Total Revision: Ear, Nose and Throat

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1 Essential Revision Notes

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1 Basic Sciences

HEAD AND NECK ANATOMY

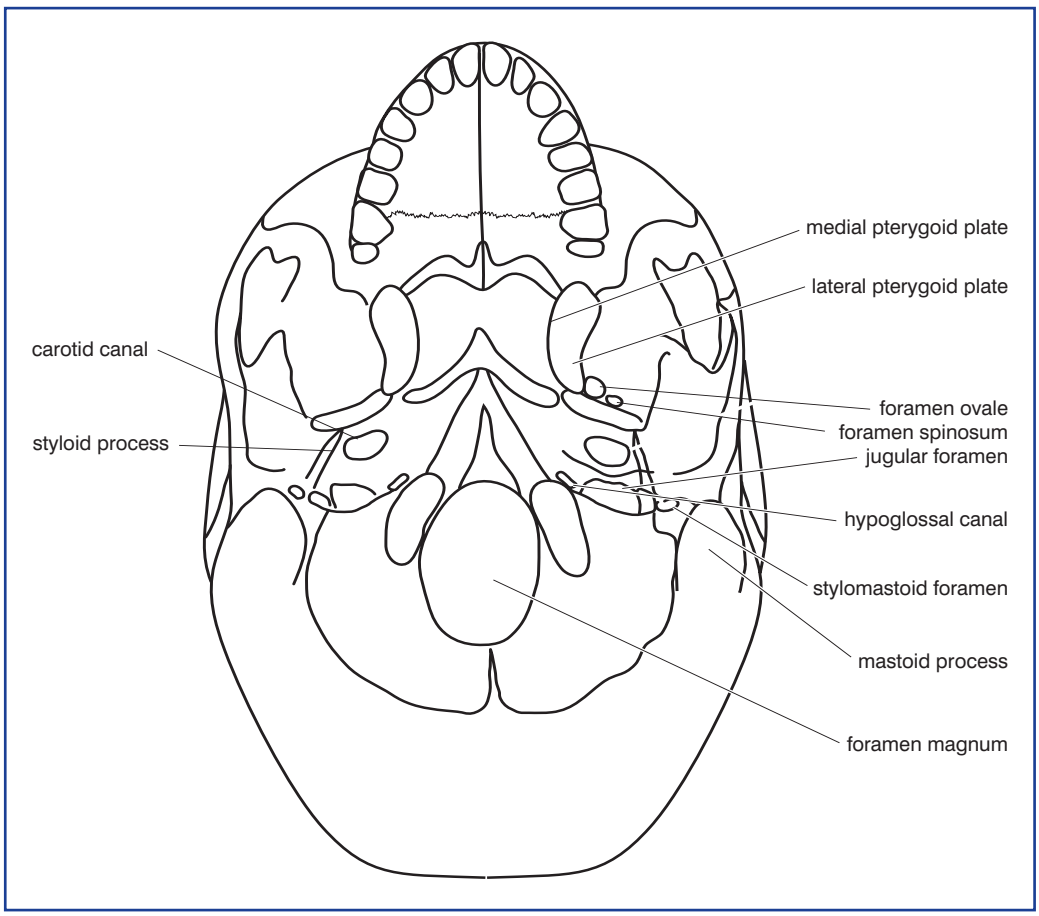

Fig. 1 The cranial fossa and nerves

Cranial nerves exit from various foramina in the skull (Table 1).

Foramen	Exiting structures
Optic canal	Optic nerve
	Ophthalmic artery
Superior orbital fissure	Ophthalmic vein
	Ophthalmic division of trigeminal nerve
	Oculomotor nerve
	Trochlear nerve
	Abducent nerve
Foramen rotundum	Maxillary division of trigeminal nerve
Foramen ovale	Mandibular division of trigeminal nerve
	Lesser petrosal branch of glossopharyngeal nerve
Foramen spinosum	Middle meningeal artery
Foramen lacerum	Internal carotid artery passes **across** foramen
	Greater petrosal branch of facial nerve
Internal auditory meatus	Vestibulocochlear nerve
	Facial nerve
Jugular foramen	Anterior compartment – glossopharyngeal nerve
	Middle compartment – vagus nerve, accessory nerve
Hypoglossal canal	Hypoglossal nerve
Foramen magnum	Spinal accessory nerve (entering)
Stylomastoid foramen	Temporal/zygomatic/buccal/mandibular/cervical/posterior auricular branches of facial nerve

Table 1 Foramina at the base of the skull and exiting structures

The external carotid artery has a number of branches, which supply various structures in the head and neck.

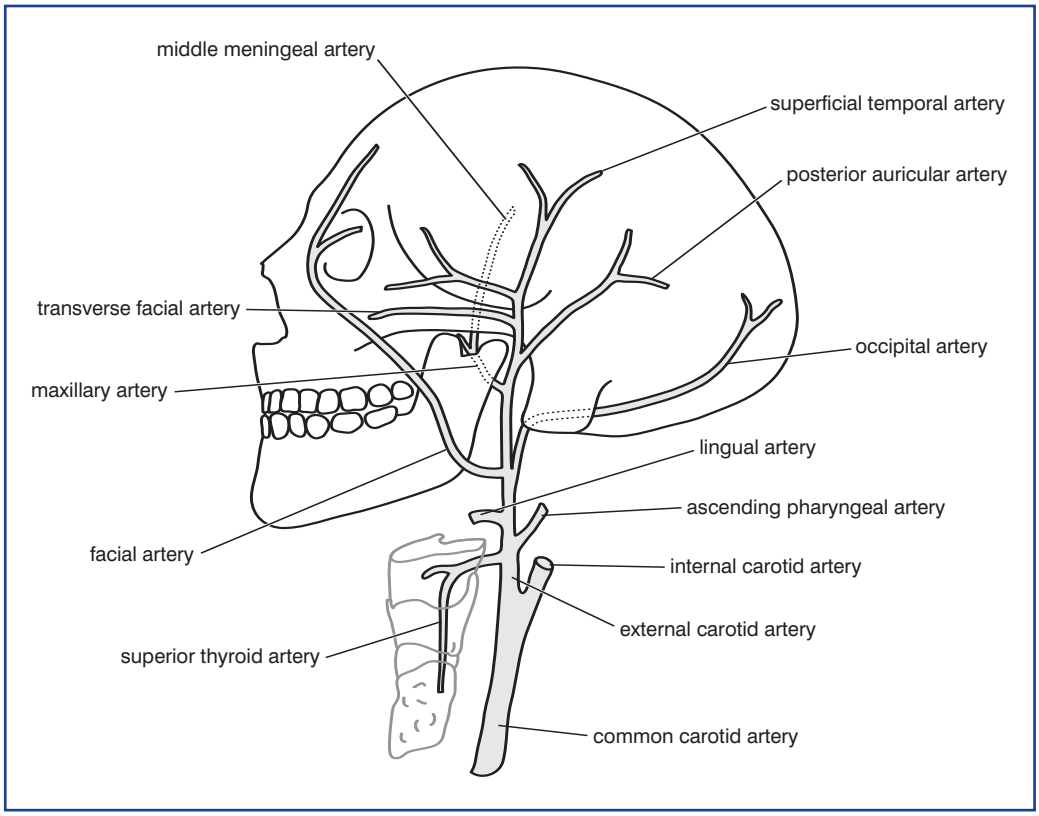

Fig. 2 The external carotid artery and its branches

INFLAMMATION

Inflammation is the response of the body to various types of injury, including:

- neoplastic
- infective
- traumatic
- autoimmune.

The resulting inflammation can be:

- acute – with a rapid onset and limited duration, or
- chronic – with a prolonged response and continuous repair taking place.

Acute inflammation

The classic components of the acute inflammatory response are:

- redness (rubor)
- swelling (tumor)
- heat (calor)
- pain (dolor)
- loss of function.

The response is caused by:

- local vasodilatation
- exudation of fluid and protein
- migration of leucocytes into the injured area.

The process is mediated by a number of chemical mediators:

- histamine
- cytokines
- nitric oxide
- protein components of plasma (complement system).

The complement system is a cascade of proteins, mutually activated in sequence, resulting in the lysing of microbes by the membrane attack complex (MAC). The most critical step is the activation of the C3 component:

- either by the **classical pathway** (triggered by an IgM or IgG antibody)
- or by the **alternative pathway** (triggered by exposure to the surface of the microbes).

Two other systems, the kinin system and the clotting system, also play a part in the acute inflammatory response.

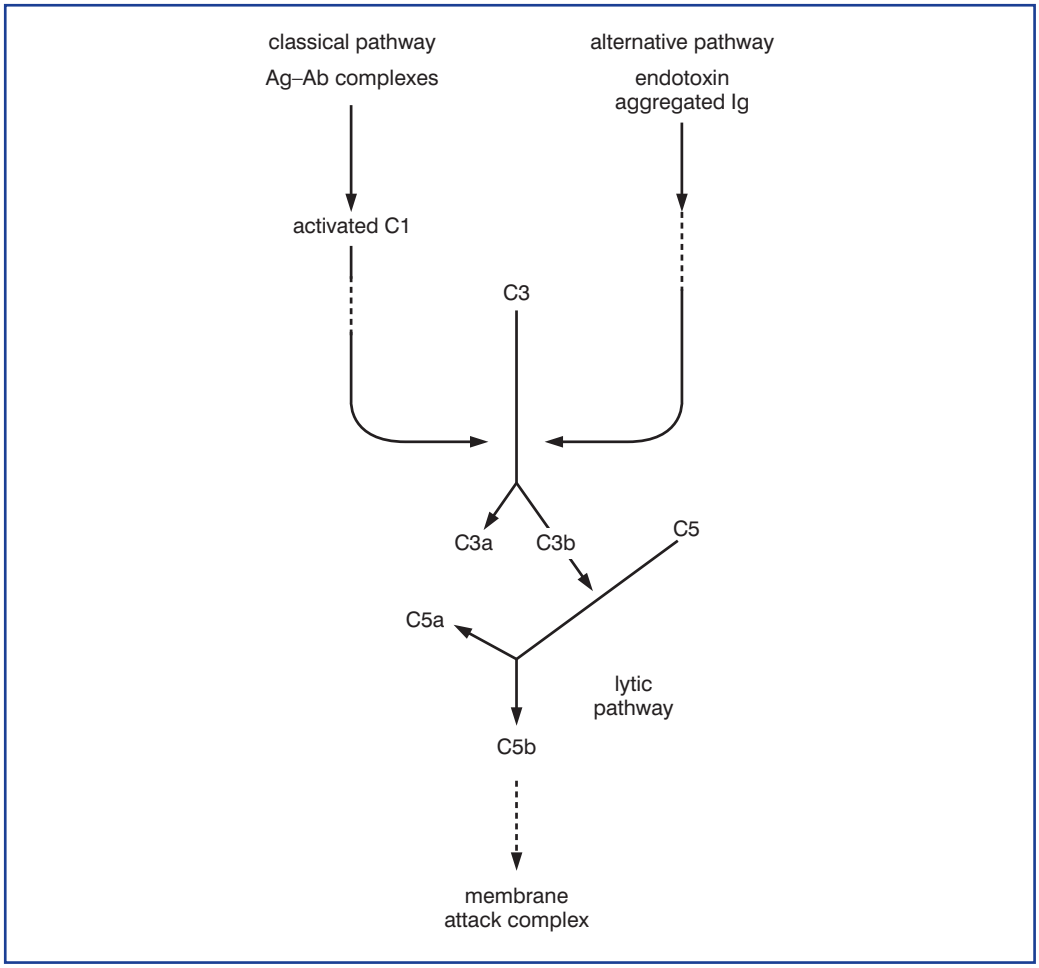

Fig. 3 The complement system

Chronic inflammation

Chronic inflammation is an ongoing but unresolved response to injury. Its characteristics are:

- proliferation of blood vessels and connective tissue
- migration of lymphocytes and macrophages
- fibrosis
- ongoing repair
- formation of granulomas.

Granulomas are focal areas of inflammation with a group of macrophages at the centre that transform into epithelial-like cells and are surrounded by a collar of lymphocytes and

plasma cells. The epithelioid cells may fuse to form 'giant cells'. The inflammation may produce central caseating necrosis (classically in the case of tuberculosis (TB)) or may be non-caseating (most other chronic inflammations).

Response to inflammation

This depends on:

- the body's immune status
- the nature of the injuring agent
- the site of the injury.

After commencement of treatment, the response will also depend on choice of treatment and effective removal of the initial stimulus.

Infections may be classified as:

- conventional (infection in previously well individuals)
- conditional (infection in the presence of a predisposing factor)
- opportunistic (infection in an immunocompromised individual).

Most infections encountered in the ENT situation are conventional.

MICRO-ORGANISMS IN ENT INFECTION

Bacteria in ENT infection

The list below gives the classification of common bacteria encountered on a surgical ward, along with the situations in which some of them might be encountered in ENT.

Gram-positive

- Gram-positive cocci
  - Aerobic
    - staphylococci (clusters)
    - *Staphylococcus aureus* – **wound infections, chronic suppurative otitis media (CSOM), acute sinusitis, furunculosis**
    - *S. epidermidis* – normal skin commensal
    - streptococci (chains/pairs) – **otitis externa**
    - α-haemolytic streptococcus
    - *Streptococcus pneumoniae* – **acute sinusitis, tonsillitis, acute suppurative otitis media (ASOM)**
    - *S. viridans*
    - β-haemolytic streptococcus – **tonsillitis**
    - Lancefield group A – *S. pyogenes* – **acute sinusitis**

    - Lancefield group B – *S. faecalis* – **dental infections**
  - Anaerobic
    - gut flora, *Enterococcus faecalis* – **tonsillitis, quinsy**
- Gram-positive bacilli
  - Aerobic
    - *Corynebacterium diphtheriae*
  - Anaerobic
    - *C. tetani*
    - *C. difficile*
    - *Actinomycetes israelii*

Gram-negative

- Gram-negative cocci
  - Aerobic
    - *Neisseria meningitidis*
    - *Moraxella catarrhalis* – **acute sinusitis, ASOM**
- Gram-negative bacilli
  - Aerobic
    - *Pseudomonas aeruginosa* – **malignant otitis externa, tracheostomy infection**
    - *Campylobacter*
    - *Haemophilus influenzae* – **tonsillitis, epiglottitis, ASOM**
    - *Legionella*
    - *Escherichia coli* – **dental infections, tonsillitis, quinsy**
    - *Proteus*
    - *Mycobacterium tuberculosis* – **chronic ENT infections**
  - Anaerobic
    - *Bacteroides fragilis*

Along with bacteria, fungi and viruses may also play a part in ENT disease.

Fungi in ENT infection

- *Aspergillus fumigatus* – this may cause **fungal sinusitis**, which is usually chronic but may be acute in immunocompromised individuals.
- *A. niger* – along with the other *Aspergillus* organisms, this may cause **fungal ear infections**.
- *Candida albicans* – this may cause **oral thrush**.

Viruses in ENT infection

Viral infection may be the precipitant cause for a bacterial tonsillitis. The following specific viruses are also encountered in ENT practice:

- Respiratory syncytial virus (RSV) – leads to chest infections, mainly in children in winter-time.

- Human immunodeficiency virus (HIV) – infects cells carrying the CD4 antigen, such as monocytes, macrophages and T-helper cells. More than half of patients may have head and neck manifestations such as otitis media, chronic rhinosinusitis, oropharyngeal Kaposi's sarcoma, lymphoma, herpes, or neck infections.
- Herpes – herpes zoster infection may lead to facial pain. In particular, if it affects the facial nerve, it may cause facial palsy. This is the Ramsay Hunt syndrome.

Antibiotics for ENT infections

The principles of antibiotic use in ENT are the same as in any branch of medicine. Effort should be made where possible to identify the causative organism before antibiotics are started. Some common ENT problems along with antibiotics indicated are given in Table 2. In all cases, erythromycin can be substituted for penicillin/amoxicillin if necessary.

Acute tonsillitis	Systemic penicillin – amoxicillin is avoided because of the risk of inducing a rash in cases of glandular fever
Quinsy/peritonsillar abscess	Systemic penicillin and metronidazole, with drainage
Acute otitis externa	Topical antibiotic drops with steroids
Malignant otitis externa	Systemic antibiotics, dependent on culture results
Acute otitis media	Systemic co-amoxiclav
Acute sinusitis	Systemic co-amoxiclav or second-generation cephalosporin
Chronic sinusitis	Broad-spectrum antibiotics initially
Epiglottitis	Chloramphenicol or third-generation cephalosporin
Cellulitis	Systemic flucloxacillin and penicillin
Wound infection	Dependent on wound culture
Post-tonsillectomy	Co-amoxiclav

Table 2 Common ENT infections and suggested antibiotics

ASSESSMENT FOR THEATRE

Fitness for ENT theatre depends on the following factors:

- the nature of the operation and anaesthetic that will be given
- the urgency of that operation
- the health of the patient preoperatively.

Preparation for theatre

Preparation for theatre may include the following:

History of the patient

This should include a history of the complaint leading to the need for the operation and its current status. Note should also be made of smoking and drinking history, drug history and family history.

Full examination

This should include auscultation of the heart and chest, and an ENT examination. The mouth should be examined as ENT operations often involve risk of damage to teeth – a loose tooth can be dislodged. Furthermore, pre-existing damage to teeth should be documented.

Special investigations

History and examination may reveal the need for an electrocardiogram (ECG) (in those over 60 years or with a history of cardiac problems), a chest X-ray (in those with clinical signs of chest disease) or an echocardiogram (in those with clinical signs of heart failure, or unexplained heart murmurs). Cross-match, group and save, full blood count, electrolytes, thyroid function, sickle cell status and/or clotting may be appropriate, depending on the nature of the operation and the patient's medical and drug history.

Thromboembolic and antibiotic prophylaxis

ENT operations rarely require antithrombotic prophylaxis, but in a patient with a history of thrombosis and likely immobility after an operation, mechanical and chemical prophylaxis may be indicated:

- Mechanical – thromboembolism-deterrent (TED) stockings, early mobilisation.
- Chemical – low molecular weight heparin is the most common agent, which should be discontinued 24 hours prior to administration or withdrawal of epidural anaesthesia/analgesia to prevent bleeding into the epidural space.

Antibiotic prophylaxis is rarely indicated in ENT surgery, but it may be necessary in patients with previous cardiac problems, patients who are immunocompromised, or operations where infection is likely to be present in or around the operation site.

Consent

It is important that patients being treated are fully aware as far as possible of what interventions are being proposed for their condition. To a certain extent, patients give 'implied' consent to basic actions such as history taking and examination by simply attending a

medical facility. However, informed consent should be sought for any other intervention, as failure to do so may constitute an assault on the patient.

The procedure of obtaining informed consent may or may not involve the signing of a form, but in all cases the principles remain the same. An explanation of the following should be given:

- the details of the procedure
- the consequences if the procedure is not carried out
- the expected experiences during and after the procedure, such as pain levels
- the risks and potential side-effects, including a statistical likelihood if possible.

There should be an opportunity to ask questions.

Although it is often good practice to involve the family in any consent procedure, it is not mandatory. If an adult patient is not competent to give informed consent, the decision rests with the treating doctor to act in the patient's best interests. In these cases, all decisions must be carefully documented. Treatment may also be given in emergency situations without consent, as long as it is given in the patient's best interests. However, consent must be obtained as soon as the patient is capable of giving it.

For patients under 14 years of age consent should be obtained from the parents, although of course the child should not be left out of the conversation – a special consent form exists for the signature of the parents or appointed guardian (Form 3). For children aged 14 and 15, the doctor taking consent must make a judgement about whether the child is able to understand fully the procedure they are about to undergo, and all its implications and risks. The doctor should document this so-called 'Gillick competence' if the child is to sign the consent form.

A consent document is not legally binding; rather it acts as some degree of proof that a doctor has explained to a patient the nature of the procedure they are about to undergo and the risks of doing so. At any stage a patient may change their mind about proceeding.

There are certain situations where a doctor may treat without informed consent:

- A patient who is unconscious may be treated in their best interest, as long as no form of valid 'advance directive' exists to direct the medical personnel.
- Patients who are mentally incapable of making fully informed consent may be treated in their best interest – a special consent form exists for this purpose (Form 4), which is signed by the doctor only.

WOUNDS AND HEALING IN ENT

Wounds are created and repaired in the course of surgery, and it is essential to understand the process of wound healing, which consists of three phases:

- Acute inflammatory response – a clot is formed around the wound, with vasodilatation and influx of inflammatory cells, in particular neutrophils in the first 24 hours. Then the macrophages become more important, continuing the process of phagocytosis and secretion of cytokines (for example, transforming growth factor β (TGF-β), epidermal growth factor (EGF), platelet-derived growth factor (PDGF).
- Cell proliferation and deposition of extracellular matrix (proliferative phase) – fibroblasts secrete extracellular matrix and collagen; angiogenesis takes place, forming granulation tissue. There is wound contraction due to the action of myofibroblasts.
- Remodelling of the extracellular matrix (maturation phase) – lasts for many months and leads to a gradual increase in the strength of the wound, up to a maximum of 80%.

The commonest wounds in ENT surgery are of the face, an area that heals quickly given the right conditions. However, it is also an area about which the patient is highly sensitive with regard to slight asymmetry or changes in alignment. So, for the best result, care must be taken in the positioning of facial incisions to put the least possible tension on the healing wound. The skin has natural tension lines, and incisions placed on these lines tend to heal with a narrower and stronger scar, leading to more favourable results. In the face and neck they are most readily identified as the lines of wrinkling, and this fact can be used pre-operatively to mark the best possible position for an incision.

Delayed wound healing

Risk factors for delayed wound healing can be classified as follows:

- Local risk factors – infection, haematoma, mobility, foreign body, dirty wound, surgical technique, ischaemia.
- General risk factors – older age, cardiorespiratory disease, anaemia, obesity, diabetes mellitus, malnutrition, malignancy, steroids.

Poor healing may result in wound dehiscence, ie the partial or total disruption of layers of the operative wound.

Scars

All wounds form a scar in the process of repair. However, the healing response may become exaggerated. If excessive scar tissue is formed but is limited to the site of the original wound, a hypertrophic scar results. However, if the tissue extends beyond the boundaries of the original wound, a keloid scar results. Risk factors for hypertrophic and keloid scars include:

- young age
- black skin
- male sex
- genetic predisposition
- site – sternum, shoulders, head, neck

- wound tension
- delayed healing.

It is rare to find malignant change in a scar – if present, this is usually a squamous cell carcinoma, called 'Marjolin's ulcer'.

Drains in ENT

Head and neck surgery may result in wounds at risk of haematoma formation – if this is near the trachea, airway compromise may result, eg after thyroid surgery. To avoid this, drains may be placed in the area to minimise dead space. Drains in ENT are usually closed and often attached to a suction system, such as a Redivac drain. In the case of large abscesses, occasionally an open drainage system such as a Penrose or corrugated drain is left in situ after incision of the abscess.

HAEMATOLOGY

Haemostasis

Haemostasis is the cessation of bleeding, and involves a sequence of complex events:

- exposure of subendothelial tissue
- vasoconstriction
- adherence of platelets
- degranulation of platelets
- activation of the coagulation cascade
- platelet plug with fibrin support
- fibrinolysis and remodelling.

Platelets are a key factor – they bind to subendothelial collagen via von Willebrand's factor. They release their content(s), including fibrinogen and thromboxane A_2. The coagulation cascade is also a crucial factor, and it is shown in Fig. 4.

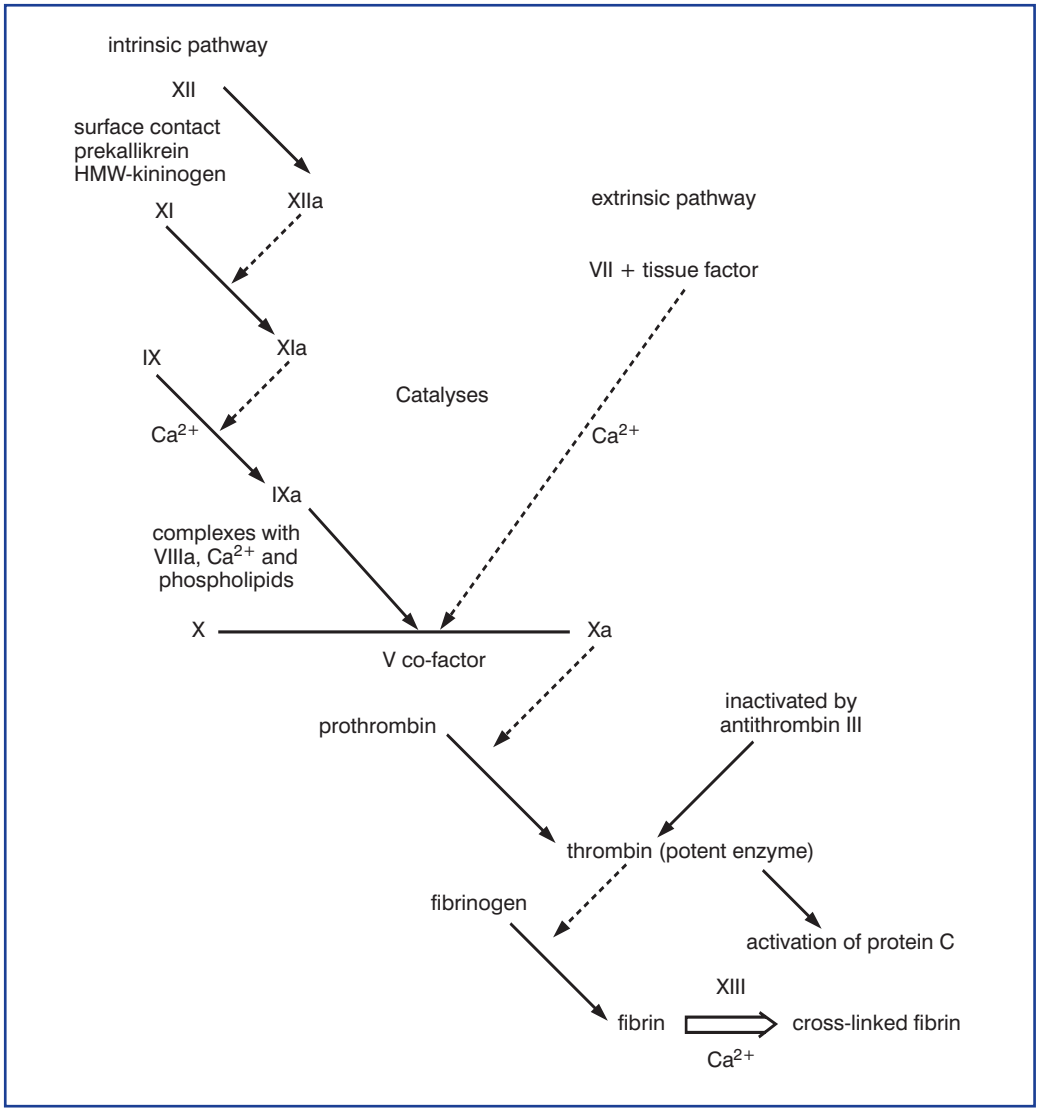

Fig. 4 The coagulation cascade

Fibrinolysis is carried out by plasmin, which is converted from inactive plasminogen by a number of factors, notably tissue plasminogen activator (tPA). Clotting time is measured by:

- activated partial thromboplastin time (APTT) – which measures the intrinsic as well as the common pathway
- prothrombin time (PT) – which measures the extrinsic as well as the common pathway
- thrombin time (TT) – which detects deficiencies of fibrinogen
- platelet count.

The way in which these times are altered gives a clue as to the underlying bleeding disorder, as shown in Table 3. Warfarin acts by blocking the synthesis of vitamin K-dependent factors and heparin activates antithrombin III, reducing fibrin formation – it is neutralised with protamine. Non-steroidal anti-inflammatory drugs (NSAIDs) affect the synthesis of thromboxane A_2 by platelets and thereby decrease their thrombotic action.

	PT	APTT	TT	Platelet count
Liver disease	Increased	Increased	Usually normal	Decreased
Warfarin	Increased	Usually normal	Normal	Normal
Heparin	Usually normal	Increased	Increased	Normal
Factor VII deficiency	Increased	Normal	Normal	Normal
Factor VIII deficiency	Normal	Increased	Normal	Normal
Factor XI deficiency	Normal	Increased	Normal	Normal
Disseminated intravascular coagulation	Increased	Increased	Increased	Decreased

Table 3 Changes in clotting times in various bleeding disorders

Haemophilia A is an X-linked deficiency in factor VIII, haemophilia B is also X-linked and is deficiency of factor XI – the two are clinically indistinguishable and are characterised by bleeding into soft tissue and joints after trauma. The commonest congenital bleeding disorder is von Willebrand's disease. There are also various congenital prothrombotic conditions – for example, protein C and S deficiencies, and antithrombin III deficiency.

Blood transfusion

Blood for transfusion comes from voluntary, healthy donors, and is known as 'whole blood'. However, it is usually split up into its constituent parts, namely:

- Red blood cell concentrates – plasma is removed, as it decays after a few days; this is the commonest transfused product but provides less volume replacement than whole blood; needs to be ABO-compatible.
- Platelet concentrates – can be used to treat thrombocytopenia or consumptive coagulopathy (eg disseminated intravascular coagulation (DIC)); needs to be ABO-compatible.

- Fresh frozen plasma (FFP) – contains all coagulation factors and can correct coagulopathies; needs to be ABO-compatible.
- Cryoprecipitate – produced by slow thawing of FFP; useful in the case of a fibrinogen deficiency, eg DIC.
- Factor concentrates – specific factor concentrates used to treat specific deficiencies.
- Albumin – only useful for diuretic-resistant oedema.

Red cells carry naturally occurring antigens, classified according to the ABO system. Other antigens may appear after sensitisation through transfusion or pregnancy. ABO incompatibility is the commonest cause of death due to transfusion – white cells or platelets may also produce a less severe transfusion reaction, due to the action of other antibodies. Other possible complications of blood transfusion are as follows:

- infection, eg HIV, hepatitis, human T-lymphotrophic virus (HTLV)
- fluid/iron overload
- hypothermia
- hyperkalaemia
- hypocalcaemia
- metabolic acidosis
- acute respiratory distress syndrome (ARDS)
- DIC.

ANALGESIA

Physiology of pain

Pain is defined as an unpleasant sensory and emotional experience associated with actual or potential tissue damage. There are four stages:

1 **Transduction** – tissue damage results in the release of arachidonic acid metabolites such as leukotrienes, prostaglandins and thromboxane A_2. These act at free nerve endings.
2 **Transmission** – free nerve ending stimulation transmits pain along A and C fibres.
3 **Modulation** – natural and synthetic opioids may act centrally or in the spinal column via opioid receptors to modulate the pain experience. Mechanical stimulation (eg rubbing) may also inhibit pain.
4 **Perception** – this occurs in the thalamus and sensory cortex.

Pain control

Pain is managed best by anticipating it, rather than by waiting for it to manifest itself. It often requires more than one analgesic agent. Options for analgesia are as follows:

- Opioids – these may be given orally, intravenously, intramuscularly or topically. They act at opioid receptors, and have many side-effects, such as constipation, nausea, respiratory depression and addiction. They may be given in the form of a patient-controlled system, which allows the patient to anticipate their own analgesic needs.
- Local anaesthetics – local infiltration into the wound, a regional block or an epidural block may produce profound analgesia but side-effects such as hypotension and respiratory depression should be monitored.
- NSAIDs – these have anti-inflammatory, analgesic and antipyretic properties; side-effects include gastric damage, bronchospasm and gout.

ANAESTHESIA FOR ENT

Anaesthesia is defined as the rendering of part or all of the body insensitive to pain or noxious stimuli. Anaesthesia for ENT procedures presents challenges that are slightly different in comparison with anaesthesia for other procedures.

General anaesthesia

ENT procedures often involve relatively minor procedures on relatively fit patients. However, major operations on head and neck cancers involve substantial risk to the airway in patients who smoke on a regular basis and have other anaesthetic risk factors. Other airway operations on patients with obstructive apnoea will require careful anaesthetic monitoring before, during and after the procedure. Insertion of a tracheostomy or correction of airway stenosis involves careful co-ordination with the anaesthetist to ensure a safe airway throughout the procedure. General anaesthesia induces narcosis, analgesia and muscle relaxation.

General anaesthesia – usual sequence

- Premedication/sedation
- Induction of anaesthesia
- Muscle relaxation
- Maintenance of anaesthesia
- Reversal of anaesthesia and recovery

Premedication

Premedication is not compulsory, but it may be necessary to lessen anxiety, dry secretions, prevent anaesthetic-related emesis or increase vagal tone. Common agents for pre-medication are:

- benzodiazepines, eg midazolam
- opioids, eg morphine

- anticholinergics, eg glycopyrrolate
- antacids, eg cimetidine.

Operations on the nasal passages often require vasoconstriction to decrease mucosal swelling, and this is achieved by application of an intranasal solution preoperatively. A common choice is Moffett's solution (a mixture of cocaine, adrenaline, sodium bicarbonate and saline).

Induction of anaesthesia

Anaesthesia can be induced by inhalational or intravenous (iv) agents. Propofol is a commonly used iv agent (thiopental sensitises the pharynx and cannot be used with laryngeal airways). Children may require inhalation of a gas such as halothane. These agents act quickly to lower the level of consciousness to one conducive to the operation. Complications of induction agents include laryngeal spasm that may exacerbate airway problems.

Muscle relaxation

Paralysis is sometimes employed in ENT procedures. **Depolarising muscle relaxants**, such as suxamethonium, act quickly on acetylcholine receptors to cause muscle fasciculation and then relaxation. Suxamethonium has a short half-life, but it cannot be used in patients with a history of bronchospasm, myasthenia gravis or malignant hyperpyrexia.

Non-depolarising agents such as atracurium have a slower onset but last longer. They can be reversed by means of neostigmine. Patients with myasthenia gravis are more sensitive to non-depolarising agents than other patients. For ENT procedures in patients with difficult airways, muscle relaxation should be used with caution as it may leave the patient apnoeic and needing ventilation, which may then be difficult to administer.

Maintenance of anaesthesia

This is usually achieved by inhalational agents:

- enflurane
- isoflurane
- sevoflurane.

Nitrous oxide is only a weak anaesthetic but potentiates the effects of other anaesthetics. Hence it is often used in conjunction with them. However, it diffuses rapidly into any air-containing space and so increases the pressure in the middle ear. This can lead to problems with middle-ear grafts, as their position may be altered by this increase in pressure.

Surgery that involves a risk of bleeding in the laryngeal or tracheal area presents a particular challenge for the anaesthetist, as it can precipitate airway compromise. Laser use in the airway carries a risk of fire, and precautions should be taken, such as the use of a metallic tracheal tube and filling of the cuff with saline.

Local anaesthesia

- This involves temporary blockage of transmission of nerve impulses by altering the permeability of the membranes of nerve cells.
- It may be used alone or in combination with a general anaesthetic to increase analgesia.
- Acidic solutions are used – which are prompted to work by the alkalinity of the surrounding tissue. So, in infected, acidic conditions they may not be effective.

Local anaesthetic may be administered in a number of different ways:

- topical, eg application of Moffett's solution to the nose for intranasal procedures
- direct infiltration, eg lidocaine for excision of superficial lesions or suturing
- nerve block, eg the superior laryngeal nerve (applying lidocaine to the pyriform fossae)

Epidural/spinal anaesthetics are not used for ENT procedures.

Complications

- Toxicity – inadvertent iv administration or over-rapid absorption may lead to perioral effects, which progress to drowsiness, seizures, coma and collapse; all local anaesthetics have maximum doses dependent on weight – this maximum may be increased by the use of agents such as adrenaline, which slow absorption and prolong period of action.
- Allergic reaction – this usually causes an immediate reaction and may lead to bronchospasm, laryngeal oedema or cardiovascular collapse. Adrenaline and iv fluids should be given, and antihistamines and bronchodilators may be necessary.

OPERATING EQUIPMENT FOR ENT SURGERY

As in all branches of surgery, ENT has many specialised operating tools. These can be classified as follows:

- equipment used to carry out procedures in the outpatient department, eg wax hooks, microsuction, nasal specula, cautery sticks
- equipment used to carry out particular operations, eg ear micro-instruments, tonsil retractors, laryngoscopes, oesophagoscopes
- airway adjuncts, eg tracheostomy tubes, stents
- aids to visualisation, eg microscopes, nasoendoscopes, functional endoscopic sinus surgery (FESS) instruments.

Laser

Lasers are being used increasingly in ENT surgery. Laser stands for **L**ight **A**mplification by **S**timulated **E**mission of **R**adiation, and provides a way of directing energy to a very specific point. This energy can be focused using lenses, and its effects on surrounding tissue vary according to the type of laser used, the duration of action and the density of the beam.

Uses of laser in ENT surgery

- Ear – fine surgery around ossicles and stapedectomy, welding of grafts.
- Nose – cautery, dacryocystorhinostomy, management of hereditary haemorrhagic telangiectasia (HHT).
- Throat – excision of benign lesions, debulking of tumours.

IMAGING/RADIOLOGY

ENT uses all modes of imaging to aid diagnosis and treatment.

Plain X-rays

These may be used in the case of sinus disease but have now been superseded by computed tomography (CT) scans. Lateral neck X-rays are often taken in cases of suspected foreign bodies in the throat. However, there is often very little useful information to be gained as foreign bodies may not be radio-opaque and may be confused with normal structures in the throat.

Computed tomography

CT scans provide information about bony structures and soft tissues in the head and neck area. Sinus disease is well visualised, and iodine contrast provides further information about abnormal blood flow in lymph node enlargement and neoplasia. Dense bone in the skull base area is not well visualised, but the temporal bone is well seen.

Magnetic resonance imaging (MRI)

MRI performs well in imaging soft tissue. It works by aligning protons in living tissue in magnetic fields, which then relax when the field is turned off and produce radio waves that can be received and analysed. The signal is composed of two components, T1 and T2.

- T1 component relates to the time taken for spinning protons to return to their normal position – T1-weighted images provide high anatomical definition, including the soft tissues.
- T2 is related to excited protons moving out of phase with each other – T2 images show up abnormal tissue better, such as inflamed and neoplastic tissue.
- Short tau inversion recovery (STIR)-weighted images are similar to T2 but with fat signal suppression, which can provide clearer information about the outline of tumours.
- Gadolinium is a commonly used contrast agent and, like iodine, shows up areas of increased vascularity.

Ultrasound

Ultrasound is an effective, quick and safe method of imaging soft-tissue swellings in the head and neck area. It can be used in conjunction with fine-needle aspiration to provide further information. It is, however, very operator-dependent.

Contrast studies

Barium swallows are commonly done for the assessment of swallowing problems, pharyngeal webs and pharyngeal pouches. If aspiration, perforation or leakage is a risk, water-soluble contrast can be used.

IMMUNOLOGY

Physiology of the immune system

Stimulation of the immune system by exposure to pathogens leads to increased resistance to that pathogen in the future. The components of the immune system can be classified as:

- **non-specific defences**, such as skin, complement system and neutrophils
- **specific defences**, such as lymphocytes, which are triggered by exposure to antigens.

The sequence of events in the immune response is as follows :

1. Antigens are 'presented' by any cell using the class I major histocompatibility complex (MHC) molecule (Table 4) or by dendritic cells and macrophages, which use the class II MHC molecule.
2. A peptide fragment of the antigen is then combined with the relevant MHC molecule and expressed on the cell surface.
3. Class I MHC molecules are recognised by CD8+ T lymphocytes (cytotoxic T cells) which kill cells infected with the pathogen. Class II molecules are recognised by CD4+ T-helper cells, which produce soluble immune system mediators such as interferons and interleukins.
4. These substances then stimulate various specific aspects of the immune system. B lymphocytes are stimulated as a result and differentiate into plasma cells. These cells secrete immunoglobulins, helped by T-helper cells.

MHC type	Found in	Recognised by	Effect
Class I	Every cell	CD8+ T-cytotoxic cell	Kills infected cells
Class II	Macrophages/dendritic cells	CD4+ T-helper cell	Production of immune system mediators

Table 4 Major histocompatibility complex class I and II

Structure of immunoglobulins

Immunoglobulins are antibodies that have the structure shown in Fig. 5. They are made up of a constant and a variable region, or can also be thought of as a light and heavy chain, or an Fab (antigen-binding) fragment and an Fc (complement) fragment. The binding of antibodies to antigen leads to lysis of bacteria, initiation of the classical complement pathway and killing of the infected cell, among other effects.

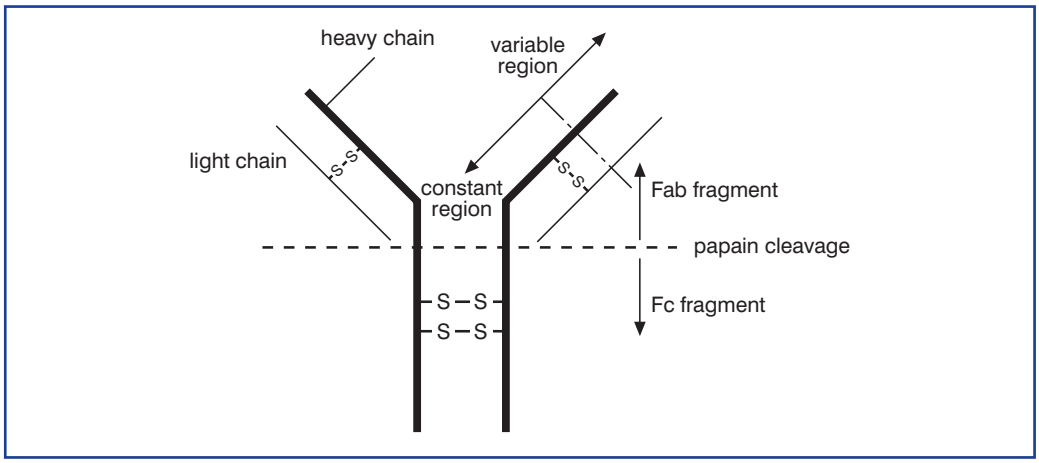

Fig. 5 Antibody structure

Classification of immune responses

The immune system may respond inappropriately to activation by antigens in a **hypersensitivity reaction**. These responses have been classified by Gell and Coombs as shown in the box.

- **Type I (anaphylactic or immediate)** – exposure leads to IgE formation. This binds to mast cells and basophils, leading to release of mediators on subsequent exposure. Hay fever, anaphylactic reaction and allergic rhinitis are examples. Sodium cromoglycate and steroids may act to inhibit mediator release by stabilising lysosomal membranes.
- **Type II (cytotoxic) –** this is mediated by antibodies against antigens, and results in the activation of cells themselves or of complement, leading to cell damage. Examples are graft rejection and transfusion reaction.
- **Type III (immune complex-mediated)** – immune complexes of antigen with antibody lead to complement activation and tissue damage. An example is systemic lupus erythematosus (SLE).
- **Type IV (cell-mediated/delayed)** – this reaction is mediated by sensitised T lymphocytes, leading to T-cell activation and recruitment of macrophages, resulting in damage. TB and transplant rejection are examples.
- **Type V (stimulatory)** – antireceptor antibodies stimulate cell function. Examples are Graves' disease and myasthenia gravis.

PHYSIOLOGY FOR ENT

Cardiac physiology

In any surgical specialty, a basic knowledge of physiology is important. In particular, cardiovascular and respiratory function may be relevant to a patient's pre-, peri- and post-operative management.

Heart muscle is specialised to perform involuntary pulsatile contraction. Its structure is similar to skeletal muscle, ie it is made up of sarcomeres, which, in turn, are made up of thick (myosin) and thin (actin) filaments. The force that each sarcomere can exert partly depends on the initial length of that sarcomere. In other words, when the sarcomere is very short, or very long, less force is exerted. There exists an optimal length of sarcomere at which maximum force is exerted on contraction. **Starling's curve** (Fig. 6) is based on this characteristic.

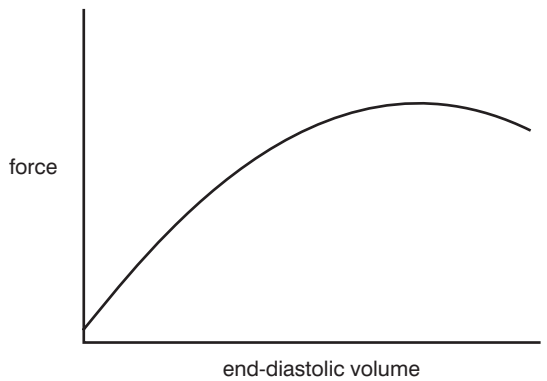

Fig. 6 Starling's curve

Up to a point, therefore, increasing the blood volume in the heart can increase the force of contraction of the heart. Cardiac output and stroke volume also vary in the same way with end-diastolic volume. Another factor that increases contractility of the heart is the concentration of calcium ions in the cells – more intracellular calcium results in a greater force of contraction.

Cardiac cells transmit a contraction impulse between each other and so contract in a synchronised fashion. A wave of depolarisation passes through the heart by this means. Contraction is initiated by the sino-atrial and atrioventricular nodes as well as the Purkinje fibres in the ventricles. All these cells have their own intrinsic rate of impulse generation (self-excitation). The sinoatrial node fires the fastest, at a rate of around 100 beats per minute (bpm). The atrioventricular node is next, and the Purkinje fibres fire at 40 bpm. If the sinoatrial node fails to fire, the cells with the next highest rate of firing will take over.

Vagal activity slows the intrinsic firing rate and sympathetic hormones and sympathetic innervation increase it. Sympathetic nerve fibres (C8–T5) cause noradrenaline release at nerve endings, acting on cardiac β receptors, increasing heart rate and force. Parasympathetic fibres (via the vagus nerve) cause acetylcholine release, which acts on muscarinic receptors and causes a slowing of the heart rate.

Regulation of stroke volume takes place due to the following factors:

- Preload – Starling's Law implies that the initial fibre stretch of the cardiac muscle will influence stroke volume. This in turn depends on filling time of the atria and how much compliance (stretchability) is present.
- Contractility – this is increased by preload, the presence of hormones such as adrenaline, thyroxine and glucagons, and by certain drugs. It is decreased by hypoxia, acidosis and alkalosis, electrolyte imbalance, and of course by drugs.
- Afterload – this is the tension of the ventricular walls required to eject the blood into the systemic circulation. Aortic stenosis and increased systemic vascular resistance increase afterload and vasodilators decrease it.

Transport of oxygen

- Oxygen is mostly transported bound to haemoglobin.
- Haemoglobin contains four O_2-binding haem molecules.
- A small amount of O_2 is also transported dissolved in plasma
- The oxygen dissociation curve (Fig. 7) determines the relation between O_2 saturation and Pa_{O_2}.

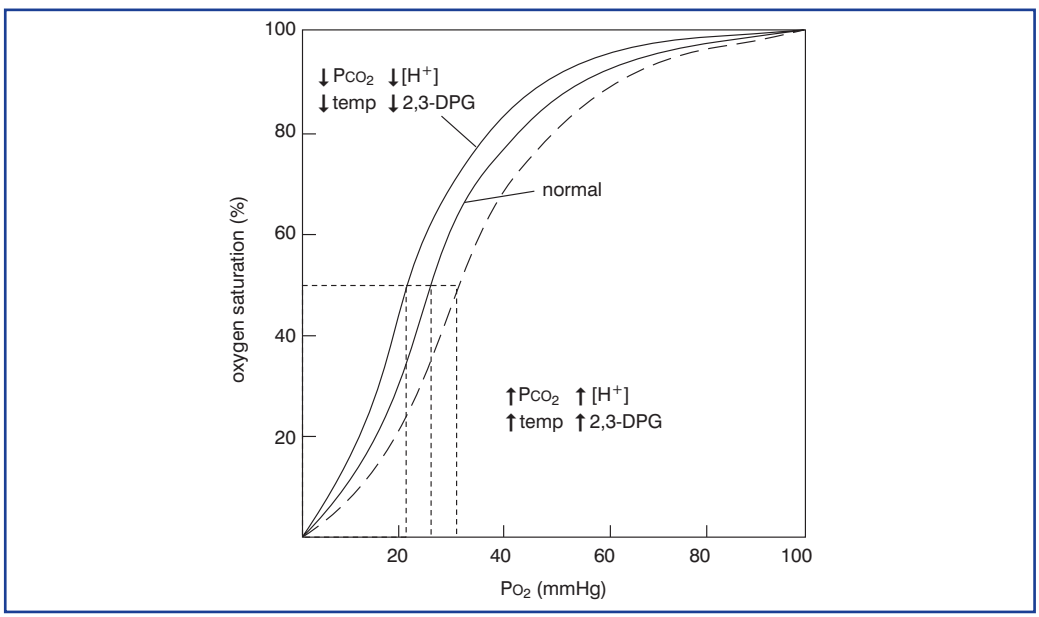

Fig. 7 Oxygen dissociation curve

Factors shifting curve to the left (increased affinity for O_2):

- decreased $Paco_2$
- decreased $[H^+]$ (Bohr effect)
- decreased 2,3-DPG
- decreased temperature
- HbF, carboxyhaemoglobin.

The opposite will shift the curve to the right.

Transport of carbon dioxide

CO_2 is transported in three ways:

- dissolved as CO_2 in plasma
- reacts with amines in deoxyhaemoglobin to form carboxyhaemoglobin
- reacts with H_2O to form H^+ and HCO_3^-, transported as sodium bicarbonate.

Deoxyhaemoglobin is a weaker acid than oxyhaemoglobin and can carry more CO_2 (Haldane's effect).

Control of respiration

Respiration is controlled by a number of feedback loops based on chemo-, baro- and mechanoreceptors, as well as voluntary control. These mechanisms as shown in Fig. 8.

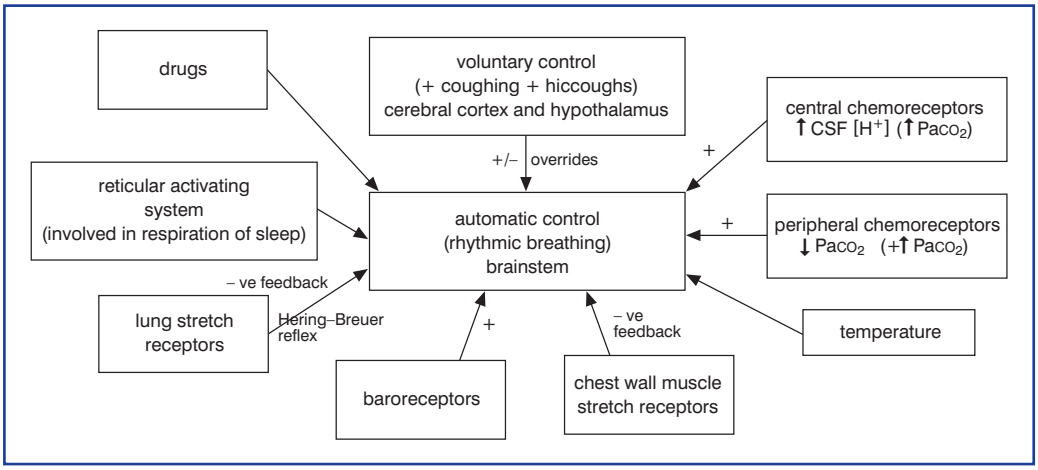

Fig. 8 Control of respiration. Hering–Breuer reflex = negative feedback from lung stretch receptors as the lung inflates

AIDS TO VENTILATION

If a patient's ventilation is inadequate, intubation of some kind may be necessary. Endotracheal intubation is indicated in the following cases:

- Glasgow Coma Scale (GCS) score < 8
- impaired gag reflex
- to prevent a rise in intracranial pressure (ICP)
- to enable suction of secretions
- severe hypoxia or metabolic acidosis
- risk of upper airway obstruction.

A cuff prevents aspiration, but may cause stenosis and tracheomalacia if the pressure is too high. When endotracheal intubation is not possible, a surgical airway may be necessary. In an emergency, **surgical cricothyroidotomy** is performed, ie an incision made in the cricothyroid membrane and a cuffed tube is inserted. In a more controlled situation, there are other options:

- Surgical tracheostomy – an incision is made through the second and third tracheal rings. It is indicated in the following situations:
  - when weaning off an endotracheal (ET) tube
  - to enable suction of secretions
  - chronic ventilation
  - to facilitate oral care.
- Percutaneous tracheostomy – this technique involves progressive dilation of a puncture hole in the trachea, which does not require transfer to theatre, and may be performed by doctors in an intensive care unit (ICU).
- Mini-tracheostomy – a small tracheostomy tube is inserted in the cricothyroid membrane to enable suctioning of secretions, but it is not suitable as a definitive airway as it is not cuffed.

CLINICAL GOVERNANCE AND AUDIT

The concept of clinical governance is a relatively new one. It complements the increased autonomy of financial governance that has been introduced in the British health system, and **gives clinicians responsibility for monitoring their own group and individual performance and improving that performance continuously**. Various aspects of clinical governance include:

- audit
- evidence-based medicine (EBM)
- continuing education
- teaching
- research
- risk management.

Audit

For surgeons in training, audit is one of the commonest methods by which they will be involved in clinical governance. Audit is the process by which clinical staff collectively review, evaluate and improve their practice with the aim of improving standards. From this definition follows the structure of the audit cycle (Fig. 9).

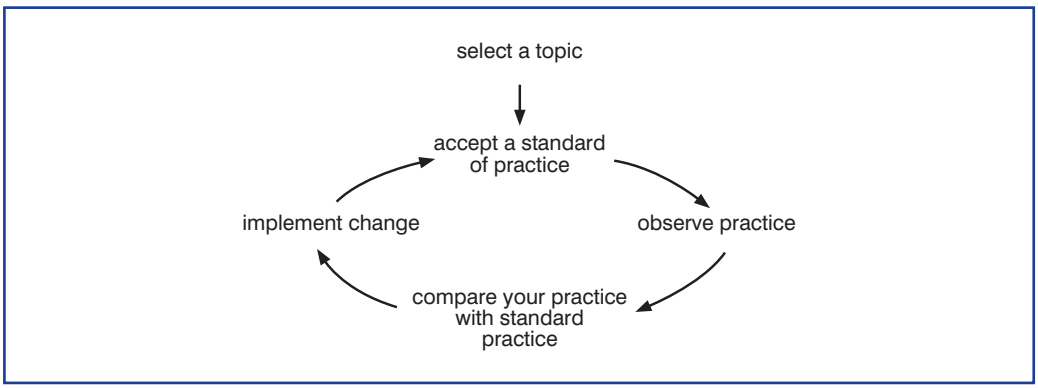

Fig. 9 Audit cycle

Evidence-based medicine

EBM is the practice of basing medical decisions on research. It involves:

- formulating the clinical question to be answered
- gathering evidence on this question
- assessing the quality and applicability of the evidence available
- arriving at a conclusion about the appropriate decision for the individual case being considered.

This process includes a critical appraisal of the literature involved. Double-blind randomised controlled trials represent the best quality evidence, but these may not always exist or even be possible. Studies may be subject to bias not mentioned in the publication, for example:

- Publication bias – negative findings are less likely to be published.
- Response bias – those not responding may be at the extreme ends of the range of subjects studied.
- Analysis bias – randomised controlled trials should be analysed on an intention-to-treat basis, not according to what treatment the patient finally ended up receiving: this is because, in real life, patients are free to change treatments, and it is the initial management strategy that is being assessed.