Chapter 26: Anaesthesia for otolaryngology

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The wider range and the increase in technical skills developed in otolaryngology over the last 20 years have greatly increased the demands made on the skill and knowledge of the anaesthetist. Anaesthetic problems and the very real influence of anaesthetic techniques on the ultimate outcome of the surgery, quite apart from morbidity and mortality resulting from ordinary anaesthetic complications, are now well recognized. There is no branch of surgery in which good communication and cooperation between surgeon and anaesthetist are more important than in otolaryngology. Central to all anaesthesia in otolaryngology is the control of the airway, and problems are particularly likely in operations around the nose, pharynx and larynx as a consequence of the anaesthetist having to share the airway with the surgeon. In a few cases the airway may be compromised by disease even before operation, and in such patients the problems will necessarily be compounded. In this chapter, both local and general anaesthetic techniques in the adult are discussed, while anaesthesia for infants and young children is dealt with in the relevant chapter in Volume 6. The aim of this chapter is not to try to teach the surgeon general anaesthetic techniques but to help him to understand some of the anaesthetist’s problems in this field.

Preoperative preparation and preanaesthetic medication

As in all surgery, it is important to evaluate the physical condition of each patient and to achieve the healthiest possible state before surgery, this being an important determinant of peri- and postoperative morbidity and mortality (Goldman et al, 1977). Patients for minor surgery who are judged fit at an earlier outpatient general medical examination can be admitted as day cases on the morning of operation or kept in overnight after operation. In all other cases, except where associated medical problems require earlier admission, patients should be admitted to hospital on the day before surgery.

Preoperative assessment

A careful assessment of general medical history, with particular attention to cough, sputum production, breathlessness or pain in the chest, is essential. A physical examination must pay particular attention to the cardiovascular and respiratory systems.

Cardiovascular disease

Pre-existing cardiovascular disease is seldom a contraindication to general anaesthesia and surgery, provided that the patient is treated to attain optimum fitness before operation. Avoidance of anxiety is particularly important and good general anaesthesia, after adequate premedication, may well be preferable to surgery under local anaesthesia. Adequate control of hypertension will ensure better stability of the blood pressure during anesthesia (Prys-Roberts, Meloche and Foex, 1971), and therapy should be continued up to the time of the operation. Cardiac failure should be adequately treated with digoxin and diuretics.

Patients with stable angina which is controlled by drugs are able to tolerate both surgery and anaesthesia well, as myocardial work is decreased under general anaesthesia.
However, patients with unstable, poorly controlled angina, and those who experience pain at rest, are in a high-risk category for any operative procedure. In the latter case, otolaryngology should, if possible, be postponed until the condition of such patients has been improved by drugs or by coronary artery surgery. Patients with angina should have their usual coronary artery dilator drug included in the premedication. Elective surgery should be postponed for 6 months after myocardial infarction.

A severe degree of heart block requires careful preoperative assessment by both anaesthetist and cardiologist, and the insertion of a pacemaker may be indicated before operation.

**Respiratory disease**

Upper respiratory tract infection is a contraindication to elective surgery as it is a cause of excessive secretions together with hyperaemia and irritability of the respiratory tract with a predisposition to laryngospasm and increased surgical bleeding. In the presence of an active chest infection, anaesthesia should also be avoided and appropriate antibiotics administered. Patients with a chronic productive cough should have physiotherapy, and bronchodilators may be required to improve the respiratory function; in these patients, premedication with promethazine will help to reduce the respiratory secretions and bronchospasm. However, some chronic infections, such as nasal discharge secondary to obstruction of the nasal passages, may never clear up without surgery. It is essential that all factors are taken into consideration before a decision on the postponement of an operation is made.

Asthmatic patients tolerate general anaesthesia well, as most inhalational agents are bronchodilators. The patient should use his usual bronchodilatory medication up to the time of operation, and premedication should avoid bronchoconstrictors such as morphine; phenothiazines or benzodiazepines are suitable, and an aminophylline suppository may also be helpful. Postponement of surgery will be indicated in cases where the patient suffers acute attacks of bronchospasm immediately before the operation or during the induction of anaesthesia.

**Diabetes**

Diabetics should be properly controlled before surgery, and this is generally possible except in emergencies. A suitable regimen is to omit the oral hypoglycaemic agent or insulin on the morning of the operation; then to perform a blood glucose estimation and to administer a 5% dextrose infusion with an appropriate amount of soluble insulin added to the solution. This intravenous fluid is run at about 1.5 mL/kg per hour with additional fluid, if required, being given as physiological saline. Many patients are able to return to oral feeding on the day of the operation, thus eliminating the need to add potassium to the infusion fluid. It is usually not necessary to switch patients from oral hypoglycaemic agents to soluble insulin before the day of operation. Indeed, it may not be necessary for patients for minor surgery to undergo any change in their treatment regimens. The blood glucose level should be checked immediately after an operation or intraoperatively during long procedures, and maintained at around 10 mmol/L. The subject of perioperative management of diabetes is fully discussed by Walts et al (19810 and Bowen et al (1982).
Concurrent drug therapy

Concurrent drug therapy should be noted and given proper consideration, particularly for those on beta-adrenergic blocking drugs, monoamine oxidase inhibitors, corticosteroids, contraceptive drugs or tricyclic antidepressants.

Beta-adrenergic blocking drugs

The administering of beta-adrenergic blocking drugs, which are widely used in the treatment of hypertension and angina, should be continued up to the time of the operation. It may be advisable to substitute short-acting agents for long-acting members of the same group but, on the whole, problems are not usual when combined with modern anaesthetic agents. If necessary, adverse effects of beta blockade can be controlled by drugs producing inotropic and chronotropic stimulation by way of alternative pathways.

Monoamine oxidase inhibitors

The use of pressor amines will be contraindicated in the presence of antidepressant monoamine oxidase inhibitors as the combination will produce a dangerous hypertension. This antidepressant therapy would also preclude the use of nasal decongestants such as ephedrine and phenylephrine. Narcotics, particularly pethidine, can produce dangerous side-effects such as excessive respiratory depression, hypo- or hypertension, sweating, nausea and collapse. In addition, any drug metabolized by the liver will be potentiated. If possible, general anaesthesia should be avoided for 3 weeks after stopping these drugs.

Corticosteroids

Patients on prolonged steroid therapy may need steroid cover during operation and in the immediate postoperative period. Patients undergoing minor surgery should either not be given any steroid cover, or be given a single dose of hydrocortisone hemisuccinate along with their premedication or at the time of induction of general anaesthesia.

Contraceptive pill

Patients on contraceptive pills should probably stop taking these tablets for at least one complete month before surgery; but for emergency operations, prophylaxis with subcutaneous heparin (Minihep) or dextran infusion should be considered to reduce the risk of postoperative venous thrombosis.

Pregnancy

Pregnancy in the first or last trimester is usually considered a contraindication to elective surgery. There may be a small increase in the risk of miscarrying in the early stages of pregnancy and, if a miscarriage should occur, the operation will certainly be blamed. In the final weeks of pregnancy, increased intra-abdominal pressure may lead to respiratory embarrassment, and the risk of gastric regurgitation will be increased, with very serious consequences should aspiration into the respiratory tract occur. In the middle trimester, there is no contraindication to general anaesthesia.
Preoperative investigations

Routine investigations include haematological and electrolyte screening and, at the same time, blood can be taken for grouping and cross-matching if significant operative blood loss is anticipated.

When anaemia is detected, the cause should be sought and treated. Patients originating from Africa, the Middle-East, the eastern Mediterranean, India and the West Indies should be screened for sickle-cell disease. Patients with the homozygous form (90% of haemoglobin in the abnormal sickle HbS form) run a very high risk of haemolysis and multiple infarctions under general anaesthesia, as the abnormal HbS forms crystals when oxygen tension is reduced. A haematological opinion should be sought before surgery, and the latter should be performed under local anaesthesia, if possible. Patients with the heterozygous form, the so-called sickle-cell trait (not more than half the haemoglobin in HbS), are able to withstand both the operation and the anaesthesia well, provided that they are in optimum condition before surgery and that dehydration and hypoxia (both global and regional) are avoided.

If the patient gives a history of abnormal bleeding and disease is suspected, and if platelet count or bleeding, clotting or prothrombin times reveal any abnormality, then consultation with a haematologist may be necessary.

Diuretics taken over long periods may produce electrolyte disturbances, particularly hypokalaemia; and a serum potassium level of less than 3 mmol/L should be treated before surgery.

Serial blood glucose estimations are required in diabetic patients.

Urine examination is carried out routinely and, if there is any doubt about renal function, particularly in patients requiring hypotensive anaesthesia, plasma creatinine levels should be checked.

Elderly patients, and all subjects with a history suggestive of cardiovascular disease, should have an ECG and chest X-ray to check for myocardial ischaemia, incipient failure and cardiac enlargement. In other cases, a chest X-ray is of questionable value and greater emphasis should be laid on a physical examination of the respiratory system. Other special investigations, such as thyroid and respiratory function tests, should be carried out as indicated.

Preoperative medication

Sedative drugs

A careful and reassuring explanation, with respect to the operation itself, the method of inducing anaesthesia and the immediate postoperative period, is more likely than any sedative drug to dispel the apprehensions of the patient. This information should be given well in advance of surgery and can be repeated with advantage on the night before the operation.
In the case of the very anxious patient, reassurance alone may not be sufficient and sedation may be required from the time of admission to hospital - diazepam 2-5 mg thrice daily being suitable. A good night's sleep before surgery should be the aim, and many patients require a sedative such as nitrazepam 5-10 mg. Patients who are accustomed to taking night sedation should be given their usual drug in a slightly increased dose. The elderly are not usually apprehensive, and a preoperative explanation, together with reassurance, is frequently all that is required.

Various premedicants can be given 1-2 hours before surgery, depending on the patient's condition and the anaesthetist's individual preference. Preoperative pain is unusual in elective otolaryngology and there is no rational requirement for the traditional opiates such as morphine or papaveratum; an oral anxiolytic drug is usually adequate and diazepam 0.2 mg/kg has become popular in recent years.

Anticholinergic drugs

The use of anticholinergic drugs, such as atropine, has been questioned (Mirakhur et al, 1978). These drugs produce subjective discomfort with a dry mouth and throat and loss of visual accommodation. Atropine is usually omitted before the induction of controlled hypotensive anaesthesia because of the potential problem of tachycardia. If necessary, such drugs can be given during operation; however, if a dry mouth and throat are required during surgery, preoperative IM atropine is more effective than intraoperative IV administration. When atropine is omitted, bradycardia is more likely to occur during laryngoscopy, particularly with the use of the suspension laryngoscope, and during application of topical anaesthesia to the larynx; so careful monitoring and readily available intravenous atropine are necessary. Glycopyrrolate (5-10 μg/kg IM or IV) is an alternative to atropine, providing both a better drying effect and more cardiostability (Mirakhur and Dundee, 1983).

Prophylactic antibiotics

Bacteraemia is common during any oral operation, including endoscopies (Bayliss et al, 1983). Patients with congenital heart disease or valve lesions, who run the risk of bacteraemia during surgery, require antibiotic cover before and during operation. Amoxycillin 20 mg/kg to a maximum of 1 g IM at the time of premedication or, more logically, IV during induction, followed by a single oral or IM dose of amoxycillin 10 mg/kg (to a maximum of 500 mg) 6 hours later, is recommended. Patients with prosthetic valves or those who have had endocarditis are at special risk, and they should be given gentamicin in addition to amoxycillin. Patients who are already taking penicillin, or who are allergic to penicillin, can be given oral erythromycin (20 mg/kg) 1 hour before operation and half that dose 6 hours later (Simmons et al, 1982).

Prophylactic anticoagulants

The risk of venous thrombosis is less following operations on the head and neck than it is compared with some other sites. Nevertheless, patients having prolonged surgery with extensive dissection, or who are otherwise especially at risk, should be considered for prophylactic subcutaneous heparin: 5000 units being given before operation and 5000 units
at 12-hourly intervals thereafter, until the patient is fully ambulant. Unfortunately, this treatment will increase the risk of postoperative haemorrhage.

Local anaesthesia

With modern anaesthesia and the general availability of trained anaesthetists, the demand for local anaesthesia has declined; however, there are still circumstances and operative procedures where the latter will be indicated. Local anaesthesia plays an important part in minor outpatient and day case surgery, and also in more major operations where specialist anaesthetic services are not available. Some of these procedures are used primarily to produce vasoconstriction, rather than analgesia, and are then frequently combined with general anaesthesia.

Patients who have a dread of general anaesthesia should, when practicable, be offered local anaesthetic blocks as an alternative; but if there is a patient who is temperamentally unsuited for any type of surgery under local anaesthesia, this should be recognized. Knowledge of regional blocks is necessary when permanent nerve destruction for the relief of intractable pain is required, this being accomplished either by injecting alcohol or phenol, or by using percutaneous thermocoagulation techniques.

Pharmacology of local anaesthetic agents

Local effects

Clinically useful local anesthetic agents are either aminoesters or aminoamides which, when applied in sufficient concentration to the site of action, prevent the conduction of electrical impulses by the membranes of nerve and muscle. Local anaesthetics may provide analgesia by topical application to mucosal surface or by being injected around peripheral nerve endings or in the vicinity of nerve trunks. The ideal agent should have adequate potency, short latency, good penetration and diffusion, low toxicity and controllable duration of action with complete reversibility; it should also be water-soluble and stable in solution to permit heat sterilization; in addition, it should be non-irritant, non-antigenic and not interfere with wound healing. Excellent local anaesthetics are available, but the search for the ideal drug is still continuing.

In a myelinated nerve, the site of action of these drugs is at the nodes of Ranvier where the myelin sheath is thin or absent; two or three adjacent nodes must be exposed to the agent (6-10 mm of nerve) as the electrical impulse is capable of jumping one or two nodes. The conduction of nerve impulses is interrupted because the generation of an action potential has been prevented by the obstruction of the inward flow of sodium ions through the nerve membrane. A certain minimum concentration of local anaesthetic agent is necessary in order to block impulse conduction in a nerve fibre within a reasonable time, and the greater the diameter of the fibre, the greater is the concentration required. The duration of action depends on the firmness of the bond between the analgesic agent and the nerve membrane, and on the rate of drug removal, which occurs as a result of dilution by tissue fluid, diffusion away from the nerve, bloodstream uptake and metabolic inactivation.
Local anaesthetic drugs in solution exist in two forms, namely the uncharged base and
the charged cation, with the degree of ionization depending on the pH of the solution and the
buffering effect of the tissue fluid at the injection site. It is only the lipid-soluble free base
that can penetrate the fat barrier of the nerve sheath. The greater the acidity of the tissue
fluid, the higher is the ratio of ionized particles to the free base. When a local anaesthetic
solution is injected into inflamed tissues, which have poor buffering properties and a low pH
(pus has an acid pH), it tends to be ineffective.

All local anaesthetics, with the exception of lignocaine and cocaine, cause peripheral
vasodilatation by their direct action on arterioles; in contrast, lignocaine has little effect,
whereas cocaine causes vasoconstriction.

General systemic effects

In addition to local effects, local anaesthetic agents have important actions on other
systems in the body. These actions become manifest when toxic plasma levels are reached as
a result of too rapid absorption for destruction and excretion to maintain a safe equilibrium.
Systemic effects are sometimes used therapeutically, for example the effect of intravenous
lignocaine can be used to control ventricular extrasystoles, but they are usually regarded as
side- or toxic effects depending on the severity of the patient's response which is determined
both by the nature of the drug and the plasma concentration. Factors controlling plasma
concentration are those of the dose and concentration of the drug used, the vascularity of the
injection site (or mucosal surface) and the rate of the drug's removal from the plasma. The
most important toxic manifestations are in the central nervous and cardiovascular systems.

Central nervous system

Local anaesthetic agents pass readily from the bloodstream to the brain but, at
recommended clinical doses, serum levels remain well below toxic concentrations unless
inadvertent intravascular injection occurs. Toxic plasma levels initially depress cortical
inhibitory pathways, thus allowing unopposed excitatory activity which cause restlessness,
visual and auditory disturbances (tinnitus), garrulousness, slurred speech, shivering and
muscular tremors leading to convulsions; a state of generalized central nervous system
depression will follow. However, in the case of some drugs, such as lignocaine, the excitatory
phase may not be manifest, with toxicity becoming apparent initially as depression.

Cardiovascular system

The cardiovascular effects of local anaesthetics can occur either indirectly, by the
inhibition of autonomic pathways - for example during epidural anaesthesia - or directly by
depression of the myocardium and its conducting system, and relaxation of the vascular
smooth muscle. These direct actions result in the slowing of the heart, a reduction in cardiac
output and a fall in blood pressure.
Respiratory system

Central stimulation causes an increase in the rate and depth of respiration but, as the medulla becomes depressed, breathing in turn becomes rapid and shallow. Respiration ceases during convulsions and, unless resuscitative measures are taken, severe hypoxia will result.

Prevention and treatment of toxic effects

In both head and neck surgery, the toxic effects of local anaesthetics are unlikely to be a consequence of absolute overdosage, as large amounts are seldom injected. High plasma concentrations can result from direct intravascular injection (particularly intra-arterial), or from excessive amounts being placed on mucous membranes which will facilitate quick absorption. Toxic features resulting from intravascular injection occur rapidly, usually within 10-20 seconds. Clinical are caused mainly by central nervous system reactions to the drug. Cardiovascular changes are usually the result of hypoxia caused by respiratory depression. If signs of toxicity occur, the infiltration of local anaesthetic solution must cease, 100% oxygen should be administered by way of a face mask, and a good venous line secured. Frequently, such measures will be sufficient, but if muscle twitching continues, 5-10 mg diazepam should be given intravenously. An anaesthetist must be summoned so that the patient can be paralysed with a muscle relaxant, the trachea intubated and respiration controlled should convulsions occur. Cardiovascular depression, as indicated by hypotension and poor peripheral circulation, is treated by correction of hypoxia, moderate head-down tilt and infusion of crystalloid fluids together with inotropic agents, if necessary.

Hypersensitivity reactions to local anaesthetic agents

Hypersensitivity reactions are rare, and toxic reactions to small doses are usually the result of intravascular injection. True hypersensitivity reactions result in cardiovascular collapse, bronchospasm and cutaneous oedema. The treatment consists of rapid intravenous infusion of crystalloid solutions together with bronchodilators, such as aminophylline or adrenaline, and artificial ventilation with 100% oxygen. Again, hypoxia is the greatest danger and primary treatment must be directed towards achieving adequate oxygenation.

Local anaesthetic agents

The flexibility of local anaesthetic techniques is in large part due to the wide variety of agents now available, and it is possible to select a drug that will fulfil most of the requirements of any particular type of block.

Properties of special concern are those of effectiveness, speed and duration of action, spreading power, toxicity and surface activity. The surgeon should limit his repertoire of local anaesthetic drugs in order to familiarize himself thoroughly with the use of each one of them. Therefore, in the following sections on specific anaesthetic drugs, only the few most commonly used agents will be discussed.
Cocaine

Cocaine is an ester of benzoic acid that is hydrolysed by plasma cholinesterase. It is heat labile and is broken down by autoclaving. Cocaine is employed solely for topical application because it is too toxic for parenteral use. It is an excellent surface analgesic with a marked vasoconstrictor effect which is due to its ability to block the re-uptake of catecholamines released by adrenergic nerve endings; catecholamines thus accumulate at the active receptor sites. The effect of any additional sympathetic stimulation or catecholamines (either exogenous or endogenous) is potentiated and excessive sympathetic activity is responsible for many of the signs of cocaine toxicity.

Cocaine stimulates the central nervous system from above downwards causing euphoria and a reduction in the sense of fatigue; because this is an addictive effect, the drug is in the controlled category. Medullary stimulation causes an increase in both blood pressure and respiratory rate which can lead to depression, with coma or convulsions and respiratory failure. Cocaine sensitizes the myocardium to adrenaline, and toxic plasma levels can cause ventricular fibrillation. The addition of adrenaline to cocaine is both unnecessary and dangerous as the risk of cardiac arrhythmias is thereby increased.

Cocaine is absorbed from mucosal surfaces, and stronger solutions may be absorbed more slowly than weaker solutions because of more intense vasoconstriction. Nevertheless, the weaker solutions may well be safer. Cocaine is used as a 4-20% or as a 20% paste, the maximum dose being about 3 mg/kg with an absolute maximum of 200 mg in the fit adult. The duration of its action is about 60 minutes. On account of cocaine's toxicity, its use is best confined to the nose, where only small quantities are required. For the purpose of inducing anaesthesia of other mucosal surfaces, such as the larynx and trachea, lignocaine is much safer than cocaine.

Lignocaine hydrochloride

Lignocaine (Xylocaine, lidocaine USP) is an aminoacyl amide, a derivative of acetanilide, which is heat stable and can be autoclaved. It is metabolized in the liver, the metabolites being excreted in the urine. Lignocaine is a very effective local anaesthetic with a rapid onset of action and good diffusing properties. It does not cause vasoconstriction but, unlike most local anaesthetics, neither does it cause much dilatation. On injection, it has a duration of action of about 1 hour which can be prolonged to 2-3 hours by the addition of vasoconstrictor agents. When injected intravenously, lignocaine has antiarrhythmic properties on the heart. The absorption of large doses of lignocaine may produce drowsiness and amnesia as a result of depression of the central nervous system.

Lignocaine is absorbed effectively from mucous membranes and is a useful surface anaesthetic in concentrations of 2-4%. The 2% preparation is available in viscous form for oral analgesia. The more effective 4% solution can be used in hand-operated sprays or in the form of nasal packs and applicators. There is also a pressurized 10% aerosol spray which gives 10 mg lignocaine per dose. Ointments and gels in 2-5% concentrations can be used for lubricating tubes and instruments. For topical use the maximum safe dose is about 3 mg/kg with an upper limit of 200 mg in the fit adult. The onset of action is rapid but of short duration (approximately 20 minutes).
Infiltration anaesthesia can be accomplished with 0.5% lignocaine, with or without the addition of adrenaline (1:20,000), but nerve blocks require 1-1.5%. The maximum dose depends on the concentration of solution used and on whether or not adrenaline is added. The maximum dose of plain 0.5% lignocaine is 3 mg/kg (not exceeding 200 mg total) and 7 mg/kg when adrenaline is added (total maximum 500 mg). These doses should be reduced in the following cases: when stronger solutions are used; when patients are elderly or are not physically fit, and when the injection is into very vascular regions.

Prilocaine hydrochloride

Prilocaine (Citanest) is an effective local anaesthetic; it is chemically related to lignocaine, but is less toxic and longer acting. Prilocaine is as effective as lignocaine for surface analgesia and its systemic effects are similar. Dose for dose, prilocaine is less potent than lignocaine, but with large doses (over 500 mg) cyanosis may occur as a result of the formation of methaemoglobin which is harmless, unless accompanied by severe anaemia or circulatory impairment, and usually disappears within 24 hours. If necessary, the condition can be treated with intravenous methylene blue 1 mg/kg. The maximum safe dose without a vasoconstrictor is 6 mg/kg (0.5% solution), and with a vasoconstrictor it is 8 mg/kg, these doses being reduced in elderly and frail patients. Concentrations used are similar to those for lignocaine. The addition of adrenaline improves the duration and quality of the action of prilocaine but less impressively than with lignocaine. Prilocaine is the agent of choice when adrenaline is contraindicated.

Bupivacaine hydrochloride

Bupivacaine (Marcain) is a potent, effective and long-lasting local anaesthetic agent, with the addition of adrenaline marginally increasing the length of action. Bupivacaine remains active for about 4 hours (6 hours with the addition of adrenaline), but residual anaesthesia may be present for up to 36 hours. This is particularly useful in cases where postoperative analgesia is required or where the duration of surgery is uncertain. It is not an effective surface analgesic. Bupivacaine is about four times as potent as lignocaine although this is offset by its greater toxicity. It is used in concentrations of 0.25-0.5% for peripheral nerve blocks, with or without adrenaline, the maximum safe dose being 2 mg/kg. In otolaryngology, the main use for bupivacaine lies in regional nerve blocks where a prolonged action is required.

Other local anaesthetic agents

There are many other local anaesthetic agents, some of which are just as effective as those mentioned; but others, such as procaine which is less effective than lignocaine and amethocaine which is much more toxic, have been superseded by these newer agents.

Vasoconstrictors

The addition of a vasoconstrictor to a local anaesthetic solution has the effect of delaying the rate of absorption, prolonging the anaesthetic's action and reducing the maximum plasma level attained for any given dose, thereby decreasing the danger of toxic reactions.
Vasoconstrictors will reduce tissue bleeding, which is particularly important when working in very vascular areas.

**Adrenaline**

Adrenaline (epinephrine) is the vasoconstrictor most commonly used in association with local anaesthetic agents, and it is present in many commercial preparations of lignocaine, prilocaine and bupivacaine. These preparations are convenient but their concentration of adrenaline is often too high: 1 in 80,000 being common, whereas 1 in 200,000 is all that is required. In addition, these preparations tend to have a low pH (on account of the antioxidant added to prevent oxidation of the adrenaline), which results in a decrease in the free base and in a reduced penetration of nerve axons. It is often more satisfactory, therefore, to add adrenaline just before using the local anaesthetic. The dose of adrenaline by injection should not exceed 0.01 mg/kg, with a total maximum of 0.5 mg (0.5 mL of 1:1000 solution) in the fit adult. Overdosage, usually the result of intravascular injection, may cause anxiety, vertigo, pallor and palpitations. Systolic blood pressure rises while the diastolic pressure falls and cardiac output increases. However, severe tachyarrhythmias (which can terminate in ventricular fibrillation) and the consequent fall in cardiac output may lead to a fall in blood pressure and peripheral circulatory failure.

When combined with a local anaesthetic solution, or saline, infiltration of adrenaline causes ischaemia of the skin and related tissues, which both reduces bleeding and aids surgical vision, but adrenaline usually dilates blood vessels which supply muscle. Adrenaline must not be infiltrated into tissues supplied by end arteries lest arterial insufficiency and tissue necrosis result. The use of adrenaline is contraindicated in patients who are receiving drugs which sensitize the myocardium to catecholamines. These include the general anaesthetic halothane, methoxyflurane and trichloroethylene. Tricyclic antidepressants (but not monoamine oxidase inhibitors) inhibit the destruction of catecholamines and can potentiate the systemic effects of adrenaline (Boakes et al, 1973). Advanced ischaemic heart disease, marked hypertension and thyrotoxicosis also contraindicate the use of adrenaline.

**Felypressin**

Felypressin (Octapressin) is a synthetic vasoconstrictor with a low systemic toxicity which has little effect on blood pressure and does not increase myocardial irritability. The main disadvantage of felypressin is that it may take 15 minutes for the maximum vasoconstrictor effect to occur. Felypressin is a possible alternative to adrenaline when that drug is contraindicated. It is more effective than adrenaline in prolonging the action of prilocaine (Goldman and Evers, 1969), and is commercially available with 3% prilocaine in a 2 mL dental cartridge (felypressin 0.03 unit/mL).

**Phenylephrine hydrochloride**

Phenylephrine (Neosynephrine, Neophryn) is a synthetic vasoconstrictor but compared to adrenaline its action is weak; it is used in a concentration of 1:20,000. Phenylephrine does not produce tachycardia but does sensitize the myocardium to catecholamines, and probably has the same contraindications as adrenaline.
Local anaesthetic techniques

Nose and paranasal sinuses

Topical anaesthesia, using 4-10% cocaine or 4% lignocaine with adrenaline, is widely used for minor surgery on the nose, such as the removal of polyps, local electrocautery and antral puncture. The nasal cavities are first sprayed with the anaesthetic solution, the patient having been warned not to swallow any excess solution - this is particularly important when cocaine is being used, for fear of gastric absorption. After a few minutes, with good illumination and using a speculum and forceps, each side of the nose is packed with half-inch (12.7 mm) wide ribbon gauze soaked in the anaesthetic solution. The packs are removed after about 10 minutes and a wool applicator, soaked in the anaesthetic solution, is inserted at an angle of 20° to the floor of the nose until bone is felt at a depth of 6-7 cm, the end now lying adjacent to the sphenopalatine foramen. A second applicator is inserted along the anterior border of the nasal cavity until the anterior end of the cribriform plate is reached at a depth of about 5 cm. Before surgery on the nose under general anaesthesia, a similar technique is used to provide vasoconstriction, with the applicators being omitted.

For extensive surgery on the nose, a more thorough technique of inducing anaesthesia of the nasal mucosa may be required, using a modification of Moffett's technique (Curtiss, 1952). The nasal cavity is sprayed as before and the patient is then put into a supine position, with a pillow under the shoulders to extend the head until it is upside-down. The patient is told to breathe through the mouth and 2 mL of 5% cocaine solution are injected into each side of the nasal cavity, as near to the roof of the nose as possible, using a special angulated cannula. The patient remains in this position for 10 minutes and then, after pinching the nose to prevent any solution being sniffed back and swallowed, is helped to roll into a prone position, with the head being kept down throughout. The remaining fluid drains out of the anterior nares, and if any runs down the back of the patient's throat, he should be instructed to spit it out and not to swallow it. A similar technique can be used to produce vasoconstriction of the nasal mucosa for surgery under general anaesthesia. The procedure can be carried out after induction of anaesthesia, with the patient placed in the tonsillectomy position, any excess solution being removed by suction. The anaesthetic solution may extend through the sphenopalatine foramen to the maxillary nerve, and then analgesia will be sufficient for operations on the antrum; otherwise a separate maxillary nerve block is necessary for surgery in this region. If there is any possibility of the ethmoidal air cells being entered the anterior ethmoidal nerve must also be blocked. Infiltration of the columella of the nose with lignocaine through a fine needle will provide analgesia of that area not supplied by the anterior ethmoidal nerve and also help to separate mucous membrane from the septal cartilage, aiding operations on the nasal septum.

For external ethmoidectomy, a block of the anterior ethmoidal and infratrochlear nerves and infiltration along the line of incision is required; and a maxillary block is a wise precaution against the possible danger of surgery extending into its territory.

Maxillary nerve block

The maxillary division of the trigeminal nerve is blocked as it crosses the upper part of the pterygomaxillary fissure. An 8 cm needle, with a marker 5 cm from the tip, is inserted
1 cm below the inferior margin of the zygoma and overlying the anterior border of the masseter muscle, where a vertical line from the lateral orbital margin crosses a horizontal line through the middle of the upper lip. The needle is directed backwards at an angle of 30° from the horizontal and upwards and inwards, in such a direction that when viewed from the front, the shaft of the needle lies in a line which passes through the pupil. The marker indicates the maximum depth of insertion and the needle point will then lie in the pterygomaxillary fissure. The needle may be arrested at a depth of 4 cm by the upper part of the lateral pterygoid plate, but even here an injection is effective. After an aspiration test, 4 mL of local anaesthetic solution are injected and then a further 4 mL as the needle is slowly withdrawn over a distance of 1 cm.

**Anterior ethmoidal nerve block**

The anterior ethmoidal and infratrochlear nerves can be blocked together at their origin from the nasociliary nerve in the upper half of the medial wall of the orbit, 2.5 cm from the orbital margin. A 5 cm needle, with a marker 2.5 cm from the tip, is inserted 1 cm above the inner canthus and directed horizontally backwards. The needle passes between the medial rectus and the inner wall of the orbit, well away from the eyeball. At a depth of 2.5 cm, the tip lies close to the anterior ethmoidal nerve where it enters its foramen. One millilitre of local anaesthetic solution is injected and then a further 1 mL as the needle is slowly withdrawn. If bone is encountered at a depth of less than 2.5 cm, the needle should be withdrawn almost to the skin before it is redirected.

**Pharynx, larynx and trachea**

Topical anaesthesia can be used for direct laryngoscopy, and it is also of great value in the case of tracheal intubation in the awake patient with a compromised airway, in whom general anaesthesia may precipitate complete respiratory obstruction. A similar technique can be used for fibreoptic or rigid bronchoscopy, but for the latter general anaesthesia is to be preferred. Local infiltration anaesthesia may also be used for tracheostomy.

Topical anaesthesia of the oropharynx is obtained by giving the patient 5-10 mL of 2% Xylocaine Viscous, and instructing him to spread it around his mouth and retain it on the back of his tongue. After a few minutes, any remaining fluid should be spat out. With the patient sitting up, any remaining pharyngeal reflexes can be obtunded by spraying the soft palate and oropharynx with 4% lignocaine. With the tongue held forwards, a swab, soaked in 4% lignocaine and held by Krause's laryngeal forceps, is inserted in turn into each piriform fossa by sliding it over the back of the tongue and keeping close to the lateral pharyngeal wall. Once in the fossa, the swab is held there for about 1 minute, to block the internal laryngeal nerve which at this point lies just deep to the mucosa. This will produce anaesthesia in the region extending as far as, and including, the upper surface of the vocal cords. Anaesthesia of the trachea can be obtained by spraying lignocaine through the cords under direct vision, or by injecting 1-2 mL of 4% lignocaine through the cricothyroid membrane in the midline. With the latter technique, aspiration of air confirms the correct position of the needle, which must be withdrawn rapidly after injection before the patient coughs. The total dose of lignocaine should not exceed 3 mg/kg. Rigid bronchoscopy under topical anaesthesia is an unpleasant experience for the patient, and a fairly heavy premedication should be used which is consistent with the patient's ability to cooperate during the examination and to cough.
on command at the end. The combination of a narcotic and a sedative such as diazepam or midazolam is suitable.

In the case of fiberoptic bronchoscopy, sedative premedication will probably not be required (Pearce, 1980). Here, the instrument is passed through the nose, thereby necessitating surface anaesthesia of the nose, pharynx, larynx and trachea. A 4% lignocaine aerosol from a hand nebulizer is used to anaesthetize the nose, and this usually penetrates to the pharynx and sometimes to the larynx. When the nose has become numb, the bronchoscope is introduced and advanced far enough into the pharynx to obtain a view of the vocal cords which are then sprayed through the bronchoscope. After 1 or 2 minutes, the instrument is passed through the cords and into the trachea. If there is any coughing, small doses of 2% lignocaine should be injected through the instrument as it is being advanced (Clarke and Knight, 1977).

Alternatively, excellent topical anaesthesia of the tracheobronchial tree can be obtained by using an aerosol in association with a ventilator. Most patients will accept positive pressure ventilation from a face mask for the few minutes that are required to nebulize 5 mL of 4% lignocaine, using a Bord Mk7 ventilator (Newton and Edwards, 1979).

**Local anaesthesia for tracheostomy**

The simple infiltration of the skin and subcutaneous tissues in the region of the intended incision is usually quite adequate. Alternatively, complete anaesthesia of the lower half of the neck can be obtained by blocking the clavicular branches of the superior cervical plexus. This is done on each side by injecting about 5 mL of local anaesthetic solution just beneath the midpoint of the posterior border of the sternomastoid muscle. Deeper layers of the wound are managed by simple infiltration. The injection of 2-3 mL of 4% lignocaine transtracheally into the lumen will reduce coughing and straining when the trachea is opened.

**Ear**

Local anaesthesia has been used for almost all types of ear surgery and it is effective for simple brief procedures such as removal of lesions of the pinna. For long operations, local anaesthesia can be trying for both patient and surgeon; the delicate nature of the operation and the use of the operating microscope mean that the patient must lie absolutely still, the slightest movement being greatly magnified. The advantages claimed are that an awake patient will be in a position to evaluate a hearing change and that a dry operative field can be maintained, but it has never been a popular technique in Britain for major ear surgery. If the operation involves working anywhere near the labyrinth, then dizziness, nausea and even vomiting can make the procedure impossible.

Local anaesthetic solutions will not normally penetrate the drum but can be forced through it by means of an electric current, iontophoresis. This is a method whereby a direct current is passed through an anaesthetic solution which has been instilled into the external auditory meatus. The positive electrode, a stainless steel needle, is put into the meatus and the negative terminal is placed over the mastoid, forcing the positively charged ions of local anaesthetic into the tympanic membrane, thereby inducing anaesthesia of the drum.
For permeatal operations, analgesia can be obtained by injecting 0.5 mL of 1% lignocaine with adrenaline posteriorly, just lateral to the junction of the bony and cartilaginous parts of the meatus, while 0.25 mL of the anaesthetic are injected superiorly, inferiorly and anteriorly at the same depth. Analgesia is enhanced by two further injections, of 0.2 mL each, being given superiorly and inferiorly at points 5 mm lateral to the margin of the tympanic membrane. This technique provides analgesia to the meatus adjacent to the drum, but more extensive blocks are required if other parts of the meatus, the auricle or skin over the mastoid are involved in the incision.

The auriculotemporal nerve can be blocked by injecting 1.5-2.0 mL lignocaine just anterior to the meatus. The operator's index finger is placed in the auditory canal and advanced until arrested by its bony part; the needle point is then advanced until it is adjacent to the tip of the finger. The great auricular and auricular nerves can be anaesthetized behind the pinna, using a common skin puncture. A weal is raised in front of the lower anterior border of the mastoid process, a 7 cm needle is inserted and directed upwards, and 2-3 mL of anaesthetic are placed between the mastoid process and the meatus. The needle is then withdrawn and redirected upwards and backwards to pass posteriorly to the ear canal until its tip is cranial to that structure; 2-4 mL of solution are injected as the needle is being withdrawn.

If anaesthesia is required within the tympanic cavity, 4-6 drops of sterile 4% lignocaine may be instilled; but it may be necessary to repeat this process because the irregular shape of the tympanic cavity can result in the uneven distribution of the anaesthetic. Local anaesthetic solutions injected in this way can cause dizziness and nausea lasting up to 8 hours; this is thought to result from diffusion of the anaesthetic through the round window membrane (Simmons, Glattke and Downie, 1973).

**General anaesthesia**

**Drugs used in general anaesthesia**

It is not possible to describe the pharmacology of all the drugs used in general anaesthesia, but some of the principal agents will be mentioned. General anaesthetic agents can be divided into inhalational and intravenous categories, although some of the latter can be given by other routes. Inhalational agents can be subdivided into gases and volatile liquids.

**Nitrous oxide**

At the present time, nitrous oxide is the only widely used anaesthetic gas. It is non-flammable, colourless, slightly heavier than air and has a sweetish odour. It is a weak anaesthetic and, if hypoxia is to be avoided, it is very difficult to produce general anaesthesia by using this agent alone. Subanaesthetic doses (50%) are used for analgesia, but for general anaesthesia, 60-70% nitrous oxide in oxygen is supplemented with other agents. Nitrous oxide depresses the central nervous system, causes a slight depression of myocardial contractility together with some alpha- and beta-adrenergic stimulation and, after prolonged use, causes depression of the bone marrow function.
Nitrous oxide is 34 times more soluble than nitrogen in the blood (Thomsen, Terkildsen and Arnfred, 1965) and because of this disparity, it will diffuse into any air-containing cavity more rapidly than nitrogen will exit. In the normal middle ear, an increase in pressure (limited by the passive opening of the eustachian tube) and a slight increase in volume, resulting from the outward bulging of the tympanic membrane, occur during inhalation of the gas. The rate of rise of pressure with the inhalation of 70% nitrous oxide is about 0.1-0.2 kPa/min (10-20 mmH2O/min), reaching a maximum of 3.9 kPa (400 mmH2O) in 30 minutes (Davis, Moore and Lahiri, 1979). The rate of rise of pressure will be increased if respiration is assisted, using a bag and mask, because gas can be forced into the ear cavity from the nasopharynx (Casey and Drake-Lee, 1982). The rate of diffusion is even more rapid into ear clefts with a markedly subatmospheric pressure (Drake-Lee, Casey and Ogg, 1983). When nitrous oxide is discontinued, a negative middle ear pressure may develop on account of the gas diffusing out more rapidly than nitrogen can enter to replace it (O'Neill, 1980). Nitrous oxide can have no effect on a middle ear cavity which is completely filled with fluid, as there will be no air-containing space with which the gas can equilibrate (Gates and Cooper, 1980). This situation is probably unusual because some air remains, at least in the mastoid air cells, in most fluid-filled ear clefts.

These pressure changes can be either beneficial or harmful. An atelectatic tympanic membrane may be forced into a normal position and contour (Graham and Knight, 1981) and serous fluid may be displaced (Marshall and Cable, 1982). Tympanic membrane rupture has been reported after nitrous oxide inhalation (Man, Segal and Ezra, 1980), particularly in patients with pre-existing ear pathology. A conductive hearing loss, lasting 1 or 2 days (Waun, Swietzer and Hamilton, 1967), and the displacement of ossicular reconstruction and tympanic membrane grafts have also been reported (Patterson and Bartlett, 1976). During middle ear surgery, it may be advisable to discontinue nitrous oxide for a few minutes before the ear is closed with a tympanic graft, in order to avoid both the ballooning of the graft while it is being fixed in position and the retraction of the graft after the operation.

Halothane, enflurane and isoflurane

Halothane, enflurane and isoflurane are the three volatile anaesthetic agents most widely used today. They are all non-flammable. Halothane is twice as potent as enflurane, with isoflurane occupying an intermediate position. These agents are administered from calibrated, temperature compensating vaporizers, each liquid having its own specific vaporizer. Halothane is used in a concentration of 0.5-2% for maintenance of anaesthesia, and higher concentrations can be used for induction. All three have a depressant action on respiratory and cardiovascular systems which is proportional to the depth of anaesthesia. The hypotension that results from halothane is due to myocardial depression and a reduction in cardiac output, while with isoflurane, the fall in blood pressure can be attributed to reduction in peripheral vascular resistance. Halothane sensitizes the myocardium to catecholamines, and arrhythmias are not infrequent, especially if marked hypercarbia is allowed. For this reason, the use of halothane is contraindicated if it is planned to use adrenaline infiltration during surgery. Enflurane or isoflurane are compatible with adrenaline, with isoflurane being noted particularly for the maintenance of a stable cardiac rhythm during anaesthesia.

There is evidence that halothane may cause gross disturbance of liver functions, with severe and often fatal jaundice; this is more likely to occur if repeated administrations are
given within 6 months. The complication is fortunately very rare, but, when repeated anaesthesia is necessary within this period, consideration should be given to the use of an alternative agent.

**Intravenous anaesthetic agents**

The induction of anaesthesia in the adult today is almost always by the intravenous route, one notable exception being in the patient with a compromised airway. Sodium thiopentone is still the most commonly used drug and it produces a smooth quiet induction in one arm-brain circulation time. Much emphasis has been placed on the respiratory and cardiovascular depressant effects of thiopentone, but in the healthy patient these are, in practice, minimal. If it is used with care, and given slowly and in reduced dosage to the ill patient, thiopentone is a very satisfactory agent in skilled hands. Like all intravenous anaesthetic agents, it should never be given by those unskilled in airway maintenance, tracheal intubation and artificial respiration. Recovery from thiopentone results from the redistribution of the drug in the body and not from rapid detoxication, as a large amounts of unmetabolized drug remains for at least 12 hours after its administration. Thiopentone is not ideal for day case anaesthesia. Methohexitone, while not giving as smooth an induction as thiopentone, is more rapidly metabolized and will produce less of a hangover effect; thus it is a more suitable intravenous barbiturate for outpatient anaesthesia.

Many other drugs have been developed for intravenous anaesthesia but most have been discarded, either because they had no obvious advantages over thiopentone, or produced hypersensitivity reactions in too high a proportion of patients, or had other toxic effects. Of these drugs, two remain at present (1985): etomidate, which is rapidly metabolized but produces a high incidence of involuntary movement during induction, and propofol (diisopropyl phenol), also rapidly metabolized, which is still undergoing clinical trials and shows considerable promise.

**Muscle relaxant drugs**

Since the introduction of tubocurarine into anaesthetic practice, a large number of myoneural blocking agents have been developed over the last four decades, all capable of producing profound relaxation of striated muscle during light general anaesthesia. Tubocurarine is still in use and is popular in the technique of controlled hypotensive anaesthesia as it produces a fall in blood pressure by histamine release and ganglionic blockade, as well as a non-depolarizing myoneural blockade. Suxamethonium, the only depolarizing relaxant in common use, is valuable when a short period of relaxation is required, for example, to facilitate laryngoscopy and tracheal intubation. Pancuronium is widely used in general anaesthetic practice, its main disadvantage being the propensity to cause tachycardia. The newer relaxants, atracurium and vecuronium, which do not have any significant effect on the cardiovascular system and are easy to reverse, even after prolonged administration, are gaining wide acceptance.

**Narcotic drugs in general anaesthesia**

Opiates are frequently given either intramuscularly before operation or, more logically, intravenously during surgery, to augment the effects of general anaesthetic agents and to
provide a carry-over effect of analgesia into the postoperative period. Morphine (0.15-0.2 mg/kg) or papaveratum (0.2-0.25 mg/kg), with a duration of action of about 4 hours, is often used. The longer-acting levorphanol (25 μg/kg), with an action lasting 6-8 hours, may be more appropriate if the main aim is that of postoperative pain relief. The short-acting narcotic drugs, such as fentanyl (with a duration of action of about 20 minutes with a single 1-2 μg/kg dose) or ultrashort-acting alfentanil (with action lasting about 4-5 minutes after 5-7 μg/kg) may also be used intraoperatively to prevent reaction to painful stimuli. Narcotic drugs cause dose-related respiratory depression, most marked after intravenous administration, which can be reversed by intravenous naloxone (0.4 mg). Repeated or very large doses of fentanyl can, after excretion in the bile, be reabsorbed from the gut and cause unexpected delayed respiratory depression about 6-8 hours after administration. Nausea and vomiting frequently ensue from the use of narcotics, and will require treatment with antiemetics.

Antiemetic drugs

If antiemetic drugs are not used, about one-third of all patients undergoing anaesthesia and surgery will suffer nausea and vomiting afterwards, an in certain types of surgery (for example operations on the middle ear), the proportion is very much higher. The cause may be peripheral or central in origin, the latter arising either from the stimulation of the chemoreceptor trigger zone or by the activation of labyrinthine reflexes. Antiemetic drugs act either in the periphery or on the vomiting centre or both. The most useful drugs belong to the antihistamine or phenothiazine groups. Perphenazine is one of the most effective of these drugs, particularly in doses of about 5 mg, but the drug is liable to produce distressing extrapyramidal side-effects in a proportion of patients if it is repeated too frequently. It is much less likely to produce side-effects if the dosage is halved, but it thereby becomes less effective than cyclizine. Cyclizine, an effective antiemetic with few side-effects, can be given parenterally as the lactate (50 mg every 6-8 hours) or by mouth as 50 mg hydrochloride tablets. In severe cases of vomiting, such as that associated with labyrinthine disturbances, there is often an associated agitation, so the addition of a benzodiazepine (eg, diazepam) can be helpful in settling the patient.

When opiates are used in premedication, the addition of an antiemetic will decrease the probability of nausea. This is particularly useful if atropine, which has an antiemetic action, has been omitted.

Techniques of general anaesthesia in otolaryngology

The majority of adult patients will have induction of anaesthesia with an intravenous barbiturate (for example thiopentone 4-6 mg/kg) followed by a muscle relaxant and, when the muscles of the jaw and larynx are relaxed, a direct laryngoscopy can be performed followed by tracheal intubation, with the tube being passed through either the mouth or the nose. Anaesthesia is maintained either by artificially ventilating the paralysed patient through the tracheal tube with nitrous oxide, oxygen and an inhalational anaesthetic agent or, if a short-acting relaxant (suxamethonium) is used, by allowing spontaneous breathing of the anaesthetic mixture. If artificial ventilation is being used, a cuff will be necessary on the tracheal tube to provide an airtight seal. Oral tracheal intubation is preferable to the nasal route as the former avoids damage to delicate nasal mucosa and allows the passage of a larger tube with
less resistance to respiration. However, intubation does not guarantee a free airway, for tubes can become kinked (less likely with modern kink-resistant tubes), obstructed with secretions or the tip can be occluded by resting on the carina or against the wall of the trachea. If the tube is too long it will pass into a bronchus, usually the right, resulting in ventilation of one lung only with consequent cyanosis. If the tube is too short it may become dislodged, which is particularly likely if the patient's head is flexed or rotated. A tracheal tube does not ensure protection of the air passages from soiling by blood or secretions. A carefully placed throat pack and an inflated cuff on the tube, to make an airtight fit with the tracheal wall, are necessary; a cuffed tube without a pack will not always prevent blood seeping past into the trachea.

If anaesthetic hoses between the tracheal tube and the anaesthetic machine become disconnected during spontaneous respiration, the patient will become too lightly anaesthetized; but with a paralysed, ventilated patient, respiration will cease and life-threatening hypoxia result. These disconnections can occur very easily, often as the result of the patient's head being moved by the surgeon, and pass unnoticed when the head is covered with sterile towels; therefore, the surgeon should give a warning, and the anaesthetist should check the circuit, each time the position of the head is altered. The use of a disconnection alarm, fitted to the anaesthetic circuit, is a wise precaution.

The aim is to establish and maintain a perfect airway and, while minor degrees of obstruction may not be life-threatening, the resultant venous congestion will increase bleeding. Light anaesthesia which results in coughing and straining on the tracheal tube, particularly likely if the tube is moved as occurs when the position of the patient's head is altered, will have the same result.

Anaesthesia for tonsillectomy

The fact that adenoidal tissue is normally absent in the adult means that a nasotracheal tube is suitable for this type of operation, but a kink-resistant reinforced oral tube, together with a gag with a split tongue plate, can also be used. Spontaneous breathing is usually allowed and the standard tonsil position is used with the nasopharynx dependent to protect the airway from blood; therefore, a cuffed tube is not necessary. Stiff plastic nasal tubes should be softened in warm water before use to reduce trauma to the nasal mucosa; many anaesthetists still prefer the older softer red rubber tubes. There is no place nowadays to attempt a tonsillectomy under general anaesthesia using an insufflation technique without a tracheal tube.

Post-tonsillectomy bleeding

Reoperation to control secondary or reactionary bleeding after tonsillectomy presents a considerable hazard for the patient and poses formidable problems for the anaesthetist. Bleeding in the early postoperative period may be largely masked until blood loss is of significant proportions; this is because the patient, drowsy or even asleep, may swallow blood as quickly as it is lost from the circulation, and the first external sign of bleeding may be vomiting of blood. It is essential at this stage that a reliable intravenous line be established and blood sent for grouping and cross-matching, if this has not already been done. It is ill-advised to give sedative drugs in the hope that bleeding will stop, as these will tend to mask
further bleeding and also increase the risk of aspiration of blood into the respiratory tract. It is important not to anaesthetize the patient until resuscitative measures are well in hand and cross-matched blood is available, with adequate volumes of crystalloid solution being given while awaiting the blood. While it must be assumed that the patient's stomach is full of blood, most of these patients will be too distressed to tolerate the passage of a gastric tube, and the attempt may further aggravate the bleeding.

Problems during induction of anaesthesia include the probability of blood in the stomach, with the risk of vomiting or regurgitation, and the danger of aspiration. An additional hazard is the presence of varying amounts of blood clot and active bleeding in the pharynx which can obscure the larynx, or even cause respiratory obstruction. The anaesthetist must decide whether there will be any difficulty in intubating the trachea, and careful inspection of the pharynx in the awake patient is necessary. If no difficulty is anticipated, a rapid sequence intravenous induction is generally acceptable; that is preoxygenation, an intravenous induction agent and suxamethonium, cricoid pressure (to prevent passive regurgitation of stomach contents) and intubation using a cuffed oral tube, avoiding, if possible, positive pressure ventilation of the patient before intubation. In the presence of active bleeding or numerous large blood clots, it is advisable to maintain spontaneous respiration until there is absolutely no doubt that intubation can be achieved. The technique then is to induce anaesthesia with oxygen and halothane with the patient in the head-down left lateral position, with cricoid pressure being applied by an assistant from the time of loss of consciousness. When the patient is deeply anaesthetized, direct laryngoscopy and intubation, again with a cuffed oral tube, is carried out. Adequate suction must be available at all times and an adequate selection of working laryngoscopes and a variety of tracheal tubes must always be to hand. Most of the disasters from pharyngeal bleeding occur as the result of loss of control of the airway.

**Anaesthesia for the patient with an obstructed airway**

Respiratory obstruction, of varying aetiology and severity, may occur in any part of the respiratory tract; trismus may or may not be present.

In patients with severe obstruction, who are in obvious distress and using the accessory muscles of respiration, the only safe method of inducing general anaesthesia is by first establishing the airway in the conscious patient. If the obstruction is in the region of the glottis, it may be possible to perform a direct laryngoscopy under local anaesthesia and intubate the trachea, thereafter rapidly inducing intravenous general anaesthesia. However, if the obstruction is higher or trismus is present, then the only safe choice may be a tracheostomy performed under local anaesthesia. The cardinal rule is that general anaesthesia should not be induced until the airway is secured.

With lesser degrees of obstruction, induction of anaesthesia may be attempted by using an inhalational technique and maintaining spontaneous respiration until direct laryngoscopy shows that intubation is possible, but the surgeon must be available for emergency tracheostomy or cricothyrotomy, should this be necessary. Intravenous induction of anaesthesia is dangerous as respiration can cease and the anaesthetist may not be able to ventilate the patient or visualize the larynx.
In some cases, it may be feasible to use a fibreoptic bronchoscope. First, a suitably sized nasotracheal tube is threaded over the instrument and then, under local anaesthesia, the tube and endoscope are passed together through the nose, the larynx visualized, and, once the tip of the instrument is through the glottis, the tube is pushed down into the trachea. Because of swelling and anatomical distortion, the procedure can be very difficult and should be attempted only by those skilled in the use of this instrument.

Blind nasal intubation should not be attempted when the airway is compromised as, on account of the distorted anatomy, it is unlikely to succeed and it may precipitate complete obstruction. This manoeuvre is of value in the unobstructed patient in whom laryngoscopy is impossible, for example, a patient with a wired fractured mandible.

In all cases where a partially obstructed airway is present, medication with opiates is dangerous because depression of the respiratory centre may lead to a complete cessation of breathing. In lesser degrees of respiratory embarrassment, a benzodiazepine (for example diazepam) may be helpful.

**Anaesthesia for nasal surgery**

The principles of anaesthesia during surgery on the nose are those of faultless maintenance of anaesthesia and protection of the airway, with recognition of the probability of blood and debris draining into the pharynx during operation, of the possibility of systemic effects from the use of vasoconstrictor drugs, and of the likelihood of preoperative and postoperative nasal obstruction. Auffed oral tube together with a pharyngeal pack should be used to prevent soiling of the respiratory tract. Throat packs should be moistened with water, not with paraffin or other insoluble lubricants which may cause lipid pneumonia if they enter the lungs. Spontaneous ventilation is satisfactory for most nasal operations. If cocaine or catecholamines are used topically, it is safer to avoid the use of halothane; enflurane or isoflurane are suitable alternatives as they are much less likely to be associated with arrhythmias. Controlled ventilation using muscle relaxants, nitrous oxide and oxygen, with narcotic or inhalational supplementation, is particularly suitable in very prolonged procedures or where a rapid recovery of consciousness is desired. It is also a valuable technique in patients with a respiratory disease with an irritable respiratory tract, in whom deep anaesthesia would otherwise be necessary to prevent coughing or straining on the tracheal tube.

The patient should be put in a slightly head-up position to help reduce bleeding and improve surgical access. At the end of the operation, care must be taken to remove the pharyngeal pack, clear the pharynx of blood by suction, turn the patient into the semi-prone position and insert a pharyngeal airway before removal of the tracheal tube. If a nasal pack is to be left in place, the security of this must be checked to ensure that there is no likelihood of it becoming displaced and thus liable to being inhaled. A good airway, adequate anaesthesia, correct position of the patient and efficient use of topical vasoconstrictors are usually adequate to reduce bleeding to a minimum, but with the occasional difficult rhinoplasty or extensive cancer surgery, the use of controlled hypotension may be considered.
Anaesthesia for endoscopy

Laryngoscopy and microlaryngoscopy

During laryngoscopy, the surgeon requires a clear view, immobile cords and adequate space for inspection and instrumentation; while good anaesthetic practice demands overall safety, adequate respiratory exchange, protection of the lower airways and reliable and speedy recovery of reflexes at the end of the procedure. Very many different anaesthetic techniques have been described to try to meet these requirements. Light premedication, such as oral diazepam, is preferred in order to facilitate quick postoperative recovery. It is important to avoid narcotics if airway patency is suspect, and the precautions already described for induction in this situation must be taken. Most cases for laryngoscopy have a clear airway, and the main argument about anaesthetic technique is whether or not a tracheal tube should be used and, if not, how adequate ventilation should be maintained.

Using a tracheal tube

The use of a small nasal or oral cuffed tracheal tube secures the airway, provides protection from soiling and allows adequate ventilation. The 31 cm long, 5 mm tracheal tube, described by Coplans (1976), with a high volume (10 mL) low pressure cuff is suitable, so controlled ventilation using muscle relaxants is essential. The main disadvantage is that of interference with good surgical access, particularly for lesions on the posterior aspect of the vocal cords. In addition, tubes can become occluded as the result of surgical manipulation and they are a fire hazard if laser surgery is used.

Using a catheter

Instead of a 5-6 mm tracheal tube, a small catheter (14 FG or smaller) can be positioned with the tip midway between the cords and the carina. After an intravenous induction, anaesthesia is deepened using an inhalational agent and the larynx and trachea are thoroughly sprayed with 4% lignocaine before introduction of the catheter. Oxygen and anaesthetic agents are insufflated through the catheter, with the patient being allowed to breathe spontaneously (Hadaway, Page and Shortbridge, 1982). Although this method is claimed to give better surgical access, the disadvantages are the lack of protection of the lower airway, some slight movement of the cords with each respiration and the surgeon being subjected to exhaled anaesthetic gases.

Using jet ventilation

Variants of the Sanders (1967) technique of jet ventilation for bronchoscopy have been adopted for microlaryngoscopy, the great advantage being the unobstructed view of the larynx. The method uses the Venturi principle of entrainment of air by means of a tube within a tube, the larger tube being the trachea or the laryngoscope. The gas (oxygen) in the inner tube is under pressure and thus it expands at the point of exit, producing a suction effect which entrains the surrounding gas (air) in the larger tube, the result being a magnified gaseous thrust. Basic equipment consists of a bayonet-fitting nipple plugged into the high pressure oxygen outlet, connected by high pressure tubing to a manual valve, which permits control of both the frequency and length of inspiration. From the valve, plastic tubing connects the
system to a jet ventilating needle or catheter. Different points of delivery of the jet have been used and all have relative advantages and disadvantages.

If the tip of the jet is kept within the laryngoscope, then, in order to maintain effective ventilation, the tip of the laryngoscope must be kept close to the cords and the lumen of the instrument kept in line with the axis of the trachea. The cords must be fully relaxed and the lesion must not cause mechanical obstruction to air flow. Large tumours can have a ball valve effect and thus render ventilation ineffective. In these circumstances, it is best to use a tracheal tube in the first instance in order that the bulk of the tumour can be surgically reduced before jet ventilation is started. The advantage of having the jet in this position is that it avoids the need for a tube of any kind having to pass through the glottis.

The tip of the jet can protrude beyond the laryngoscope and pass a short way through the larynx, otherwise dangerously high intratracheal pressure may be generated with resultant barotrauma. The size of the ventilating needle will vary according to the weight and age of the patient. A plastic nasal catheter can be used instead of a needle, the tip being placed 3-5 cm above the carina (Tobias, Nasser and Richards, 1977).

A more recent method of jet ventilation during laryngoscopy involves the use of the modern high frequency ventilator (Eriksson and Sjöstrand, 1977; Smith, 1982). Ventilation at about 100 breaths per minute, with small tidal volumes, can provide good gas exchange with low airway pressures. The gas can be delivered by a nasal or oral catheter with the tip well below the level of the cords (Babinski, Smith and Klain, 1980). A fairly stiff catheter, 3.5-4.0 mm in diameter, is necessary for this procedure because fine flexible catheters vibrate quite violently with the rapidly alternating pressures and this can damage laryngeal or tracheal mucosa. The ventilator driving pressure can be varied, but although lower pressures decrease the vibratory cord movement, they tend at the same time to produce less effective ventilation. The advantages over conventional jet ventilation include a decreased risk of mucosal trauma and the resultant risk of surgical emphysema, and more effective ventilation in patients with respiratory disease.

Jet ventilation techniques are not without risk and fatalities have been reported (Vivori, 1980). The risk of barotrauma is increased if the jet exit is inside the trachea. Factors such as any obstruction to expiration, small mucosal tears or the use of too high gas pressures further increase the risk of surgical emphysema or pneumothorax. The risk is decreased if the jet is kept within the laryngoscope, precautions are taken to ensure that the airway is clear and ventilation is not started until the laryngoscope is in place and the relaxed unobstructed cords can be seen. Chest movements must be observed at all times and it is wise to commence with a low ventilating pressure (100-130 kPa in the adult). If a plastic catheter is used, it should be of reasonable stiffness to avoid a whiplash injury to the trachea (Ruder et al, 1981) and it is important that the tip is in the correct position at all times. If the catheter becomes displaced out of the trachea, large volumes of gas can be jetted into the gastrointestinal tract. When using a catheter with side holes, it is possible for gas to be forced down the oesophagus while the catheter tip lies below the glottis and ventilation appears to be normal (Chang et al, 1978). If the catheter tip goes beyond the carina, one-lung anaesthesia will result and dangerously high intrabronchial pressures may be generated.
When the jet needle is placed within the laryngoscope, gas can be forced down the oesophagus if the axis of the instrument is not kept in line with the trachea. In addition to the dangers of gross abdominal distension, even small amounts of gas forced into the stomach will greatly increase the risk of regurgitation of gastric contents. If it is suspected that gas has entered the stomach, it is a wise precaution to pass a gastric tube and to keep it in place until the patient is awake.

Jet ventilation systems may not be able to ensure adequate ventilation in patients with a very low lung compliance. In obese patients and those with obstructive airways disease it may be safer to use a small cuffed tracheal tube and manual ventilation.

Laser surgery of the larynx

Laser surgery poses special problems for the anaesthetist. If rubber or plastic tubes are used they must be protected from the laser beam because, if hit directly or contacted by a beam reflected off a shiny metal surface, they are liable to be ignited. Flammable anaesthetic agents must not be given and it is wise to avoid high concentrations of oxygen. Nitrous oxide, which supports combustion at high temperatures, should not be used. Similarly, lubricants should be restricted to aqueous solutions because ointments and other greases may be flammable. Combustible tracheal tubes must be protected by being wrapped in aluminium foil tape as far down as the cuff, or by being covered with muslin which has been previously soaked in saline. Cuffs can be protected by saline-soaked cotton swabs, but great care must be taken to ensure that they are all removed at the end of the procedure. If the patient has a tracheostomy with a plastic tube, the latter can be protected in a similar manner. It is difficult to maintain adequate protection of tracheal tubes at all times; gaps can appear between the layers of tape, and if the tape is broken the sharp edges can injure mucosa (Patil, Stenlurg and Zauder, 1979). Flexible all-metal tubes can be used but they are liable to be traumatic and it is not possible to provide a cuff. Tracheal tubes made of silicone elastomer, and coated with a layer of silicone containing reflective aluminium oxide, are claimed to be non-flammable. The aforementioned problems can also be avoided by using jet ventilation through a metal needle jet.

Cardiovascular risks during laryngoscopy

Manipulation in and around the larynx may cause a rise in blood pressure, tachycardia, dysrhythmias, myocardial ischaemia and even cardiac arrest. The risks are particularly great if coronary artery disease is present and in patients with uncontrolled hypotension. The risks can be minimized by preoperative treatment of hypertension, adequate anaesthesia and ventilation, and by careful monitoring. Beta-adrenergic blocking drugs before or during induction may be helpful, as may small intravenous doses of lignocaine. Tachycardia and the hypertensive response to laryngoscopy can be moderated by careful application of topical anaesthesia to mouth, pharynx and larynx after the patient is asleep. The procedure must be carried out slowly, one stage at a time, to avoid triggering the reflex; and although this may be tedious, it may be of great value to the high risk patient. Levels of anaesthesia which are too light, hypercarbia or hypoxia are all liable to increase the incidence of dysrhythmias. The most dangerous combination is tachycardia and hypertension, with the former decreasing the time available for coronary perfusion of the deeper layers of the myocardium, and the latter increasing the workload of the heart. Strong et al (1974) reported a 1.5% incidence of
myocardial infarction or myocardial ischaemia following microlaryngoscopy, but the incidence rose to 4% in those with a previous history of cardiac disease. As many of these infarctions are symptomatically silent, a routine postoperative ECG is recommended for those patients thought to be at risk.

ECG monitoring and frequent blood pressure measurements are essential during laryngoscopy. If hypertension or dysrhythmias should develop during the operation, it may be necessary to release the suspension laryngoscope for a few minutes, deepen anaesthesia and sometimes to give beta-adrenergic blocking agents.

**Bronchoscopy**

General anaesthetic techniques for bronchoscopy are as numerous and as varied as those for laryngoscopy. The procedure may be diagnostic or therapeutic using a rigid or fibreoptic bronchoscope. As in laryngoscopy, the aim is to provide a quiet patient with fully obtunded gag and cough reflexes, who will quickly recover both consciousness and protective reflexes once the examination is completed. The choice of general anaesthetic technique is dependent, to some extent, on the purpose of the examination, how long it is likely to take and the condition of the patient. Retention of spontaneous respiration is preferred in those cases where there is a foreign body, where there are copious bronchial secretions or where a broncho-pleural cyst or fistula is present. In all techniques, sufficient general anaesthesia, topical anaesthesia and intravenous narcotics should be provided to prevent the occurrence of hypertension and dysrhythmias associated with stimulation of the tracheobronchial tree.

For rigid bronchoscopy, anaesthesia can be induced in any suitable way and continued with the patient breathing a mixture of oxygen and inhalational anaesthetic agent. When the anaesthetic is deep enough, the glottis and upper trachea are sprayed with a local anaesthetic solution and a ventilation pattern bronchoscope is inserted. Spontaneous ventilation may continue with the anaesthetic mixture being delivered through the side port of the bronchoscope, and with the eyepiece-obturator in position as often as possible. It is quite possible to assist or even control ventilation in these circumstances. An alternative is to use the short-acting muscle relaxant, suxamethonium, in intermittent doses and to control ventilation throughout.

Another option is to use the jet injector technique already described for laryngoscopy. The high pressure jet of oxygen is applied at the operator's end of an open bronchoscope and room air is entrained during flow through the jet so that the lungs are inflated with a mixture of oxygen and air (Sanders, 1967; Komesaroff and McKie, 1972). The advantage of this method is that adequate ventilation can be maintained while the surgeon is able to work unimpeded by eyepieces. The disadvantages are that blood and particulate matter may be blown down the tracheobronchial tree, and anaesthesia cannot easily be maintained with inhalational agents. The use of the injector requires an anaesthetized and paralysed patient.

High frequency jet ventilation techniques have also been used employing rates of up to 300 breaths per minute (Smith, 1982; Vourc'h et al, 1983). Movements in the bronchial tree are reduced to a minimum with these techniques - a particular advantage during laser surgery - and an additional benefit is the minimal spread of blood and debris.
The use of general anaesthesia is not common for fibreoptic bronchoscopy but may be performed by passing the fibreoptic bronchoscope through a tracheal tube, with ventilation of the patient continuing using the space between the tracheal tube and the bronchoscope (employing a 'chimney' or side-arm connector for the breathing circuit). In some patients with tracheostomies it may be possible to pass the fibrescope through the nose or mouth and along the trachea behind the tracheostomy tube, thus allowing ventilation to be continued more easily than when the instrument has to be passed through the tracheal tube.

Bronchoscopy is well tolerated by patients who have no obvious loss of cardiac or pulmonary reserves. However, in the case of those patients with grossly impaired cardiac or ventilatory function, it is not a procedure to be undertaken lightly as complications can occur as a consequence of the inevitable increase in hypoxaemia. While trauma to the teeth and oropharyngeal tissue during the passage of the endoscope may result from inappropriate manipulation of the instrument, trauma to the tracheobronchial tree is more likely to be attributable to coughing or movement of the patient. Bleeding, even to a minor degree following a biopsy, may pose a threat to life by obstructing the airway; therefore, tracheobronchial suction through the bronchoscope must be continued until bleeding has stopped or become insignificant. Pneumothorax is rare, usually only occurring when excessive airway pressure has been allowed to develop as a result of a ventilatory obstruction or the use of injector ventilation with an inadequate expiratory airway. Any cardiovascular disturbances which may arise are similar to those seen during laryngoscopy and, as such, they respond to the same treatment.

Pharyngoscopy and oesophagoscopy

General anaesthesia for these procedures does not present any problems because tracheal intubation provides safety for the patient and good access for the surgeon. Good muscle relaxation is necessary for passage of the oesophagoscope and will also prevent sudden movement which could result in laceration or perforation of the oesophagus while the endoscope is in place. Cardiac dysrhythmias do sometimes occur as the instrument passes behind the pericardium, but they are rarely troublesome.

Tracheostomy

The key to a satisfactory tracheostomy is the prior securing of the airway by tracheal intubation or bronchoscopy, thus allowing the surgeon to perform an unhurried operation in an un congested field. If the patient has significant airway obstruction, the technique for induction of anaesthesia already described for the patient with an obstructed airway must be used. While anaesthesia and intubation are always desirable for the optimum operating conditions, general anaesthesia may, in a few cases, be too dangerous and a tracheostomy should be performed under local anaesthesia. The anaesthetist must never be persuaded to embark on a general anaesthetic against his better judgement, as he is the only person who can fully appreciate both the difficulties that may be encountered and the extent of his own experience and capability in overcoming them.
**Major head and neck surgery**

**Laryngectomy**

Careful preoperative preparation is necessary in laryngectomy patients as many suffer from chronic obstructive airways disease and require intensive preoperative physiotherapy. An assessment of laryngeal airway patency is essential. Most patients will have had a laryngoscopy and biopsy carried out a few days before, so an accurate assessment of the airway will already have been made by both anaesthetist and surgeon. If a different anaesthetist is to be involved with the laryngectomy, then consultation with the surgeon and anaesthetist who were present at the biopsy is advisable. Some patients may develop oedema after the trauma of biopsy, so a good airway at the time of the biopsy is not an absolute guarantee that all will be well a few days later. When there is doubt, the procedure described for induction of anaesthesia in the patient with a partially obstructed airway is followed. Occasionally, preliminary tracheostomy under local anaesthesia may be the safest course.

Care must be taken during intubation to avoid trauma to the neoplastic area, and bleeding; therefore, a smaller tube than usual may be necessary. A cuffed oral armoured tube is less likely to be occluded during surgical manipulation than an unarmoured type. Once the airway is established, there will be few problems in most cases. If spontaneous ventilation is allowed, then anaesthesia should be based on an inhalational agent. It is usually more satisfactory to employ muscle relaxants and controlled ventilation throughout what is often a long operation in a poor risk patient. Before the trachea is divided, the patient is ventilated with 100% oxygen; after the division, the tracheal tube is removed and a sterile cuffed tracheostomy tube is inserted into the tracheostome by the surgeon. The change-over should be performed as quickly and smoothly as possible. The surgeon who does not appreciate the need for speed in this manoeuvre may cause the anaesthetist considerable anxiety, as may the sudden discovery of a missing or inappropriate connection or piece of tubing. At this stage, care must be taken that blood does not enter the trachea. If spontaneous ventilation is allowed, anaesthesia must be deepened before the change-over period to prevent the patient from coughing, but as the patient is breathing spontaneously there will not be quite the same urgency. Bleeding can be quite brisk at times, particularly if a block dissection of neck glands is necessary, and controlled hypotension may have to be considered.

**Pharyngolaryngectomy**

A radical one-stage operation using a stomach transplant together with total oesophagectomy is a procedure that is used for the surgical treatment of some types of hypopharyngeal cancer. Anaemia, malnutrition and electrolyte imbalance may be present at the preoperative stage. Nutrition of those patients who have difficulty in swallowing liquids can be improved by nasogastric feeding, with parenteral nutrition rarely being required. There is a high incidence of postoperative chest infection after this particular operation, and the importance of preoperative physiotherapy and attainment of maximum respiratory function before surgery must be emphasized.

Access to the patient may be difficult during the operation because of the number of operators and their assistants who are present; therefore the anaesthetist may be more
dependent than usual on monitoring devices, and must insist on having access to one arm on an armboard.

Controlled ventilation is used throughout the operation and deliberate lowering of the arterial blood pressure may be helpful at certain stages. Graft circulation can be compromised by hypotension, so blood pressure must be restored to normal levels before the stomach is brought up into the neck. However, the use of controlled hypotension is not without risk in these debilitated patients, and many anaesthetists feel it to be both unnecessary and dangerous (Condon, 1971; Plant, 1982). Blood loss is substantial, particularly during mobilization of the thoracic oesophagus by blind digital dissection, and this may be largely concealed in the thorax. Dysrhythmias and dramatic falls in blood pressure, resulting from interference with venous return, are not infrequent at this stage.

When the trachea becomes unsupported after mobilization of the oesophagus, the fragility of the posterior wall of the trachea can cause rupture in the membranous part at the site of the tracheostomy tube cuff, thus making inflation of the lungs difficult or impossible (Bains and Spiro, 1979). Manual ventilation with 100% oxygen until the stomach is drawn up into the neck to tamponade the leak may cope with the situation (Plant, 1982), but a short cuffed trachea or endobronchial tube may be necessary and should be available. The pleura on one or both sides is frequently torn during blind dissection of the oesophagus, so the routine placement of bilateral chest drains is advisable.

Tracheal reconstruction

Operations involving the resection or repair of the trachea are a challenge to the anaesthetist. The problems of providing adequate oxygenation and facilitating carbon dioxide removal with an open trachea, combined with the need to provide good operating conditions, are formidable. Periods of hypoventilation may be unavoidable and careful monitoring is required in order to detect quickly the onset of serious hypoxaemia. Airway management usually necessitates a multi-stage approach, and each step must be fully planned before operation, with the nature, site and extent of the obstructed segment and the proposed method of surgical repair all being taken into account.

The anaesthetic technique depends on the degree of narrowing of the trachea and the level of the lesion. Controlled ventilation using an uncuffed small bore orotracheal tube inserted into the distal segment is a suitable technique, provided that the narrowing is not too severe (Heifetz, 1974; Borgan and Privitera, 1976). If the trachea is deviated, the bevel of the tube should be cut in such a way that the opening does not lie against the tracheal wall. With a very tight stenosis, it may be unwise to force through even a small diameter tube, for fear of subsequent bleeding and oedema, and in this case an ordinary tracheal tube is positioned just proximal to the stenosis.

The presence of the tracheal tube interferes with the surgical access during the anastomosis. This can be overcome by withdrawing the tracheal tube into the proximal segment and passing a catheter down the orotracheal tube into the distal segment of the trachea after its division, with ventilation being maintained by means of an injector or high frequency ventilation (Lee and English, 1974; Ellis, Hinds and Gadd, 1976; Rogers et al, 1985). If the stenosis is very low, two tubes may be passed, one down each bronchus. The
catheter(s) must be secured by a stitch to the tracheal or bronchial walls. As soon as the anastomosis is completed, the catheter(s) should be withdrawn and ventilation continued through the tracheal tube. Alternatively, a cuffed tube may be inserted into the distal cut end of the trachea or into a main bronchus (Kamvyssi-Dea et al, 1975), which is then removed before completion of the anastomosis, at which time an orotracheal tube is passed down through the cut ends. A technique using spontaneous ventilation may occasionally be employed in high tracheal stenosis, but the danger of opening the pleurae during the operation must be borne in mind.

Anaesthesia during the period of the tracheal anastomosis is usually maintained with intravenous drugs (Vyas, Lyons and Dundee, 1983), except where a tracheal tube has been passed into the distal segment when inhalational agents can be used. When spontaneous ventilation is used, the wound is flooded with oxygen by way of a tracheal tube placed in the proximal segment during the anastomosis.

**Monitoring during major head and neck surgery**

Monitoring should include continuous ECG display and blood pressure measurement, preferably using a direct method with radial artery cannulation which also allows access for blood gas analysis both during and after the operation. Central venous pressure measurement can be useful to help estimate fluid replacement; however, neck catheters are impracticable, while central venous catheters placed by way of an arm vein are reasonably satisfactory but their position must be checked by radiography.

Air embolism, although rare, is a potential complication. Air may sometimes be seen entering one of the neck veins, but the condition is best detected by using an end-tidal carbon dioxide monitor which will indicate a sudden fall in the gas tension should this mishap occur. A precordial Doppler flow transducer can also be used for the diagnosis, whereas hypotension, tachycardia and ECG changes (dysrhythmias and evidence of right heart strain) are late signs. Treatment consists of any measure which will increase venous pressure, such as continuous positive pressure ventilation and compression of jugular veins. The patient is ventilated with 100% oxygen and, as soon as is practicable, is placed in the left lateral head-down position. It may be possible to aspirate air through the central venous catheter. In extreme cases, external cardiac massage and transthoracic aspiration of air from the right ventricle with needle and syringe may be necessary. General supportive measures include the use of inotropic agents and correction of metabolic acidosis.

Long procedures can result in marked falls in body temperature and this should be monitored by means of a rectal or oesophageal temperature probe; falls in temperature are minimized by using a heating blanket. The use of a peripheral nerve stimulator will provide a useful guide to the state of neuromuscular blockade and the timing of further doses of muscle relaxants.

Patients should be transferred to an intensive care unit after most types of major head and neck surgery.
Controlled hypotension

Ever since surgery began, bleeding has been a problem both to the surgeon, when blood obscures the operative field and makes precise technique difficult, and to the anaesthetist, when the volume of blood lost is large. The difficulties for the surgeon are greater when the operation involves very small structures, often in confined cavities such as the middle ear. In these situations, even small amounts of blood make successful reconstructive surgery very difficult or sometimes impossible, and it is generally agreed that a reduction in blood pressure is useful, indeed often essential, in this type of surgery. Indications for controlled hypotension in other types of head and neck surgery are less clear-cut.

The aim of the anaesthetist is to provide conditions which give the surgeon the best operating field possible, while at the same time not endangering in any way the life or well-being of the patient. The exact relationship between the level of blood pressure and the degree of reduction of bleeding has not been firmly established, and there is no evidence that profound hypotension, with systolic pressures as low as 30 mmHg, or even less (Kerr, 1977), is any more effective than a more moderate lowering of arterial pressure (Donald, 1982).

There are other factors in addition to arterial pressure which are important in wound bleeding. Venous pressure, which plays an important part in venous oozing, can be reduced at the operation site by careful posturing of the patient, securing a free airway and by ensuring a complete absence of any coughing or straining on the tracheal tube. Local vasoconstriction can be produced by means of a vasoconstrictor. Infection will result in vasodilatation and increased bleeding.

Arterial blood pressure is directly proportional to cardiac output and peripheral resistance, and is maintained in normal circumstances by the autonomic nervous system and by hormonal control. These mechanisms tend to resist attempts to lower the arterial pressure with the result that resistance to drugs and techniques aimed at producing hypotension is frequently experienced, particularly in the young, robust patient.

The safety of any state of lowered blood pressure will be determined by the ability of the reduced perfusion pressure to maintain an adequate blood flow to vital organs, particularly to the brain and the heart. Both these organs are able to maintain their perfusion over a wide range of pressure change by local autoregulation, provided that their vasculature is normal. Serious reductions in blood flow in the healthy brain or heart are unlikely to occur provided that the arterial pressure is not reduced too rapidly (so that autoregulatory mechanisms have time to come into play) nor to too low a level (not less than 60 mmHg except in exceptional circumstances). When the patient is tilted feet downwards, to reduce venous pressure at head level, adequate allowance must be made for the difference in arterial pressure between the point of measurement and the brain, allowing a 2 mmHg fall in pressure for every 2.5 cm difference in vertical height.

A fall in arterial oxygen tension occurs when arterial blood pressure is reduced in anaesthetized patients (Wildsmith, Drummond and MacRae, 1975). This is a consequence of ventilation/perfusion inequalities in the lung caused by a reduction in cardiac output, a fall
in pulmonary pressure and a depression of the normal hypoxic vasoconstrictor mechanism which diverts blood flow from parts of the lung with a low alveolar oxygen tension.

Preoperative assessment is very important before controlled hypotension to exclude the possibility of cardiovascular, cerebrovascular and pulmonary disease. Anaemia is a contraindication. Hypertensive patients should be regarded as having a diseased cardiovascular system and many anaesthetists would therefore be reluctant to induce hypotension in such patients. Diabetic patients likewise are prone to atheroma, and those with severe long-standing disease are not suitable for hypotensive techniques. Similarly, a history of angina or coronary artery occlusion or cerebral vascular accident would be a contraindication.

If lung disease is present, pulmonary function tests may be helpful in assessing its severity, as may a blood gas analysis; however, caution is required if arterial pressure is to be lowered, and arterial carbon dioxide and oxygen tensions should be monitored during surgery.

Pregnancy or the use of contraceptive medication are also contraindications.

Age is not of itself a contraindication and older patients, if they are normotensive and fit, will tolerate a moderate reduction in pressure. Elderly patients are very sensitive to hypotensive techniques and care must be taken to avoid excessive reductions in blood pressure.

Blood pressure should not be reduced when a hypotensive technique is thought to carry a significantly increased risk, as will be indicated by the general condition of the patient, the past history or abnormal findings in ECG, chest X-ray, pulmonary function tests or routine blood analysis. If factors which increase wound bleeding are avoided, reasonably satisfactory results can often be obtained without recourse to a reduction of arterial pressure. Local injections of vasoconstrictor solutions may also help in these situations.

**Techniques of controlled hypotension**

Blood pressure is reduced by lowering peripheral vascular resistance or decreasing cardiac output, or by a combination of both. Tilting the patient head-up will hydrostatically decrease arterial pressure at the operation site, as well as decreasing cardiac output by pooling blood in the lower parts of the body, thereby reducing venous return to the heart. Inhalational anaesthetic agents decrease blood pressure either by myocardial depression, as in the case of halothane, or by peripheral vasodilatation as occurs with isoflurane. Some muscle relaxants increase cardiac output by increasing the heart rate (eg, gallamine), and so are unsuitable for hypotensive anaesthesia, while others have no effect on the heart rate or peripheral resistance (eg, vecuronium and atracurium) and are, therefore, quite suitable for this technique. Tubocurarine, with its hypotensive action, is also popular.

Drugs which are used specifically to lower blood pressure are many and varied; the four most commonly used are listed in Table 26.1. With the exception of labetalol, the drugs are administered by continuous infusion and the blood pressure reduction is controlled by the rate of infusion.
Table 26.1. Drugs commonly used to produce controlled hypotension

<table>
<thead>
<tr>
<th>Drug</th>
<th>Method and rate of administration</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trimetaphan camsylate</td>
<td>Infusion 3-4 mg/minute</td>
<td>Max 1 g</td>
</tr>
<tr>
<td>Sodium nitroprusside</td>
<td>Infusion 50-500 μg/kg per minute</td>
<td>Max 1.5 mg/kg*</td>
</tr>
<tr>
<td>Nitroglycerin</td>
<td>Infusion 150-500 μg/minute</td>
<td></td>
</tr>
<tr>
<td>Labetalol</td>
<td>Test dose followed by single bolus</td>
<td>0.1 mg/kg</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.15-0.4 mg/kg</td>
</tr>
</tbody>
</table>

* Rarely necessary to exceed 0.5 mg/kg.

**Trimetaphan camsylate**

Trimetaphan camsylate has a direct dilatory effect on vascular smooth muscle and causes histamine release, but its hypotensive effect is mainly due to ganglionic blockade (Adams and Hewitt, 1982). Blood pressure falls within 4 minutes of beginning the infusion and the full effect is produced within 10 minutes. Likewise, blood pressure usually returns to prehypotensive levels within a few minutes of stopping the infusion, but recovery may be delayed following prolonged administration or the giving of large doses (Adams, 1975). The hypotensive action is markedly potentiated by halothane and other inhalational agents. Tachycardia is often a problem, particularly in the young adult, causing a rise in cardiac output, increased wound bleeding and difficulty in lowering arterial blood pressure; it can usually be controlled by beta-adrenergic blocking drugs.

**Sodium nitroprusside**

Sodium nitroprusside acts directly on vascular smooth muscle causing relaxation in both resistance and capacitance vessels. The onset of action is very rapid and the duration of its effect transient, the rapidity of response making it necessary to control accurately the rate of administration, preferably with an infusion pump. When the infusion is stopped, arterial pressure will rise spontaneously and rapidly, provided that the blood lost has been replaced; vasopressors are not required (Cole, 1978). Tachycardia can be troublesome but it can be minimized by adequate analgesia, and by the use of inhalational agents and beta-adrenergic blockers.

Large doses of nitroprusside can lead to toxic effects resulting from the release of cyanide during metabolic breakdown (Vesey and Cole, 1975), so the total dose should not exceed 1.5 mg/kg and the rate of administration should be limited to 10 μg/kg per minute. In practice, there is rarely a need for 0.5 mg/kg to be exceeded, provided that use is made of inhalational agents to potentiate the hypotensive effects. Patients suffering from metabolic disorders may be unduly susceptible to the toxic effects of nitroprusside. The use of the drug is contraindicated in cases of Leber's optic atrophy (a rare inherited disease), and tobacco amblyopia and in the presence of neuropathies secondary to vitamin B12 deficiency, in all of which cyanide metabolism is abnormal. It is also to be avoided in patients with hepatic or renal failure.
**Nitroglycerin**

Nitroglycerin (glyceryl trinitrate) produces dilatation of the peripheral veins, with a lesser vasodilatory effect on the resistance vessels. It has a slower, less effective and less certain action than nitroprusside in producing hypotension, and the return of pressure to normal levels is also slower. This can be an advantage in that it makes abrupt swings in blood pressure easier to avoid (Fahmy, 1978); however, the difficulty in obtaining adequate hypotension in some patients is a disadvantage (Chesnut et al, 1978).

**Labetalol**

Labetalol is an amide with both alpha- and beta-adrenergic blocking properties, decreasing blood pressure by a reduction in peripheral vascular resistance and by a decrease in cardiac output (Richards, 1976). The drug shows a remarkable synergism with the volatile anaesthetic agents in producing a hypotensive effect, and arterial pressure can be quickly and easily controlled by adjustment of the inspired concentration of the volatile agent. Blood pressure can be raised after operation by withdrawing the volatile agent and, if necessary, giving atropine intravenously to increase the heart rate.

**Monitoring during hypotensive anaesthesia**

Careful monitoring of the patient is essential during controlled hypotension and, although particular attention must be paid to the level of arterial pressure, other vital observations must not be ignored.

**Blood pressure**

The choice of the method of measuring blood pressure depends on the drugs used for producing hypotension, the degree of reduction in pressure desired and the nature of the operation. If a rapidly acting and powerful agent such as nitroprusside is to be used then direct invasive monitoring with a cannule in the radial artery, providing a beat-by-beat observation of the pressure, is essential (Cole, 1978). When slower acting, less powerful agents are used to produce moderate falls in pressure, and when little blood loss is likely, a non-invasive method of pressure measurement is adequate. At low pressures, palpation of peripheral pulses is difficult, and a Doppler sensor placed over the radial or brachial artery and a reliable sphygmomanometer, or an automatic monitor working on the oscillotonometry principle (eg, Dinamap, Critikon), should be used. A simple mechanical oscillotonometer (von Recklinghausen) is also quite accurate in the presence of vasodilatation, provided that the anaesthetist is familiar with its use.

**ECG**

A continuous display of the ECG is necessary during controlled hypotension, for observing cardiac rhythm and for detecting the occurrence of myocardial ischaemia. A preoperative ECG should always be available for comparison.
**Carbon dioxide tension**

A large decrease in arterial carbon dioxide tension, resulting from overventilation during controlled breathing, may be dangerous during controlled hypotension as the resultant cerebral vasoconstriction will further decrease cerebral blood flow. Monitoring of the end-tidal carbon dioxide tension will act as an effective guide to the arterial gas tension, and it will provide a warning should excessive ventilation lead to the washing out of carbon dioxide.

**Blood loss**

Adequate replacement of blood loss is essential because the normal circulatory defence mechanisms which compensate for a reduction in blood volume will have been abolished by the hypotensive technique. Direct measurement of blood loss by weighing swabs and, whenever possible, by estimating the haemoglobin content of the fluid in the suction bottles, is necessary during those operations where significant haemorrhage occurs. Central venous pressure measurement can be helpful in monitoring fluid replacement, but central venous pressure can be decreased by some of the agents used to produce arterial hypotension.

**Temperature**

Temperature should be monitored during long procedures; a mid-oesophageal or rectal temperature probe is satisfactory.

**EEG**

Monitoring of the cerebral function would seem to be an ideal means of obtaining a warning when an excessive decrease in arterial pressure is compromising cerebral perfusion. The conventional EEG has marked limitations because of the complexity of the equipment and the difficulty in interpreting the record, but the cerebral function monitor is a portable and much simpler machine providing a continuous integrated record of overall cerebral electrical activity. Unfortunately, factors other than arterial pressure can have marked effects on the level of cerebral electrical activity, including those of depth of anaesthesia, blood carbon dioxide tensions and hypothermia. However, during stable conditions a decrease in the level of the cerebral function monitor trace could indicate a decrease in cerebral perfusion with cortical depression and act as a warning to increase arterial pressure.