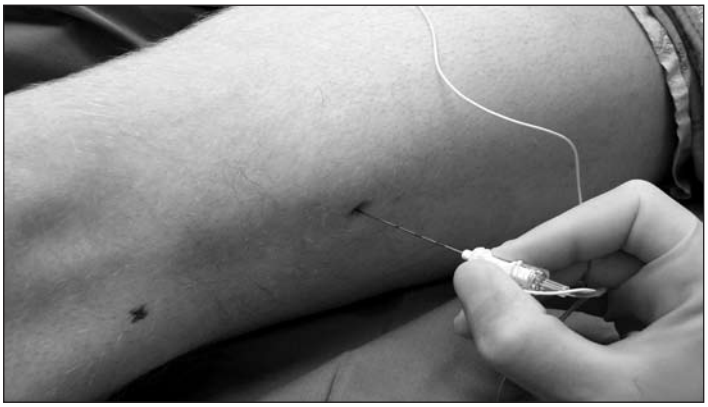


Figure 10. *Needle insertion in a horizontal plane to hit the femur*

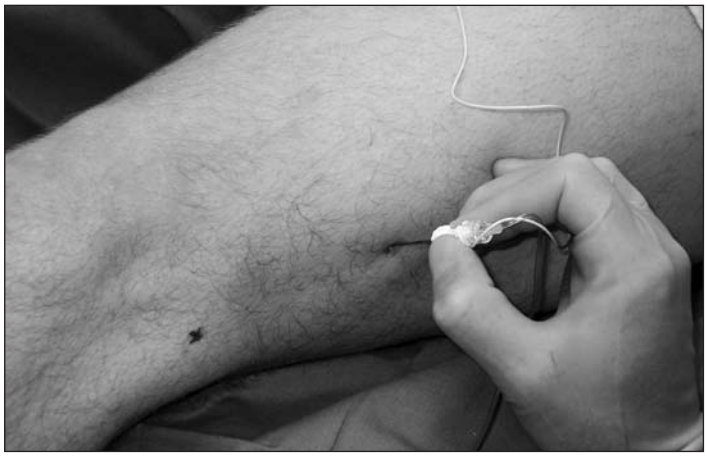


Figure 11. *The needle is withdrawn to the skin and then advanced at an angle of 30-45° posteriorly (towards the bed) until calf or foot movement is seen*

Potential complications

These should be discussed with the patient prior to attempting the block and include: vascular puncture, haematoma, nerve injury and failure of block.

Popliteal Nerve Block – Posterior approach

Anatomy

The popliteal fossa is bordered by the biceps femoris laterally, and by semimembranosis and semitendonosus medially, forming a triangle. The base of the triangle is formed by the popliteal crease. The sciatic nerve lies lateral to the popliteal artery and vein, dividing into the tibial and common peroneal nerves.

Technique

- The block can be performed with the patient in the prone position or supine, with the hip and knee flexed.
- With patient supine, the hip and knee are flexed to 90°, asking an assistant to support the lower leg.
- The borders and apex of the fossa are identified. A point is identified, 6-8 cm proximal to the popliteal crease, and 1 cm lateral to the midline. (The apex of the triangle is in the midline).
- Using a 50mm stimulator needle, the same end-point is sought as with the lateral approach.
- The same volume of local anaesthetic agent is used.

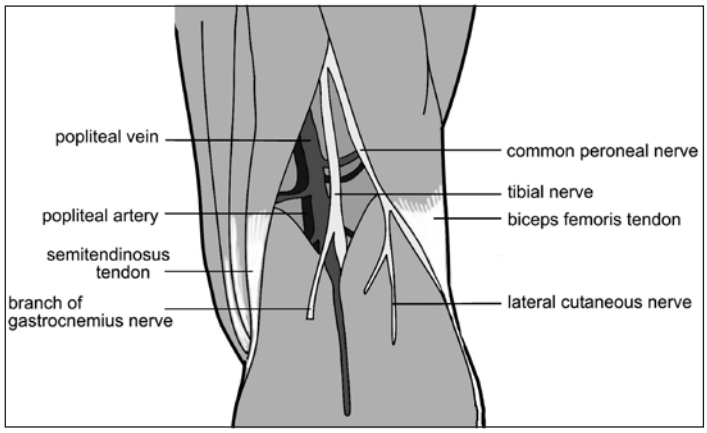


Figure 12. *The anatomy of the popliteal fossa (posterior aspect).*

Complications

As for lateral approach.

SAPHENOUS NERVE BLOCK AT THE KNEE

The saphenous nerve should be blocked for all surgery involving the medial ankle. A 25G needle is inserted 2cm medial to the tibial tuberosity. A fan of local anaesthetic agent (8-10ml) is infiltrated from this site to the posterior part of the medial tibial condyle.

Notes for specific surgical procedures

Ankle/lower tibial fractures

- Neurovascular compromise for fractures involving the ankle may necessitate emergency surgery. Ensure that if the patient is insufficiently starved, you take appropriate measures during induction and emergence from general anaesthesia.
- Ask the surgeon if compartment syndrome is a risk, since more proximal blocks of the sciatic nerve may mask symptoms and should therefore be avoided.

Ankle arthroscopy – diagnostic

- Intra-articular LA injection by surgeon is sufficient.

Ankle arthroscopy – interventional (may be done as an open procedure)

- Postoperative pain is significant and popliteal block is recommended.

SUMMARY

Surgery to the foot and ankle can produce extreme intraoperative stimulation and severe postoperative pain. Use of general anaesthesia, combined with an appropriate regional technique, guided by knowledge of the surgical technique and the nerve supply of the operative area, will facilitate a smooth perioperative course and good postoperative analgesia. Spinal anaesthesia is appropriate for shorter cases.

ACKNOWLEDGEMENTS

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FURTHER READING

Morphett S. Nerve blocks for anaesthesia and analgesia of the lower limb. *Update in Anaesthesia* 2000; **11**: 56-66. Available at www.worldanaesthesia.org