Ear, Nose and Throat: Out of Africa

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Foreward

This book is a comprehensive introduction to ENT diseases for registrars, headache specialists, dentists, interns, GPs and orthodontists.

A wealth of high quality informative drawings bring the text to life with highlighted areas of special interest.

Each chapter is a complete unit in itself. We have endeavoured to compile a reader friendly book that may generate further interest in the field of otolaryngology.

Writing a book is an adventure.
To begin with, it is a toy and an amusement.
Then it becomes a mistress, then a master,
then it becomes a tyrant.

The last phase is that just as you are about
to be reconciled to your servitude,
you kill the monster, and fling him to the public.

Sir Winston Churchill

Ear

Pinna

Congenital deformities of the pinna

Pinna abnormalities may vary from minimal cosmetic aberrations (ie, bat ears), through gross deformities and malpositioning in relation to the bony external ear canal, to total absence. They may be associated with other facial and systemic congenital syndromes. Accompanying bony aberrations of the ear capsule may account for ear canal stenosis (either cartilaginous, bony or combined), which in turn may account for a conductive hearing loss. This sequence of events may affect one ear or both simultaneously. It is important to establish the presence of either cochlea or middle ear abnormalities before proceeding to surgical correction.

Management of minor deformities

Pre-auricular tag is a congenital malformation of the second branchial arch. It may be broad based or sessile and may contain a skin covered fibrous polyp with or without cartilage.

Pre-auricular sinus often becomes infected and may extend up to 2.5 cm below the skin surface. It may occur close to the facial nerve. Surgical excision is the treatment of choice. Histology may indicate complete removal of the sinus tract.

Management of larger deformities

Because function is more important than cosmetic appearance, functional correction is usually performed prior to cosmetic correction. Preoperatively parents and/or patients should be informed about the surgical limitations of the final cosmetic outcome. Microtia and macrotia (ie, too small or too large a pinna) is best left untreated if the pinna is not protruding. If it is, a bat ear correction may be advised.

Surgical correction of minor congenital aberrations may give gratifying results: for example protruding ears (bat ears) may be restored to give the patient a normal appearance. Careful assessment is necessary in the case of more severe deformities as these may need specialised reconstruction. Staged surgery may be necessary for good results.

1. Bat ears and other abnormal pinna shapes may be a cause of social hang-ups and teasing at school, ie, being dubbed as 'Dumbo' or 'windmill ears'.

2. Malalignment (pinna too low) may be associated with external ear canal stenosis - bony or cartilaginous. Middle ear abnormalities may be present.

3. Microtia (pinna too small) may be associated with other congenital ear abnormalities, ie, external ear canal stenosis or middle ear ossicular deformities. Conductive deafness may be present.

4. Macrotia (pinna too large) usually develops with ageing - 'grandmother, why are your ears so big'.

5. A pre-auricular tag may contain cartilage remnants or may consist of a skin tag only. This condition may be an isolated finding or part of other congenital ear deformities.

6. Pre-auricular sinus often becomes infected and may extend up to 2.5 cm below the skin surface.

Pinna trauma

Acute trauma of the pinna

The pinna is a prominent feature of the skull and may be exposed to various types of injuries.

Skin abrasions are usually the result of shearing forces (ie, motor vehicle accidents or sports injuries). The degree of injury depends on the magnitude of the physical force. Concomitant subdermal pigmentation by street dirt or gravel may be present. An antitetanus booster is indicated in street and sportsfield injuries.

Lacerations may be linear or jagged to various degrees. Portions of the pinna may be avulsed. Smaller segments may be re-attached as free grafts with unpredictable results.

Avulsions of larger segments have poor results when re-attached. Microvascular anastomosis may establish vascular circulation with unpredictable success rates. Therapy for ear avulsions is standard wound care and closure. If the wound is older than 12 hours it should be treated as 'open'.

Treatment

Lacerations may be primarily repaired. Debridement of devitalised tissue and approximation of skin edges with 5/0 nylon sutures may avoid infections and restore cosmetic appearance. The pinna cartilage have to be trimmed back from the edge of the laceration to ensure skin closure over the remaining cartilage. Small avulsed segments measuring less than 1 cm x 1 cm may be re-attached as composite grafts although the result is often not optimal. Larger segments may need microvascular surgery to re-establish blood circulation. Free skin grafts may be transplanted onto the pinna to cover bare cartilage so as to prevent infection.

If there is a delay between the accident and surgical repair, the avulsed segment should be kept chilled in saline to reduce cellular breakdown and cell necrosis.

Local wound care and systemic antibiotics may reduce infections.

A loose protective dressing may protect the pinna against pillow or mechanical friction during sleep.

Neglected infection from perichondritis may spread systemically with grave results. Trauma of the pinna may also be an early sign of child abuse. Further examination of the child's body for other evidence of physical abuse should be conducted.

Iatrogenic trauma

Piercing of the lobe of the pinna with unsterile instruments may promote infection and keloids with resulting cosmetic deformities.

An earring torn out of the ear lobe may result in a jagged laceration. A stretched earring hole may result from wearing too heavy earrings. These cosmetic defects may be repaired by minor surgery with local anaesthesia.

Chronic recurrent minor pinna trauma (ie, pinna pulling and/or folding) may result in a pseudocyst of the pinna or a haematoma auris. A seroma may follow incomplete absorption of the pseudocyst.

Cauliflower ear

Chronic repeated trauma to the pinna results in subperichondrial haematoma, which may be organised into scar tissue. Surgical correction of this deformity seldom has cosmetically acceptable results. Resultant narrowing of the external ear canals may lead to

wax accumulation, hearing loss and water entrapment. These cases may be predisposed to recurrent ear canal infection.

Thermal burns

With thermal burns, the prognosis depends on the extent and depth of tissue destruction. *Pseudomonas* and *Staphylococcus aureus* are both common secondary pathogens which may spread systemically, especially in the immunocompromised patient. Acute treatment consists of debridement, local antibiotic lotions, soft protective dressings, systemic antibiotics and pain control.

Bites

Animal bites may be lacerations or punctures, and segments of the pinna may be avulsed.

Treatment is the same as discussed above. If rabies is suspected the necessary prophylaxis should be implemented (ie, immediate vaccination). Human bites may carry a high risk of wound infection and HIV transmission. The most famous human bite was the Holyfield-Tyson ear bite incident during a world heavyweight boxing match in 1996 in Nevada, USA.

Frostbite

Prolonged exposure to cold may result in frostbite and partial loss of the pinna. Crystals form in the small capillaries of the pinna and the condition is accompanied by reduced blood circulation and ischemia. Initially the pinna is blanched and painless and reheating it may cause pain. The pinna then becomes red and blistered. Analgesics may be indicated during the thawing stage for pain control.

In warmer climates frostbite may occur as an occupational exposure (ie, cold storage workers).

Treatment

A wait-and-see period will determine and demarcate the extent of the tissue damage and tissue loss. Debridement and standard local wound care is indicated. Cottonwool padding and bandaging of the pinna may help control pain.

1. Skin Abrasions: The pinna skin may be injured to varying degrees by a shearing force (ie, sports injuries or motor vehicle accidents). An antitetanus booster may be required for street wounds.

2. Blow to the pinna: A fist blow to the pinna may result in a perichondrial haematoma of both the pinna and ear canal. A fist blow may not seal effectively onto the ear canal to generate a pressure wave strong enough to lacwerate the eardrum as a flat hand blow may do.

3. Pinna avulsions: Partial avulsions may be sutured with good cosmetic results. Smaller segments may be re-attached as free grafts with unpredictable results. Re-attachment following avulsions of larger segments yields poor results. Microvascular anastomosis may establish vascular circulation with unpredictable success rates.

4. Ear lobe disfiguration. An earring torn from the lobe may result in disfigurement. Surgical correction yields good results. An elliptical overstretched earring hole can be surgically corrected. A revision piercing for an earring may be done once the lobe has healed.

5. Cauliflower ear: A cauliflower ear is a cosmetic blemish that may result from an organised perichondrial haematoma. It is difficult to correct surgically.

6. Bite injuries to the pinna: Human bites may transmit mixed flora and are more difficult to manage than animal bites. HIV transmission from a human bite is a realistic possibility.

7. Frostbite: Frostbite injuries may result from prolonged exposure to cold, resulting in sluggish blood circulation, oxygen deprivation and tissue necrosis. It is an extremely painful condition. Secondary infection must be ruled out with appropriate local wound care and systemic antibiotics.

Pinna infection

Bacterial infection

In a large proportion of cases the point of entry may be iatrogenic (ie, postoperative surgical wound infections or piercing of the pinna or the lobe with unsterile instruments). Street wounds and sports injuries are less frequent causes of infection of the pinna. The ear lobe becomes oedematous, hyperemic, painful and feels doughy. The pinna is 'pushed' forward. Early infection control with local wound care as well as systemic antibiotics may prevent adverse local and systemic complications that may result in various degrees of pinna cartilage damage and subsequent cosmetic deformities.

Early drainage of a pinna haematoma under sterile conditions may prevent wound infections. Neglected suppurative perichondritis may metastasize systemically with grave results. Supportive soft padding of the pinna may help control pain.

Viral infection: Herpes zoster oticus

Unilateral vesicular eruption may occur on the pinna and around the external ear canal, the face, the soft palate and the tongue. The vesicles form crusts that drop off at about 21 days.

Accompanying facial nerve palsy (Ramsay Hunt syndrome) may need additional eye care. Complications may include sensorineural hearing loss and vertigo.

Treatment

This is an ENT emergency; delayed therapy may result in a permanent facial palsy. Palliative treatment is indicated for pain, fever and malaise during the onset of the syndrome. The vesicles on the pinna are best left alone. Warm compresses over the pinna may ease the pain and discomfort. If facial nerve palsy is present, eye care is implemented to protect the cornea from dehydration and corneal ulceration that may lead to corneal scarring and loss of vision.

Synthetic tears may be used to lubricate the cornea. An eye pad comforts the patient because a micro moist climate is established. Steroids given in a tapering dosage may reduce post herpetic neuralgia. Steroids are contraindicated if herpes zoster ophthalmicus is present.

Antiviral agents may reduce facial palsy, synkinesis and neuritic pain.

Dermatosis: Psoriasis

Psoriasis is a genetic inflammatory skin disorder and may appear as an isolated local disorder of the concha skin of the pinna and external ear canal or as an extension of systemic skin presentation. Psoriasis of the pinna may fluctuate in intensity and may spontaneously disappear. Itchiness may provoke scratching with secondary skin infection. The systemic presentation of psoriasis is beyond the scope of this chapter.

Treatment

Control the ear canal itchiness with systemic antihistamines and preclude water from the external ear canals. Scratching of the ear canal with a foreign body (ie, a matchstick or a cotton bud), may excoriate the psoriatic ear canal skin and may cause secondary infection. Regular cleaning of ear canal debris and local application of non-fluorinated steroid cream may control the scaliness and itchiness.

Psoriasis remains a part of a systemic disease.

Chondro-dermatitis nodularis chronica helices of the pina

This is an acquired unilateral condition usually in adult males. Macroscopically it appears as a raised tender solitary nodule on the free edge of the antihelix. It may be tender and painful to touch. It may resemble a basal cell or a squamous carcinoma.

Microscopy reveals an ulcerated central core. This is not a premalignant condition.

Treatment

Treatment is by primary excision including the cartilaginous projection. The specimen should be forwarded for histology to exclude a carcinoma. Recurrence is extremely unusual but occasionally the contralateral ear acquires a similar lesion.

Pinna tumours

Benign tumours

Chondroma

Chondroma is an acquired slow-growing subcutaneous painless mass on the pinna or in the external ear canal. It is removed by primary excision. Histology may confirm the diagnosis.

Keloid

A **keloid** is an acquired post traumatic skin lesion especially prevalent among pigmented races. Ear piercing or an otoplasty may induce a keloid on the pinna. Clinically the keloid may vary in size. It is painless, smooth and firm. The covering skin is normal in appearance.

Treatment is by primary excision and skin closure without tension. Infection control is important to prevent a recurrence.

Closure must be infection free and tension free. A short course of radiotherapy during the immediate post-operative healing phse may prevent recurrence of the keloid.

Malignant tumours

Squamous cell carcinomas

Squamous cell carcinomas arise in older Caucasian males with outdoor occupations (ie, golfers, farmers, fishermen), commonly on the helix, antihelix and concha.

Clinically a carcinoma appears as a painless elevated indurated ulceration with raised margins and sometimes cartilage invasion. Lymph node metastasis is aggressive to the pre-auricular glands and the vertical chain of neck glands, but it may also invade the temporal bone. Public awareness of the increasing occurrence of carcinomas and melanomas should be promoted via public education stressing the ill effects of sun rays on light pigmented skin, avoidance of sun tanning, using ample sunscreen and wide protective hats. Sun rays are most damaging between 11 am and 3 pm.

In recent years skin cancers have increased alarmingly in Caucasians living in sunny countries. Further increases are likely as the ozone layer depletes. In almost all cases small skin cancers can be cured with local wide excision and generous margins.

Treatment

Upon confirmation of the diagnosis, surgery should be aggressive. Generous margins are necessary, especially with larger lesions. Larger excisions may necessitate skin flaps and skin grafting, usually from behind the ear for colour and texture matching. Recurrence may be possible. Metastasis is related to the size of the tumour. Neck gland dissection is only

warranted with positive nodes.

Basal cell carcinoma

Basal cell carcinoma is the second most common pinna malignancy after squamous cell carcinoma. Although they may be locally aggressive, they never metastasize.

Treatment

Wide local excision with a clear margin is recommended. Radiotherapy is not indicated as it may precipitate a radiochondritis.

Melanomas

These tumours are rare on the pinna and more frequently encountered in redgeads or blondes, Caucasians and males. Melanomas have a strong tendency to metastasize and have unpredictable biological activity. Surgically amenable cases should be aggressively removed with subtotal or total auriculectomy with a prophylactic neck dissection and a concomitant parotidectomy. Chemotherapy may be considered.

In contrast to squamous cell carcinoma and basal cell carcinoma which presents on the upper rim of the helix, melanoma of the pinna may appear anywhere on the pinna. In Caucasians there is an alarming increase in melanomas. Warning signals are itchiness and redness of the melanoma.

Treatment

Excision is locally aggressive. The thicker the tumour on histology the wider the excision. In thick tumours an elective regional lymph node dissection may be prudent for possible occult metastatic disease.

1. Bacterial infection - acute perichondritis: Perichondritis may be diffuse or localised and may follow chronic pressure or be secondary to external ear canal infections. The ear feels puffy and doughy. The infection may metastasize systemically if not adequately treated.

2. Viral infection - herpes zoster oticus. Incomplete immunity to the chicken pox virus may result in a relapse infection. Serosanguineous vesicles may appear on the concha of the pinna and on the palate. The facial nerve may be affected causing unilateral facial palsy (Ramsey Hunt syndrome). The eye may also be affected due to facial palsy. Inability to close the eyelids may cause dryness of the cornea with possible corneal ulceration.

3. Psoriasis may mimic chronic otitis externa and may be associated with systemic psoriasis.

4. Chondro-dermatitis nodularis chronica helices usually follows skin trauma over cartilage and may result in a non healing ulcer. It may be confused with a malignant lesion. An excision biopsy is warranted to confirm the diagnosis.

5. Ear lobe keloid. An ear lobe keloid may result from an ear piercing procedure. After surgical removal a recurrence of the keloid may occur.

6. Squamous cell carcinoma usually occurs in light skinned persons with sun exposure (ie, farmers, golfers and processional sportsmen) and is exclusively a disease of Caucasians. Skin cancers have increased alarmingly in Caucasians living in sunny countries. Further increases are likely as the ozone layer depletes. In almost all cases small skin cancers may be cured with local wide excision and generous margins.

7. Basal cell carcinoma usually occurs in light skinned persons with excessive sun exposure (ie, farmers, golgers and professional sportsmen). With adequate margins surgical excision is successful in almost all cases.

8. Melanomas: These tumours are rare on the pinna and more frequently encountered in redheads, blondes, Caucasians and males. Melanomas have a strong tendency to metastasize and have unpredictable biologic activity. Surgically amenable cases should be aggressively removed. Melanomas are the fastest increasing occurrence of tumours in Caucasians.

External Ear Canal

Hair follicles and accessories

The external ear canal hairs become progressively fewer and thinner towards the medial end of the ear canal (ie, closer to the eardrum). The hair follicles receive and transport the secretions of the sebaceous and apocrine glands to the surface of the canal skin where they mix with the exfoliating canal skin cells and form wax. Wax consists of amino acids, cholesterol, lipids and trace elements. Wax secretion differs in quantity and quality from person to person and is racially distinct.

Wax protects the skin of the ear canal against bacterial infection. Under certain conditions (ie, prolonged contact with water as with swimming), the ear canal wax may dissolve, sometimes allowing bacteria from contaminated water to spread along the hair shaft to the hair follicle and reach the ceruminous glands, causing infection (ie, otitis externa or a furuncle).

Congenital deformities

Stenosis or atresia of the external ear canal may be unilateral or bilateral and may accompany middle ear, ossicular, or inner ear malformation. These deformities may be fibrous, cartilaginous, bony or a combination of tissue. The resultant hearing loss may be conductive, sensorineural or a mixed loss. In patients with normal contralateral hearing, correction of unilateral atresia may be postponed to adulthood.

Treatment

Congenital canal malformations should be corrected no sooner than six years of age. In the case of bilateral canal atresia, only one canal should be corrected to allow the patient to perceive speech frequencies. The contralateral ear canal may be corrected during adulthood.

Preoperative evaluation with CT and MRI scans should be undertaken to evaluate the status of the inner ear canal, the inner ear, middle ear space, ossicles, external ear canal and facial nerve. Little can be achieved if the scans reveal that the cochlea is not present or calcified; and in these cases surgery should be avoided to prevent the risk of damage to the facial nerve. Parents should be advised of the results realistically achievable, success rates and possible dangers to the facial nerve.

Trauma

Trauma to the external ear canal may be caused by superficial skin lacerations due to scratching with a foreign instrument (eg, cotton buds, a knitting needle or a match stick).

High velocity bullet or shrapnel wounds may cause skin lacerations as well as concomitant temporal bone fractures of the bony external ear canal. Skull base fractures involving the bony external ear canal can result from a high energy impact blow to the skull (ie, motor vehicle or motorcycle accidents). Concomitant intracranial injuries may overshadow the external ear canal trauma and bloody discharge, or a blood clot in the external ear canal may obscure the trauma to the bony external ear canal. If a clot is present, it should be left alone and removed only under sterile theatre conditions to avoid secondary infection with intracranial spread of the local infection.

Seborrhoea

This is a hereditary chronic inflammatory skin disorder that occurs in areas where there is a high concentration of sebaceous glands and is often part of a systemic seborrhoeic skin disorder, ie, seborrhoeic dermatitis. The ear canal may present with a greasy, scaly, chronically inflamed appearance.

Patients have a constant urge to scratch the ear canal with a matchstick or similar instrument. When it occurs on the scalp, the condition is called dandruff.

Treatment

Topical non-fluorinated steroid creams will control the itchiness. If a secondary infection is present, a topical antibiotic/steroid cream and a systemic antibiotic may be necessary to control acute ear canal infection.

Scalp dandruff must simultaneously be controlled with a scalp shampoo. If uncertainty exists as to whether the condition is psoriasis or seborrhoeic dermatitis, treat for both simultaneously.

Psoriasis

As already mentioned under psoriasis of the pinna, this is a hereditary condition and may accompany psoriasis of the pinna. Psoriasis is a lifelong affliction that may flare up or regress spontaneously.

Treatment

Control the ear canal itchiness with systemic antihistamines and preclude water from the external ear canals. Scratching the ear canal with a foreign body (ie, a matchstick or a cotton bud) may excoriate the psoriatic ear canal skin and may introduce secondary infection. Regular cleansing and removal of the ear canal debris and application of a local non-fluorinated steroid cream may control and suppress the scaliness and itchiness.

Herpes zoster oticus

The causative agent is the chicken pox virus (varicella). This self limiting disease occurs as a second stage viremia in patients with incomplete immunity against the virus.

Unilateral vesicular eruption occurs on the pinna and around the external ear canal. These vesicles form crusts adn drop off at about 21 days.

Neuritic pain may be mild and transitory. In a minority of cases persistent devastating pain may last for months or even years. Accompanying facial nerve palsy (Ramsay Hunt syndrome) may call for additional eye care.

Treatment

Palliative treagtment is indicated for pain, fever and malaise during the onset of the syndrome. The vesicles on the pinna are best left alone. Warm compresses over the pinna may ease the pain and discomfort. If facial nerve palsy is present, eye care should be implemented to protect the cornea from dehydration and corneal ulceration with possible corneal scarring and loss of vision. Synthetic tears may suffice. A self adhesive eye pad adds considerably to patient comfort.

'Malignant' otitis externa

The word 'malignant' here refers to the severity and degree of otitis externa and not to a malignancy as in a tumour. This is a potentially fatal condition.

The responsible pathogens are *Pseudomonas* strains and the condition may prevail in older diabetics. The infection spreads rapidly along the cartilaginous and bony external ear canal via lymphatics and vascular channels to the temporal bone and sigmoid sinus.

Thrombosis of the sigmoid sinus and involvement of the cranial nerves may become apparent.

Meningitis and brain abscesses complicate the intracranial spread of the infection.

Treatment

Use massive intravenous antibiotics and sessions of hyperbaric oxygen therapy if available.

Fungal infections (otomycosis)

Mycotic otitis externa may be acute or chronic. There are various causes of otomycosis: a flare up of saprophytic otomycotic strains in an immunocompromised patient; or secondarily induced strains with a concomitant bacterial infection usually associated with the overuse and abuse of antibiotics/steroid ear drops. Symptoms may resemble acute bacterial otitis externa. In the acute phase the ear canal is swollen and extremely painful with an otorrhea. A swab culture may reveal mixed fungi and bacterial strains (ie, *Pseudomonas, Proteus* and *Staphylococcus aureus).* Fungal strains may include *Aspergillus* and *Candida* species.

Treatment

Treatment consists of analgesics, local and systemic antimicrobial therapy. Ear canal cleansing to remove all infected debris may have to be done under anaesthesia due to the extreme pain and swelling of the ear canal during the acute phase.

An expandable ear wick coaked in antibiotic/antifungal ear drops may control the infection and swelling in the ear canal. The wick should be changed daily. Systemic antibiotics will control the bacterial component of the mixed infection. During the acute phase water should be precluded from the ear canal. Systemic antimicrobial/antifungal therapy is seldom needed but may be necessary in the hyperacute phase or in immunocompromised or diabetic patients.

Furuncles and carbuncles

A furuncle may present as an extremely tender abscess, usually in the outer external ear canal, with or without pus draining from the abscess. Taking a culture may reveal predominantly *Staphylococcus aureus* bacteria. The abscess originates from a hair follicle. Local adenopathy may be present. Management is by systemic antibiotics and local topical antibiotics on an ear wick.

An ear canal carbuncle is an extension of a furuncle and originates from multiple infected hair follicles. Drainage under a short anaesthetic followed by systemic and topical antibiotics on an ear wick may speedily resolve the infection. Pain control is indicated.

Foreign bodies

It is not uncommon to have a patient present with a foreign body lodged in one or both ear canals. These foreign bodies may be solid (such as a stone, peanut or maize kernel) or soft (such as cotton wool, paper, cloth or foam rubber).

In the ear canal, natural wax may be impacted into the deeper medial end of the external ear canal or even onto the tympanic membrane itself and may act as a foreign body: quite common following an attempt to clean the ear canal with an improvised cotton bud. Wax forms and is secreted by the lateral third of the external ear canal; not in the medial end of the canal.

Moths, beetles or other insects are from time to time seen lodged in the ear canal or even onto the tympanic membrane, especially in campers. The insect may be alive and cause a thunderous and alarming racket to the patient. Usually the tympanic membrane is not injured by the insect and may simply appear slightly hyperaemic.

Patients presenting with self inserted foreign bodies are usually young children or the mentally retarded.

A patient with a foreign body in the ear may present with painful external ear canal irritation and swelling. Mild conductive loss may be present, althoguh sometimes unnoticed by the patient. Once the foreign body is removed the patient joyously announces his hearing improvement.

Impacted wax, sand or a small solid foreign body in the ear canal may only be conincidentally diagnosed during a routine examination.

Especially worth mentioning is a welding spark or a slag injury in the external ear canal which may cause an agonisingly painful linear burn. Only rarely is the tympanic membrane perforated. The perforation usually heals spontaneously and nothing more than an analgesic may be required. Sometimes a slag injury may necessitate a tympanotomy to remove the foreign body.

To retrieve the foreign body

- The prime requirement is an excellent light source, preferably a headlight that allows 'hands-free' work and offers a coaxial light beam for deeper light penetration into the external ear canal.

- For solid objects in the ear canal it is best to use a ring curette: raking it rather than pushing it deeper in with forceps. Pushing the foreign body deeped into the ear canal may injure or perforate the tympanic membrane.

- For soft foreign bodies (eg, paper and cotton wool) use either a curette or angled forceps (ie, bayonet forceps).

- Do not irrigate a vegetable foreign body in the ear canal (eg, a maize kernel or a peanut), it may cause further swelling compounding the problem.

- Impacted wax or a foreign body deeply imbedded at the isthmus of the external ear canal can be removed by syringing with a water jet.

- Insects, alive or dead, may be removed with small forceps. If the insect is still alive but no instruments are available, then a few drops of oil into the ear canal will suffocate the insect, silencing it until the retrieval can be managed.

- General anaesthesia may be necessary for frightened or previously hurt patients, especially children.

- Follow up systemic antibiotics may be required for a concomitant traumatised ear.

- Always look for multiple foreign bodies ipsilaterally or contralaterally, or in other possible sites, especially in a child or mental patients.

Iatrogenic afflictions

Large mastoid cavities resulting from severe life threatening mastoiditis in the pre-antibiotic era or from a hearing rehabilitation operation prior to the contemporary stapedectomy procedures (the fenestration operation) often accumulate wax and harbour low-grade infection with a smelly discharge. They may be difficult to keep clean and dry.

A short course of topical antibiotic ointment, or tamponading the cavities with framycetin sulphate can be very effective in clearing both the infection and smelly discharge.

Exostosis and osteomata

Both of these acquired conditions appear in the older patient. Males and females are equally affected.

Osteomata are usually unilateral solitary exophytic and pediculated protrusions. Exostosis may be bilateral, multiple, wide based hyperostotic protrusion/s into the external ear canal/s. Exostosis has a strong association with cold water swimmers. The narrowed ear canal may trap water, causing itchiness that the patient counteracts by scratching the ear canal with a foreign body. Osteomata may be a predisposing factor to recurrent otitis externa.

Osteomata are easily removed by chipping off the protrusion at its pedicle using a micro-osteotome. However, an exostosis may be quite difficult to remove due to the diffuse stenosis of the external ear canal and the consequent shortage of ear canal skin coverage after the exostosis has been removed. The ear canal circumference becomes enlarged and the existing skin may not cover the newly created bony external ear canal circumference. Spontaneous skin regeneration to cover the bare bony canal may take many weeks, even months. Ultimately, a skin graft may be necessary to cover the bare bony external ear canal and speed up healing.

Carcinoma

Carcinoma of the external ear canal is a rarity and is seldom diagnosed before the condition reaches an advanced stage. It affects predominantly older patients, males and females alike.

Symptoms

Initially the patient may present with chronic earache. A bloody, purulent discharge may be present as well as moderate conductive deafness. Ipsilateral facial nerve palsy may be present due to tumour invasion of the facial nerve (fallopian) canal.

Signs

Examination may reveal an obscured eardrum due to a fulminating exfoliative mass: a punch biopsy may confirm the diagnosis. The tumour aggressively infiltrates the local and adjacent soft tissue close to the external ear canal via Santorini's fissure and Huschke's foramen. Progressive invasion via nerve sheaths, lymphatic vessels or directly to the temporal bone promotes intracranial invasion.

CT scans may reveal bony destruction; an MRI study may reveal the extent of intracranial invasion to the temporal lobe.

Treatment

Although morbidity is high, for early lesions surgery (ie, partial temporal or total temporal bone resection) appears to be superior to radiotherapy. In advanced cases mortality is high. Combined therapy (ie, surgery and radiotherapy) may be palliative. Concomitant carotid artery resection due to invasion of the tumour may result in catastrophic intracranial functional sequelae. Chemotherapy should be reserved for recurrences and metastatic lesions. The overall morbidity and mortality prognosis of this lesion is poor.

1. The external ear canal is 2.5 cm in length. The vibrissae (ear canal hair) and cerumen (wax) glands only occur in the lateral third of the ear canal. Vibrissae point outwards and may play an important role in the aetiology of otitis externa. Vibrissae are a male sexual trait.

2. Cartilaginous or bony atresia of the external ear canal may be surgically corrected with good functional and hearing improvement. CT scans are mandatory to assess the precise course of the facial nerve because this nerve may be at risk if the fallopian canal follows an abnormal course. A preoperative audiogram may reveal the level of middle and inner ear hearing.

3. Cartilaginous or bony stenosis: Narrowing of the external ear canal may be cartilaginous or osseous. A narrowed ear canal may trap water, causing recurrent ear canal infections; surgical widening may be necessary if recurrent infections cannot be controlled.

4. Trauma - External ear canal. Introducing a foreign body into the ear canal to scratch an itching ear may result in an inadvertent linear tear of the external canal skin. The tympanic membrane may be perforated and the ossicles may be disrupted causing conductive deafness. If nystagmus and dizziness is present the stapes may have been disrupted causing an inner ear leakage of perilymph. This is an ENT emergency and should be treated under sterile conditions, under general anaesthesia using a surgical microscope for illumination and magnification.

5. Seborrhoeic otitis externa is a chronic inflammatory disorder of the external ear canal skin in the area of concentrated sebaceous glands. As with dandruff on the scalp, this condition sheds skin flakes. The treatment for seborrhoeic otitis externa and for psoriasis is similar.

6. Psoriasis of the pinna and external ear canal: Psoriasis is a chronic dermatosis and may cause severe itchiness. Scratching of the ear with a foreign body may introduce a secondary infection. Psoriasis of the ear canal should be treated in the context of systemic psoriasis; a short course of systemic antihistamines may subdue the itchiness. Regular steroids applied topically to the ear canal may keep the psoriasis at bay. Water must be precluded from the external ear canal at all times.

7. Herpes zoster oticus: The causative agent is the chicken pox virus (varicella). This self limiting disease occurs as a second stage viremia in patients with an incomplete immunity against the virus. Unilateral vesicular eruption occurs on the pinna and around the external ear canal. These vesicles form crusts and drop off at about 21 days.

8. 'Malignant' otitis externa: The external ear canal may be severely swollen; to the point of occlusion. This condition prevails in the older uncontrolled diabetic patient. It is extremely painful and warrants urgent attention. The infection spreads rapidly via vascular and lymphatic channels to the temporal bone and temporal lobe. Cranial nerve involvement may become apparent.

9. Acute otitis externa: The external ear canal may be severely swollen occluding the external ear canal. This is an extremely painful condition and warrants urgent attention. Otitis externa is cellulitis of the deeper soft tissues of the external ear canal, predominantly caused by Streptococcus and Staphylococcus strains. A hair is normally sealed close to the exit of the shaft. Under certain conditions superficial infections may spread along the shaft to the accessory apocrine and sebaceous glands giving rise to an external ear canal furuncle/carbuncle with surrounding cellulitis. Systemic and local topical antibiotics on an ear wick may speedily control the infection. Pain control is warranted.

10. A hair is normally sealed along the exit of the shaft. Under certain conditions superficial infections may spread along the shaft to the accessory apocrine and sebaceous glands giving rise to an external ear canal furuncle/carbuncle with surrounding cellulitis.

11. Foreign body: A hard foreign body in the ear canal may be easily retrieved. Use an angled wax curette rather than forceps as the latter may push the foreign body deeper into the ear canal and may damage the tympanic membrane.

12. Insects in the external ear canal: An insect may become trapped in the ear canal. Its scratching against the eardrum may cause a thunderous noise - usually without any trauma. Filling of the ear canal with an oily ear drop usually drowns the insect, allowing its easy removal later. Expert removal is recommended to avoid injury to the tympanic membrane.

13. Benign tumors of the external ear canal - Stenosis: Bony ear canal stenoses may be due to exostosis of the bony ear canal. The exostosis is a dense bony extrusion and is more common in cold water swimmers. The narrowed ear canal may trap water, epithelial debris and/or wax and may predispose to recurrent ear infections. Microsurgical removal may be warranted if the canal is severely narrowed. Total stenosis may be difficult to correct and may place the facial nerve at risk. Timeous microsurgical correction often yields excellent results.

14. Benign tumors of the external ear canal - Osteoma: An osteoma of the external ear canal may occlude the canal and trap wax and water causing recurrent otitis externa. If warranted, the osteoma should be microsurgically removed. CT scans are recommended to correctly assess the position of the facial nerve and avoid its injury. A preoperative audiogram may reveal the hearing status of the middle and inner ear.

15. Malignant lesions of the external ear canal. Chronic earache and a bloody purulent discharge accompanied by progressive facial palsy should draw attention to possible ear canal malignancy. A CT scan may reveal bony ear canal destruction. An MRI examination may reveal temporal lobe invasion and carotid artery involvement. A biopsy and exploration under anaesthesia is warranted. Squamous cell carcinoma of the external ear canal may insidiously infiltrate the ear canal and surrounding soft tissue, with early invasion of the parotid and metastases occurring in the neck lymph nodes. Chronic nagging pain with low grade infection and facial nerve palsy should draw attention to malignancy of the external ear canal. Early investigation and biopsy requires a high index of suspicion. CT and MRI scans may reveal local temporal bone destruction and carotid artery infiltration.

Middle Ear

Auditory ossicles

The middle ear cavity measures roughly 1 cm in height by 0.5 cm in width - a volume of approximately 2 cc.

The auditory ossicles develop within the middle ear cavity and reach adult dimensions as chondral elements at around six months of foetal age, after which ossification begins. There are three auditory ossicles spaced between the eardrum and inner ear. The malleus (hammer) is imbedded onto the tympanic membrane, the incus (anvil) is the middle connecting ossicle and the stapes (stirrup) hydraulically seals the inner ear. The three ossicles slightly amplify sound.

Congenital/hereditary abnormalities

Rarely, the middle ear space may not be present on one or both sides (ie, no air space has developed).

The ossicles may not have developed into three separate ossicles but may be fused into one mass with a resultant conductive hearing loss. More often, one or more ossicles may separately be fixed by small areas of calcification, for example:

- The malleus head may be fixed to the attic.

- The short process of the incus may be fixed to the facial ridge.

- The stirrup may be fixed to the oval window.

- The muscle to the stirrup (stapedius muscle) may be calcified.

- Hereditary middle ear disorders (ie, otosclerosis) may fixate the foot plate of the stirrup leading to a conductive hearing loss.

- Ossification and fixation of the ossicles may also be an acquired condition due to chronic recurrent ear infections (ie, middle ear tympanosclerosis).

The above ossifications may cause conductive hearing loss of various degrees. With the aid of MRI, CT scans and audiometric studies these ossification points may be identified prior to surgical correction.

Otosclerosis is a fixation by ossification of the stirrup foot plate to the surrounding oval window. It has a familial tendency and in a small percentage of cases may be bilateral. Pregnancy may speed up the calcification and fixation of the stapes ossicle causing a marked increase in hearing loss.

Treatment

The stapes is surgically removed and replaced by a synthetic prosthesis. The results are uniformly excellent and lasting.

Traumatic tympanic membrane perforations

Blast injuries

Blast injuries may be caused by acute barotrauma (ie, explosions, a flat hand blow onto the external ear canal, water skiing or even as benign a reason as a parent playing with his/her child). The compression wave generated in the external ear canal may tear the tympanic membrane. A linear antero-inferior or a hinged tear may result.

The pain is dull. A trickle of blood may escape from the external ear canal. The effect on hearingmay vary from temporary muffling to a permanent severe degree of inner ear hearing loss. Examination of the eardrum may not be possible due to an obscuring blood clot.

Treatment

The external ear canal should be sealed off with sterile cotton wool and left alone. No drops should be instilled. Under sterile surgical conditions the ear canal should be cleaned and the tympanic membrane evaluated. The hinged segment, if present, may be lifted into position and stabilised with a disc of compressed Gelfoam. Definitive surgery may be necessary to patch up larger eardrum perforations and to correct ossicular damage.

Skull base fractures

Skull base fractures involving the temporal bone (ie, a longitudinal temporal bone fracture) may transect the tympanic membrane resulting in a linear tear. Profuse haemorrhage and cerebrospinal fluid otorrhoea may discharge from the ear canal. The eardrum and middle ear are best evaluated under general anaesthesia unless this is precluded by the patient's condition.

Meanwhile, the ear canal should be sealed off with sterile cotton wool. No drops should be instilled. The patient should be given an antibiotic to prevent infection and meningitis.

Welding spark injury

A superheated flux spark flung into the external ear canal may result in a linear burn

The slag may penetrate the eardrum resulting in a 2-3 mm round perforation which may heal spontaneously within 14 days. No drops are indicated. Sterile cotton wool may preclude water and wind from entering the ear canal.

Trauma to the eardrum

Ear canal lacerations and/or tympanic membrane perforations may result from foreign bodies introduced into the external ear canal to counteract itching. Matchsticks, toothpicks and knitting needles are frequently culprits. Sharp objects tend to produce a linear rent of the ear canal and eardrum. Cotton bud injuries may result in a stellate tympanic membrane perforation.

On initial examination the ear canal may be obstructed by a blood clot. It is prudent not to disturb the blood clot or to instill ear drops. Seal the ear canal from dust with sterile cotton wool and evaluate the tympanic membrane in sterile conditions under general anaesthesia.

Once the blood clot is removed and the ear canal is cleaned the extent of the ear canal and tympanic membrane damage may be established. Minor ear canal tears and tympanic membrane perforations may heal spontaneously. If more than 25% of the eardrum surface has been perforated a definitive myringoplasty is indicated to close the eardrum perforation. Evaluation of the middle ear ossicles may be undertaken simultaneously. Analgesia and prophylactic infection control is mandatory for a successful result.

Preoperative and postoperative hearing tests (audiograms) should be undertaken to document the extent of hearing loss and the type and extent of the injury. Medicolegal claims may be in the offing!

Infection of the tympanic membrane: Myringitis bullosa

The tympanic membrane is a trilaminar membrane separating the external ear canal from the middle ear cavity

The membrane may be primarily infected by *Mycoplasma pneumonia* causing myringitis bullosa, an acute, extremely painful condition. The *Mycoplasma pneumonia* organism is an atypical pathogen and does not have a cell wall. It is not classified as a virus or a bacteria.

On inspection the eardrum appears hyperaemic with serosanguineous filled vesicles onthe outer eardrum surface. Rupture of these vesicles under a general anaesthesia may

immediately relieve the pain. Take care not to perforate the tympanic membrane with the myringotome or the infective organism may be implanted into the middle ear cavity.

Treatment

A tetracycline, a quinolone or macrolide antibiotic may speed up healing although it is a self limiting disease with a span of 72-96 hours. Pain control is indicated using systemic analgesics and analgesic eardrops.

Acute purulent otitis media

Acute otitis media without perforation is an extremely painful infection with pus accumulation in the middle ear cavity. Urgent therapy with antibiotics and analgesics may successfully control the vast majority of cases. The middle ear infections may be recurrent; however, children outgrow this tendency between five and nine years of age.

AOM may cause a small round central perforation of the eardrum that usually heals spontaneously once the infection is under control. Second-hand smoke is a risk factor for otitis media in children.

Chronic otitis media

Chronic otitis media may result in a persistent large tympanic membrane perforation. The location, more than the size of the perforation, may have an important bearing on the prognosis for the middle ear. A central perforation has a better prognosis than a peripheral one. The latter may result in an ingrowth of skin into the middle ear cavity, a condition that could initiate an acquired cholesteatoma. A cholesteatoma is a cyst-like lesion with osteolytic properties, that may expand and exert pressure on the middle ear ossicles causing partial destruction and/or dehiscency. Conductive hearing loss and a chronic smelly otorrhoea may result.

Treatment

Surgery is indicated to correct the chronic ear perforation. The concomitant infection should be controlled. However, long standing chronic infection may be deep seated and superficial suction of debris and local ear drops may not be sufficient to eradicate the infection. Hearing tests, X-ray studies and identification of the offending microorganisms are preoperative requirements.

Surgical exploration of the middle ear (and mastoid) may sufficiently expose the infection for adequate microsurgical mechanical removal of the infected granulated tissue. Postoperative pain and infection control may be given orally or sytemically.

Glue ear (OME)

Serous otitis media, otitis media with effusion

Transient hearing loss in a child who has developed normal speech is most likely due to serous otitis media. The most common age groups affected are those between two and seven years.

Serous otitis media is a painless non purulent accumulation of fluid in the middle ear. The fluid ranges from clear to straw coloured and the consistency from ordinary fluid to thick, sticky, tenacious glue (hence the term 'glue ear'). Most cases of glue ear will resolve spontaneously; however, it is extremely important not to misdiagnose the few persistent cases. The hearing impediment caused by persistent glue ear may have a vast effect on a child's speech and cognitive development, as well as on his or her education and, later, on ability to compete with peers.

School teachers and parents are the first to note a lack of progress, personality changes, hyperactivity and speech defects, such as saying "top treat" instead of "stop street". The soft, non-vocal consonants are dropped from speech because the sufferer does not hear them.

The cause of glue ear is not yet confirmed, but many theories have been postulated (eg, unresolved infection, which may be allergy related, combined with poor functioning of the Eustachian tube). Epidemiologically, the condition is more common among lower socio-economic groups and recently its incidence appears to be increasing.

Examination

Examination reveals a dull, retracted tympanic membrane. Tympanic membrane calcification (tympanosclerosis) may be present due to previous infection and grommet insertions. The drum is retracted and the short process of the malleus is prominent. A Siegle speculum examination reveals a lack of eardrum mobility. These findings are usually present on both sides. Adenoids may or may not have been removed.

Audiometry

Audiometry may be difficult to perform in young children, except for tympanometry. The latter may reveal a flat tympanogram due to increased tympanic membrane impedance (ie, resistance of movement of the tympanic membrane). This confirms the presence of middle ear fluid. Sinus X-rays may reveal the status of the adenoids in the nasopharynx.

Treatment

Medication does not seem to hasten resolution of glue ear.

- If the patient has received treatment for six weeks and the adenoids are present, grommet insertion and adenoidectomy is indicated.

- If the patient has had an adequate adenoidectomy, an empiric ten day course of antibiotics should be administered followed by re-examination (Siegle speculum testing and tympanometry). If there is still no improvement, grommet placement is indicated.

- If the patient presents with recurrent glue ear and grommets have been prematurely extruded, a long term grommmet with a larger medial flange should be inserted. Too large a grommet may cause a permanent perforation which may necessitate a myringoplasty at a later stage.

Middle ear ventilation tubes (grommets)

The first known contemporary attempts to synthetically ventilate the middle ear via the tympanic membrane took place during the Second World War when German Stuka pilots received a thermal myringotomy of the tympanic membrane.

Ventilation tubes (or grommets) were introduced during the 1950s and were subsequently commercialised to improve middle ear ventilation and deal with drainage problems.

Middle ear ventilation tubes are made from a variety of medical grade material such as silicone rubber, teflon, stainless steel, titanium and gold.

The choice of the grommet, the size, the shape and the flange diameter depends on the expected length of stay in the tympanic membrane. Grommets may remain *in situ* from a few months to years. The length of stay in the tympanic membrane is to a large extent determined by the size of the medial flange. The choice of material (eg, silicone, rigid teflon or other material) depends on the individual surgeon and the ease of insertion into the eardrum. The grommet is inserted via a myringotomy incision, preferably in the antero-inferior quadrant of the tympanic membrane.

Why ventilate?

Indications for draining and ventilating the middle ear space are:

- In the case of glue ear hearing is immediately improved.

- In the case of a negative middle ear pressure the collapsed (sucked in) tympanic membrane may be restored to the normal position for optimal sound vibration and conduction, thus restoring the eardrum compliance.

- Restoring middle ear pressure and drainage of middle ear fluid may help curb recurrent middle ear infections.

- Children with submucous or cleft palate have poor Eustachian tube function and may need ultra long-term middle ear ventilation using a grommet with an ultra large medial flange.

- After postnasal space trauma or chronic Eustachian tube dysfunction.

Grommets may be placed in the active purulent stage of middle ear infection.

Swimming, hair washing and showering are not contraindicated as long as water is precluded from the ear canal. Various hints that help parents include:

- Place a small ball of self adhesive putty in the external auditory meatus. It sticks comfortably to the skin and is, to some degree, water repellent.

- Use a small cotton wool ball soaked in petroleum jelly: the petroleum jelly is strongly water repellent.

- Custom made ear moulds can be obtained from a hearing aid dealer.

If adenoids are still present, they should be removed when the grommets are inserted.

There are some negative aspects to the use of grommets. In rare cases they may cause a persistent perforation of the tympanic membrane, especially when a long or ultra long duration ventilation tube is placed in the tympanic membrane. These ultra long tubes are larger than the regular type of ventilation tube with a larger bush diameter.

Grommets may also be extruded before the middle ear and Eustachian tube pathology has resolved and may necessitate revised placement. Parents should be forewarned of this possibility.

Grommet insertion is a palliative therapy and treats the symptoms rather than the direct cause. Continued attention should be directed at the cause of the middle ear and Eustachian tube dysfunction.

Though the pathophysiology of glue ear is not clearly understood, grommet placement is a great adjunctive treatment for a developing child and results in dramatic hearing restoration.

Patients with a cleft palate or a submucous cleft may require the insertion of an ultra long-term ventilation tube. In such patients Eustachian tube function is permanently reduced and the middle ear is permanently at negative pressure with subsequent serous otitis media and a prolapsed tympanic membrane.

Parents should be warned that grommet insertion might be necessary on more than one occasion. Grommets may be spontaneously extruded between four months and one year after insertion. They constitute a palliative treatment for glue ear, for which the aetiology is enigmatic.

Tympanosclerosis

Tympanosclerosis of the tympanic membrane is a benign acquired pathologic calcification of the *tunica media* of the *pars tensa* of the eardrum. The plaques are the end-stage healing results of repeated attacks of acute otitis media and are rarely of audiologic or clinical significance. Hearing loss only occurs if a major portion of the tympanic membrane

is involved. Tympanosclerosis may also extend to the middle ear mucosa and ossicles with fixation of the ossicles and subsequent conductive hearing loss.

Tumours

Osteoma

Osteoma may occur not only in the external ear canal but also in the middle ear, and as they expand they may impinge on the ossicles in the middle ear cavity and cause progressive conductive hearing loss.

The dense osseous growth is well diagnosed by CT scans. Surgical removal is elective with good hearing results.

Facial nerve neurofibroma

Classically the growth occurs in the internal ear canal but may occur in the middle ear originating from the facial nerve. Surgical resection of the tumour with an end-to-end anastomoses to the facial nerve may restore facial movements.

Glomus tumours

Symptoms and signs

Conductive hearing loss may be present. Patients may complain of a distinct, pulsatile, low frequency tinnitus synchronous to their heartbeats. This is a pathognomonic symptom of a glomus tumour.

Otoscopy may reveal a distinct blue purple mass behind the eardrum. It is difficult to distinguish between a *glomus tympanicum* arising from the floor of the middle ear and a *glomus jugulare* tumour eroding into the middle ear cavity: conditions that require vastly different surgical approaches.

Hearing tests should be performed to document diagnosis and prognosis.

CT scans of the petrous bone may reveal the origins of the glomus tumour because a *glomus jugulare* tumour should, in the skull base view, show a distinct widening of the jugular bulb.

Glomus tympanicum tumours of the middle ear cavity usually arise from the floor into the middle ear cavity. These tumours are slow growing, soft expanding masses with local ossicles and bony destruction due to pressure exerted on the surrounding structures.

Acquired cholesteatoma

Although an acquired cholesteatoma is not a tumour, it may act locally as one, insidiously invading the middle ear ossicles and the bony confinements of the middle ear.

An acquired cholesteatoma may be clinically diagnosed by otoscopic or microscopic otoscopy of the tympanic membrane.

The condition originates from an attic or a marginal eardrum perforation. A purulent, sometimes smelly discharge may be present in the external ear canal. The size and site of the perforation may not reveal the true extent and degree of pathology in the middle ear and special studies are required to qualify and quantify this.

Hearing tests

If the cholesteatoma is confined to the middle ear, a 20-30 dB conductive hearing loss may indicate ossicular damage. If the cholesteatoma has invaded the inner ear, then an additional sensorineural hearing loss may be present. The patient may also complain of vestibular symptoms (ie, vertigo or a tendency to fall). These symptoms support the possibility of inner ear invasion.

CT scans may timeously confirm the extent of middle ear ossicular damage and erosion of the bony middle ear confinements.

The incus is vulnerable to cholesteatoma erosion resulting in a bony dehiscency and conductive hearing loss. The bony walls of the middle ear may also be affected by the osteolytic properties of the cholesteatoma. The thin roof of the middle ear cavity *(tegmen tympani)* may be eroded, giving way to invasion of the middle cranial fossa with possible abscess formation.

Determining the extent of cholesteatoma invasion by special studies may dictate the correct surgical approach. With the aid of hearing tests, CT scans and the use of a surgical microscope, cholesteatoma may be successfully diagnosed and timeously treated and eradicated. Follow up control studies to exclude insidious recurrence are prudent.

The behavious pattern of cholesteatoma should be explained to the patient. It is not a malignancy but may act so locally, being insidiously invasive. Although it may recur, it is a curable disease.

Referred ear pain

Temporo-mandibular (TM) joint dysfunction

TM joint dysfunction is an umbrella term encompassing two groups of patients: those with primary joint disorders and those with myofascial pain dysfunction.

TM joint dysfunction may often be misdiagnosed as an ear pathology (eg, otitis media). Many patients are treated with antibiotics for a unilateral ear infection without experiencing any improvements. Examination of the tympanic membrane shows it to be perfectly normal! In reviewing the initial diagnosis, one should start with a careful history which may identify the referred ear pain as coming from TM joint dysfunction, dental extractions, recent tooth fillings, new dentures, or a blow to the jaw (sport injuries, motor vehicle accident or assault). All these conditions may lead to chewing on one side only and

this may result in TM joint dysfunction. In some neglected cases trismus may be present.

Tension and stress may lead to tooth grinding during sleep (bruxism), which may further overload the affected TM joint and compound the issue. Young females are affected far more often than males (4:1).

Although referred otalgia is usually from dental or TM joint origin, keep in mind the malingering scholar and be aware also of the possibility of malignancy in the larynx or pharynx in the older male.

Symptoms

The patient usually describes the pain as being recent, or sometimes long standing, unilateral discomfort over the TM joint and ear canal area. The patient may be confused as to the precise site of maximum tenderness and may only describe the pain as deep dull ear pain. Pain may be referred to the ipsilateral temporal area, the masseter muscle, over the maxillary sinus, and over the anterolateral aspect of the neck.

Examination

Feel both sides of the TM joint with the index finger/s or press on the area of joint tenderness when opening or closing the jaw. Jaw crepitus may be apparent on movement, especially in the older patient with actual joint pathology (eg, rheumatoid or osteoarthritis). Jaw clicking may have little bearing on TM joint dysfunction. Palpation of the masseter muscle may elicit tenderness of the muscle.

Examination of the mouth may reveal multiple filled teeth, an absent molar, dental caries or recently acquired dentures: any of these may contribute to TM joint dysfunction.

Intra-articular administration of 0.5 mL two percent xylocaine will immediately relieve the pain and discomfort, confirming the diagnosis.

Plain X-rays of the TM joint are usually disappointingly negative. MRI scans are indicated, but should be interpreted by a radiologist skilled in this field to evaluate the ligaments and joint cartilages.

Treatment

A local heat pad over the TM joint and masseter muscle will ease the discomfort. Systemic anti-inflammatory agents (ie, NSAIDs, steroids and muscle relaxants like diazepam), will sooth the TM joint discomfort during an acute exacerbation. Ultrasound and physiotherapy may have a longer lasting benefit.

A tailored bite plate worn during sleep may alleviate the masticatory muscle spasm and dental grinding, especially during periods of stress, such as exam times. Muscle relaxants at night may prevent clenching and grinding. Psychological counselling, stress avoidance and a soft diet may also assist.

The correction of recalcitrant TM joint dysfunction lies with the maxillofacial surgeon, joint surgery being a last resort for the few failures of conservative therapy.

Diagnosis of the TM joint dysfunction syndrome requires an index of suspicion that TM joint pathology may be present in that area.

Barotrauma

Barotrauma often occurs with rapid barometric change (ie, when diving or flying).

Divers are exposed to harsh barometric changes, as are individuals in non-pressurised aeroplanes. More subtle changes occur in partially pressurised commercial aeroplanes. The most pronounced effects are on the middle ear, nose, sinuses and the upper molars. In divers, the face, specifically the portion covered by the mask, may also be affected.

Aggravating and predisposing factors are swollen nasal mucosa, as occur with an upper respiratory infection, allergy, vasomotor rhinitis, polyps or a deviated nasal septum. The pathophysiology of barotrauma is based on the fact that as barometric pressure increases, air volumes decreases (Boyle's law), hence gas containing cavities (eg, sinuses, ears, and in rare instances the teeth) may be affected. The resultant reduced gas volume causes soft tissue distortion (ie, vascular engorgement), extravasation of fluid into the adjacent soft tissue, petechiae, and ultimately haemorrhage. Pain is induced because of the stretching of the tympanic membrane and distortion of the mucosal lining of the nose and sinuses. Serous or blood-stained accumulation may occur in the middle ear and sinus cavities to compensate for the loss of gas volume.

The vast majority of dental pain is referred pain from the maxillary sinuses to the upper teeth. True barodentalgia is rare and occurs when air is trapped underneath a filling. It occurs during ascent and the tooth may remain uncomfortable throughout the flight until descent commences.

Symptom and signs

The air passenger or diver may experience acute and severe facial pain, earache and dental pain (barodentalgia), especially of the upper premolars and molars. The patient may also experience a moderate degree of deafness due to haemotympanum. Acute barovertigo may also occur due to rupture of the oval or round window membranes. Less severe and shorter lasting barovertigo may bedue to unequal Eustachian tube function resulting in unequal middle ear pressures.

Epistaxis is a hallmark of barotrauma. Bleeding from the ear canals indicates a tympanic membrane perforation, and moderate conductive loss may be present. In divers, facial nerve palsy or palsy of any other cranial nerve may be present.

Treatment for air passengers

- Air passengers in commercial airliners are not exposed to drastic barometric changes. Antihistamines or decongestants may be of value to prevent baro-otitis and barosinusitis.

Analgesics may relieve the initial pain of barotrauma.

- For adults, the Valsalva manoeuvre (ie, popping the ears while closing the nose with the forefingers) should be encouraged during descent. Infants should be held upright and breast-fed or bottle-fed to encourage swallowing, thus simulating the Valsalva manoeuvre.

- Tympanic membrane perforations are usually small and heal spontaneously. Ear drops or deep ear canal cleansing is not necessary. Preclude water from the ear canal until final healing is confirmed.

- For pilots with recurrent acute baro-otitis, a grommet insertion may provide welcome relief.

- For recurrent attacks of barosinusitis, functional nasal and sinus surgery may relieve the condition. Recurrent barodentalgia secondary to barosinusitis (the vast majority of cases) may necessitate no treatment other than nasal decongestants (ie, nose drops) and systemic decongestants prior to the flight. Many times a tooth has been unnecessarily extracted only to discover that the adjacent tooth was the apparent villain, and even the extraction of twoor three further teeth yields no improvement.

- Prophylactic antibiotics are desirable in selected cases.

Treatment for divers

- Divers are exposed to harsh barometric changes. Those with upper respiratory infection should not dive: decongestants and antihistamines may not prevent barometric complications (eg, barosinusitis or barotitis). Divers should be strongly discouraged from smoking.

- The state of the nose is of paramount importance to the diver for safe and enjoyable scuba diving. Any cause of congestion should be corrected as a long term investment for the diver, whether professional or amateur. Causes of nasal congestion, such as nasal allergy, deviated nasal septum, nasal polyps, and the common cold may all be successfully treated with medication or surgical management. Alleviation of nasal congestion will enhance nasal breathing and may even improve Eustachian tube function to equalise pressure differentials in the middle ear cavity.

- If barotrauma has occurred, rest between barotraumatic episodes for 14 days is advised. Episode after episode may occur due to insufficient recovery from the barotrauma, predisposing to and aggravating subsequent barotrauma. For divers with barotrauma, the most sensitive prognostic test is pure tone audiometry together with an impedance audiogram.

- Barotrauma should not be confused with decompression sickness where divers are exposed to too rapid decompression resulting in air bubble formation in the blood and tissues.

- In souther Africa, the decompression chamber unit at the RSA navy base at Simonstown offers 24-hour assistance for barotrauma and decompression incidents. Tel: (021) 787-3911.

1. The three middle ear ossicles are full size at birth. They are the only bones in the human body that do not grow after birth. The ear canal hairs point 'outwards' and only occur in the lateral third of the ear canal. The ear canal hair is a sexual trait and only occurs in men.

2.1. Middle ear conductive deafness - Malleus fixation: The head of the malleus may be fixed by calcification to the adjoining bony middle ear cavity (ie, the attic). This type of conductive deafness is amenable to surgical correction with good and lasting results.

2.2. Middle ear conductive deafness - Incus fixation: The short handle of the incus may be fixed to the facial recess in the middle ear by calcification. The resultant conductive hearing loss is surgically correctable with good and lasting results.

2.3. Otosclerosis: The stapes fixation is due to calcification and may cause conductive hearing loss especially noticeable in the lower frequencies. This condition is surgically correctable with excellent and permanent results.

2.4. Stapedectomy: A stapedectomy is a surgical procedure in which the conductive deafness that follows stapes fixation by calcification (otosclerosis) is corrected. A stapes prosthesis that connects the long handle of the incus to the inner ear is corrected via a minuscule hole drilled into the foot plate of the stapes (stapedotomy) using a surgical laser. This arrangement bypasses the fixed stapes and transmits sound to the inner ear. This ingenious procedure was invented and perfected by Dr John J. Shea (Snr) from Memphis, Tennessee, USA in the early 1950s. The results are excellent and lasting.

3. Blast injuries to the middle ear. The compression wave from the blast injury may rupture the eardrum and may dislocate the middle ear ossicles. The incus is most often dislocated. The resultant conductive hearing loss may be surgically corrected with good and lasting results. Inner ear damage from the blast may also result in inner ear deafness and permanent tinnitus. The inner ear damage may be moderate to profound; hearing loss may be temporary or permanent.

4. Skull base fractures may be classified as longitudinal fractures or transverse fractures. Most commonly a combination of fractures occurs. Blood from the external ear canal may draw attention to a temporal bone fracture. Violent vertigo and facial nerve palsy may be present, indicating that the fracture traverses the fallopian canal and the inner ear capsule.

5. Welding spark injury. A superheated flux spark may be flung into the external ear canal. A linear burn of the ear canal may occur and, depending on the trajectory of the spark, a 2-3 mm round perforation of the eardrum may result.

6. Traumatic tympanic membrane perforations. The eardrum may be perforated by the inadvertent sudden movement of a foreign body introduced into the ear canal to counteract itching. Most perforations heal spontaneously. The eardrum perforation should be monitored in case surgical closure of the perforation is necessary. A traumatic eardrum perforation may also include an ossicular dislocation, causing conductive hearing loss. The incus is the most vulnerable to traumatic dislocation. If violent vertigo is present, stapes dislocation with a

fistula formation must urgently be excluded. The perforation as well as the conductive hearing loss may be surgically corrected with good and lasting results.

7. Myringitis bullosa. This is an acutely painful condition of the tympanic membrane. The causative organism is *Mycoplasma pneumoniae*. This condition may resolve spontaneously within 96 hours. Rupture of the small bullae under anaesthesia brings immediate relief with a macrolide as the drug of choice for follow up antibiotic treatment.

8. Acute otitis media. The acute pain is due to the stretching of the tympanic membrane. After the eardrum has ruptured the pain subsides due to pressure relief in the middle ear cavity. A small perforation may heal spontaneously within 96 hours.

9. Chronic otitis media with ossicular disruption. Inadequate treatment of an acute ear infection may result in a persistent otorrhoea and a large eardrum perforation. Longstanding chronic middle ear infection may also erode the ossicles. The incus is the most vulnerable and may result in conductive hearing loss. This condition may be ameliorated with microsurgery.

10. Glue ear. The inspissated mucoid middle ear fluid may vary in colour from clear to straw coloured and in consistency from fluid to sticky tenacious 'glue'.

11. Ventilation tubes. Grommets for various middle ear applications are available in many patterns and materials. The reduction in middle ear complications since their availability has significantly lessened the need for middle ear surgery.

12. Tympanoscleroses are irregular whitish calcified plaques on the eardrum: a possible sign of previous middle ear infection. They are of no clinical significance.

13. Middle ear tumours - Glomus tympanicum. A glomus tympanicum tumour is a vascular tumour of the middle ear cavity. On otoscopy it can be seen as a blue tinge through the tympanic membrane. The patient may experience a pulsating tinnitus corresponding to his own heartbeat. The tumour may be successfully removed with microsurgery.

14. Middle ear tumours - Glomus jugulare. This is a benign vascular tumour arising from the jugular bulb at the skull base. Considerable bony absorption and destruction may have taken place before the diagnosis is established. Surgical removal may be successful, but at the price of damage to surrounding structures and nerves.

15. Acquired cholesteatoma of the middle ear. Skin ingrowth into the middle ear cavity via a perforation may form a sac in the middle ear cavity. The contents of the sac (ie, infected degraded skin debris) may cause a smelly otorrhoea. The bacteriology may reveal anaerobic infection resistant to many antibiotics. Timeous microsurgery is indicated to ameliorate this advanced form of ear infection before complications occur (ie, temoral lobe brain abscess, disruption of ossicles, sigmoid sinus thromboses), as well as to restore hearing.

16. Temporo-mandibular (TM) joint dysfunction. This condition is often misdiagnosed and patients are treated with antibiotics without improvement. The TM joint

may be tender on palpation during opening and closing of the jaw. The pain may be referred to the ear, temporal region, sinuses or down the neck. Excellent results may be achieved with therapeutic medical laser treatment.

17. Barotrauma may cause a negative pressure in the middle ear space resulting in a collapsed tympanic membrane. Middle ear fluid may accumulate. Ascending infection from the nasopharynx via the Eustachian tube may infect the accumulated fluid in the middle ear resulting in an acute otitis media.

18. Barostressors. Longer intercontinental flights may induce stressor symptoms in flight attendants. In certain individuals low grade hypoxia may induce fainting, headaches, fatigue, stress and nose bleeds.

Inner Ear

Congenital inner ear deafness

During the first trimester of pregnancy the inner ear of the foetus is particularly vulnerable to maternal viral infections (viral labyrinthitis) and also congenital toxoplasmosis. German measles (Rubella virus) is a notorious offender and usually only affects one labyrinth. The subsequent hearing loss may be partial, profound or total. Labyrinthine haemorrhage and anoxia during delivery, especially in premature delivery, may lead to inner ear damage and subsequent hearing loss.

It may be difficult to diagnose deafness in infants. Profound deafness may be more easily diagnosed than mild deafness. Concomitant mental retardation may complicate the issue of early diagnosis. Deaf children require special education.

Early diagnosis of deafness in infants is of paramount importance. A school for the deaf should be approached to tailor an education programme for the patient as soon as the diagnosis is confirmed. Hearing amplification is indicated from the time of diagnosis to stimulate the development of auditory and brain stem pathways.

Acute spontaneous inner ear disorders

Sudden hearing loss and acute vertigo

There are two types of acute spontaneous inner ear disorders: sudden hearing loss and acute vertigo (vestibular neuronitis).

Sudden hearing loss, which usually affects one ear, is an otological emergency that may affect patients of any age, including those who may be otherwise healthy. The hearing loss may be partial or, less often, profound. Vertigo may follow within seconds or minutes of the hearing loss. At times there is an associated tinnitus that may be very distressing.

Aetiology

In most cases of sudden unilateral hearing loss the cause is uncertain and thought to be viral. Inner ear membrane ruptures are uncommon. Rare causes include otosyphilis, Ménière's disease and tumours impinging on the hearing nerve (ie, neuronoma). In divers, a common cause is decompression sickness (ie, ascending too rapidly resulting in gas bubble formation in the inner ear).

Special examination

A hearing test to establish residual hearing may determine the site of the lesion and quantify the hearing loss for documentation. X-ray studies and MRI scans may rule out an intracranial tumour or multiple sclerosis. Serological tests for syphilis, diabetes and other endocrine disorders may be valuable.

Prognosis

The older the patient, the worse the initial hearing loss and vertigo. Younger patients with moderate hearing loss usually regain their hearing within a fortnight following treatment. The earlier the treatment, the better the results. Further improvement may occur over ensuing months.

Treatment

- Hospitalisation should be considered. Mildly sedate the patient to ameliorate panic and alarm, then administer a tapering dosage of corticosteroids over six weeks.

- Lower blood viscosity with cardiovascular aspirin.

- Carbogen inhalation (five percent CO_2 / 95 ercent O_2) should be given for 15 minutes, three times a day for three days to increase intracranial blood flow.

- Request the patient to avoid straining movements (eg, on defectation), and keep stools soft. Prevent constipation and reduce coughing and sneezing.

- Institute hyperbaric oxygen treatment combined with stellate ganglion block if available. This appears to have excellent results. A 90-minute hyperbaric session each day for 14 days will hypersaturate the inner ear blood with oxygen. Stellate ganglion block is performed on the ipsilateral side tostimulate vasodilation of the inner ear.

- Patients in whom there is a strong suspicion of inner ear membrane rupture may benefit from surgical management to close the fistula. These patients usually experience a 'popping' sensation followed by a sudden hearing loss during strenuous movements (eg, weightlifting, coughing or defecation). They may have a positive fistula test or vertigo, but clinical findings may be non-specific. A fistula test is considered positive when digital pressure applied to the external ear canal results in vertigo and nystagmus.

- Divers with decompression sickness should be treated urgently in a hyperbaric chamber when available. Emergency airlift should be considered: the longer the time lag before treatment is initiated, the poorer the result.

Perilymph fistula

Perilymph fistula is a traumatically induced microscopic tear of the membranous inner ear that may be accompanied by perilymph fluid leaking into the middle ear. The most common site of rupture is from the oval or round window. This may cause acute vertigo, hearing loss, nausea and vomiting.

The most common cause is head trauma due to a motor vehicle accident or sport injury. Scuba diving, aerobatic barotrauma, weight lifting or straining during childbirth may also be responsible.

Rarely a fistula may be congenital or may appear spontaneously. The fistula is usually present in one ear only, with the inner ear damage being partial, temporary or permanent.

Diagnosis can be established from clinical history and physical examination. Moderate to profound inner ear hearing loss may be present on the audiogram and the patient may also have tinnitus. Spontaneous nystagmus is pathognomonic of inner ear damage. A careful clinical history may exclude other conditions, which may mimic a perilymph fistula (eg, substance abuse, cardiovascular disease, allergies and neurological disorders). Bacterial labyrinthitis is a direct extension of an acute purulent otitis media.

Treatment

Therapy consists of bed rest and symptomatic treatment for nausea and vomiting. If the symptoms become worse or if there is no improvement within 72 hours, surgical exploration is indicated to locate and close the fistula with a graft.

Onmce the fistula is healed, care should be exercised to prevent sudden barometric changes (ie, flying in small planes, scuba diving, straining or weight lifting).

Benign paroxysmal positional vertigo (BPPV) (cupulolithiasis)

BPPV is anything but benign; it may be disabling and may have a dramatic impact on lifestyle. There may be a strong correlation with previous head trauma or viral infections. The patient experiences brief, transient periods of vertigo and nystagmus in certain head positions in relation to gravity. A dislodged otoconia may be displaced out of the utricle and may land on an adjacent cupula of a semicircular canal.

An acute spinning vertigo may be triggered when the head is in an unusual position (eg, when instilling eye drops). The patient may inadvertently trigger an attack during sleep when rolling over on a pillow and may wake up with a violent attack of vertigo. The intensity of the attack typically diminishes over a few minutes. Attempts to repeat the response may result in a minor reaction or none at all. BPPV is usually a self limiting disease lasting for a few weeks to several months.

Hearing tests, X-ray studies and caloric tests are all typically normal. A positive Dix-Hallpike test is pathognomonic of cupulolithiasis. Similar diagnostic manoeuvres may precipitate an attack.

Treatment

In the majority of cases, lying uninterruptedly on the side of the affected ear for 12 hours may move the dislodged otolith out of reach of the affected semicircular canal and resolve the vertigo.

Motion sickness syndrome

Motion sickness was introduced as mankind developed its technical abilities to travel. It is a normal self inflicted response to an unfamiliar motion stimulus such as air, land, sea, or space travel, or even fun rides such as swings, 'dive bombs' or figure-of-eight rides.

The early signs of motion sickness syndrome are a hollow feeling in the stomach, facial pallor, and a feeling of warmth. This can escalate to sweating, nausea and vomiting.

Prolonged vomiting may cause electrolyte imbalance, dehydration, physical exhaustion and collapse. Children are more prone to motion sickness than adults, and girls more than boys. Women are especially prone to motion sickness during pregnancy. This may be the reason why few female commercial pilots and sea captains continue working during pregnancy. There is a strong mind-over-matter factor in motion sickness and the condition is aggravated by anxiety and fear.

Motion sickness syndrome may be provoked in susceptible individuals without actual body motion by the so-called occulogyral phenomenon: a neural mismatch between visual and vestibular information such as one can experience in an aircraft simulator or a cinematic simulator.

The severity of motion sickness syndrome is in proportion to the severity of the motion sickness stimulus (ie, the motion of a small boat on a relatively rough sea compared to a large vessel on a calm sea, or a small aeroplane in turbulent weather in comparison to a large commercial airliner at high altitude). A stron psychological overlay and individual susceptibility to motion sickness may ultimately determine the severity of the motion sickness syndrome.

Hints for suscetible patients

Although motion sickness is a self inflicted avoidable illness, not all people are equally susceptible at all times. Great relief may be obtained from an otherwise miserable condition. Below are some hints that may be of assistance to susceptible patients.

Air passengers and pilots

- The passenger should fix his eyes on the horizon during take-off to minimise conflict between the visual and the vestibular system. This is especially applicable in high

performance aeroplanes.

- Sit upright with the head stabilised. Do not rotate or move the head forward or backwards (eg, when taking off shoes) during take off or when the airfract is turning in flight or the Coriolis effect may be provoked. The Coriolis effect is a vector force resulting from two individual movements perpendicular to each other (ie, moving one's head from side to side during a turn in flight). It may be deadly if sustained by a susceptible pilot at a critical phase of flight prior to landing, when air speed is still relatively high, altitude is reducing rapidly and the pilot is busy with calculations and communications.

- Susceptible passengers should request a seat halfway down the plane and next to a window in order to fix their sight on the horizon.

- It helps to keep occupied: even experienced passengers may become airsick.

- Motion sickness drugs are rarely indicated for travel on large commercial aircraft but may be appropriate for travel in a small aircraft.

Seagoing fishermen and passengers

- Occupy the mid-centre of the boat close to the centre of gravity where the least motion sickness is provoked. If possible, visually fix on a set point (eg, the shore). Abstain from alcohol or large meals prior to a trip.

Anti motion-sickness drugs

- A hyoscine patch behind the ear greatly relieves motion sickness syndrome with minimal drowsiness. The transdermal patch should be applied an hour prior to the trip. Children over ten years may use the TTS patch but older males with impaired urine flow should be warned against potential urinary obstruction when using it. After handling the patch, wash hands and avoid rubbing the eyes: double vision and dilated pupils may result, lasting some hours.

- Cyclizine (tablet or syrup) may be used as an alternative without the cholinergic properties of hyoscine. It has a low sedative effect and the syrup is safe for children. Warn patients of the interaction with alcohol.

- Combine ephedrine with the above drugs. Ephedrine also combats any drowsiness and is available in tablet form or as syrup for children.

- Cinnarizine is also a useful labyrinthine sedative.

- For lasting situations, such as professional aircrews or sailors, habituation exercises as a long term solution may be useful.

Presbycusis

Presbycusis is derived from the Greek word meaning 'old-people's hearing'. It starts in the teens in the high frequency range (ie, 8000-12000 Hz), but with ageing the hearing loss gradually moves to the lower frequency range until in the 50s and 60s it may affect the 4000-8000 Hz range.

Some octogenarians are severely handicapped while others may only experience a mild hearing loss. Males are more severely and more frequently affected than females, perhaps because males are more exposed to noise.

The aetiology is deterioration of the organ of Corti, due to ageing of the inner ear. There is no positive correlation between diabetes and presbycusis. Chronic noise exposure and a genetic predisposition may enhance the early onset of the condition.

Patient hits for improved communication

- The role of lip reading and making maximal use of non-auditory clues may aid in speech communication.

- Avoid a two person conversation in a 'cocktail party' surrounding or in a moving vehicle. The soft high frequency fricatives and plosive sounds may not be heard under these conditions (eg, "stop street" may be heard as "to ree". Misinterpretation and wrong answers may lead to depression and social isolation.

- Contemporary hearing aids may be set to the frequency spectrum of individuals' hearing loss to compensate for the hearing deficit.

Note:

The human acoustic nerve contains in the order of 70.000 fibres. A dolphin has about 300.000 fibres. This implies that the dolphin's hearing is eminently superior and sophisticated when compared to the human hearing.

Ménière's disease: Endolymphatic hydrops

Ménière's disease was described by Prosper Ménière in 1861 as a disabling disease of the inner ear. In most cases the disease is confined to one ear.

Ménière's disease is characterised by four manifestatins: fluctuating hearing loss with a typical early audiological pattern; ipsilateral high tone tinnitus; attacks of spinning or rotational vertigo; and a distinct feeling of fullness in the affected ear.

Symptoms

Recurrent incapacitating attacks may occur within seconds of each other and without warning or even during sleep. The attacks may last from a few hours to a few days and may occur in clusters (ie, within days) or may be separated by several weeks, months or years.

Between attacks, the patient is free of symptoms except for a mild unsteadiness and tinnitus. The emotional impact may be significantly disabling.

Over the years, the patient's hearing may progressively worsen while recruitment makes social activities unbearable. Recruitment is an auditory phenomenon indicative of cochlear pathology where, although the patient has a hearing loss, he is abnormally sensitive to loud sound, causing discomfort.

Other conditions (eg, a neurofibroma of the internal ear canal) may mimic Ménière's disease and must be excluded.

Special investigations to confirm the diagnosis of Ménière's disease include caloric testing of the balance organs to reveal an uneven response to stimulation of the vestibular labyrinths. This testing may also indicate the affected side.

Hearing studies may confirm the affected side and may quantify the hearing loss. X-rays may exclude underlying pathology that may mimic Ménière's disease (eg, a growth compressing on the auditory or vestibular nerve).

Medical treatment

During an acute attack the patient should lie supine and fix his eyes on one position to suppress the dizziness. Medication to suppress the vasovagal symptoms or labyrinthine supressors may be administered orally or intravenously to combat the nausea and vomiting.

Chronic treatment

Long term prophylactic therapy includes definitive dietary modification with low salt intake. Diuretics may be added to reduce the endolymphatic fluid retention of the membranous inner ear. Alcohol and caffeine-containing beverages may also exacerbate the symptoms. Avoid smoking: nicotine is a vasoconstrictor and may delay fluid absorption due to vasoconstriction in the inner ear arterioles.

Intratympanic gentamycin therapy is being evaluated as a vestibular destructive modality in severe cases. Concurrent vestibular rehabilitation is an additional form of treatment that consists of daily positional head exercises for six weeks to promote central nervous system compensation and habituation for the inner ear deficits.

Surgical treatment

Unilateral vestibular neurectomy or labyrinthectomy is reserved for the recalcitrant cases that do not respond to any other medical therapy for vertigo control. The results are doubtful if the hearing is destroyed by long-standing bilateral disease. A cochlear implant may be of value to restore hearing.

Ototoxic drugs in hearing loss

Some ototoxic drugs may cause partial or permanent damage to the inner ear resulting in tinnitus and high frequency hearing loss. In the worst cases total deafness may result.

Vestibular damage may result in vertigo, balance disorders and nystagmus. Walking in the dark may be possible only with a wide based unstable gait.

Well documented agents are the aminoglycoside antibiotics, oncologic drugs, some diuretics, anti-malarials and environmental chemicals.

Salicylate hearing loss is 100% reversible in contrast to quinine and its synthetic substitutes which may produce permanent hearing loss. Most adult cochlear implant patients are victims of ototoxicity.

Most of these drugs are excreted by the kidneys. Impaired kidney function may predispose to drug ototoxicity due to increased serum levels. Early symptoms may be a high pitched continuous tinnitus, vertigo or a tendency to fall.

If possible a hearing test should be performed before ototoxic drug therapy is commenced. Hearing should be monitored daily. High frequency hearing loss and tinnitus or vertigo may be early warning signals. In the case of many drugs vestibular symptoms precede cochlear damage.

These drugs should be avoided during pregnancy for fear of inner ear damage to the foetus.

Noise induced hearing loss (NIHL)

Any sound level over 80 dB may cause inner ear hearing loss. Two factors are of paramount importance: the duration of exposure and the intensity of sound.

Close up intense sounds lasting microseconds may cause severe permanent hearing loss (eg, fire crackers, gunshots, explosions and home workshop equipment.

Longer exposure to high intensity sounds (eg, industrial noise, music, vacuum cleaners, sirens, vehicle horns, lawnmowers and electrical kitchen appliances) may also cause noise damage.

Younger children are more prone to noise induced inner ear damage than older patients. Damage may even start during pregnancy if the pregnant mother is exposed to loud noises. Typical early audiometric findings are an island of hearing loss between 4000 Hz and 6000 Hz, but this distinction is lost with prolonged exposure.

The symptoms of inner ear damage may be high pitched continuous tinnitus with muffled hearing. These symptoms may be transient and may clear after a few weeks.

Whatever the source of the damaging noise, two basic solutions exist: protection and avoidance. Prevent excessive noise exposure during a noise induced recovery phase - damage on damage may accrue and exacerbate the noise induced hearing loss. Parents play a vital role in alerting children to possible offenders (ie, cap guns, loud radios, rattles, walkie-talkies, earphones, horns and noisy toys). These offenders should be removed or silenced.

Patients exposed to excessive noise as an occupational hazard should protect themselves against sound damage with the necessary protection devices (ie, earplugs in the ear canals and/or external ear muffs). Pain may be induced by noise levels exceeding 80 dB and should be avoided or attenuated. Earplugs attenuate sound by 25-30 dB and external sound ear mufflers may offer further protection.

With noise induced hearing loss the ear more directly exposed to the sound source is more severely affected.

Ringing in the ears after noise exposure is most likely due to a temporary or permanent hearing loss. Often the patient vaguely perceives the tinnitus as ringing in the head, but cannot localise it to either ear.

Noise damage education and early identification of damaging noise levels may save many ears from potential permanent damage - "so all may hear".

More sophisticated testing and an awareness of the possibility can identify malingering and psychogenic hearing losses.

Noise Exposure Chart

Source	Noise Level (in dBs)	Safe Exposure
Gunshot or cracker	140	none
Jackhammer	130	3-8 minutes
Rock concert	120	5-7 minutes
Power drill	100	2 hours
Lawnmower	90	8 hours
Vacuum cleaner	80	no limits.

Tinnitus

Tinnitus is defined as a hallucination of sound in the cranium. It may be classified as originating either from the inner ear (ie, sensorineural tinnitus); or from mechanical causes outside of the inner ear (ie, mechanical tinnitus).

Sensorineural tinnitus

This occurs more frequently in older men. Most patients may tolerate and live with the tinnitus but a minority request medical and psychological assistance. Suicide attempts may be a remote possibility.

The patients may pinpoint the tinnitus to the ear, but most patients experience the tinnitus as diffusely scattered noise somewhere in the cranial vault. The tinnitus may be interpreted subjectively by the patient at a loudness level of about 35 dB at a frequency of about 3500 kHz. This high-pitched tinnitus is by far the most common type

Unfortunately little is known about the pathophysiology of sensorineural tinnitus. It may be caused by damaged hair cells in the organ of Corti discharging spontaneously. The patient may interpret and perceive it as tinnitus similar to phantom pains following limb amputation.

Mechanical tinnitus

- Mechanical causes may be due to abnormal haemodynamic flow patterns causing friction and abnormal vascular turbulence. The abnormalflow pattern may be due to narrowed vessels (ie, diseased by atherosclerosis), with vessels impinged upon by bony spurs or arteriovenous fistulae. A low frequency hum synchronous to the patient's heartbeat may be perceived by the patient as tinnitus. The attending doctor may also hear the murmur with a stethoscope.

- Myoclonic spasms of the two intratympanic muscles may be heard as sharp clicks (200-300/min) especially in patients with multiple sclerosis.

- Tumours such as paragangliomas of the temporal bone typically present with pulsatile tinnitus.

- Eustachian tube dysfunction may result in a perception of body sounds, such as clicking, due to movement of exudates in the middle ear.

Treatment

Treatment is directed at the cause. Myoclonic spasms of the middle ear muscles are well treated with botulinum toxin deposited into the middle ear muscles. Surgical ablation of these middle ear muscles may be a permanent solution after a diagnostic trial with botulinum toxin injections.

Arteriovenous fistulas may be surgically ablated or embolised and/or occluded by transarterial balloon occlusion.

Noise exposure

Noise exposure may cause termporary or permanent tinnitus. Explosions close to the ear (eg, fire crackers or gunshots) may cause tinnitus as well as a high tone hearing loss. The hearing may partially return but the tinnitus may remain independently. In the first instance acoustic trauma and tinnitus may follow a short exposure to a very loud noise or prolonged exposure to moderately loud noise.

Trauma

Trauma to the skull (eg, motor vehicle accident or a blow to the ear) may cause tinnitus codependent on high tone hearing loss. The hearing may be partially affected, ie, sensorineural or conductive loss. Surgical trauma to the inner ear, ie, stapedotomy or middle ear surgery, may cause high tone sensorineural loss and sensorineural tinnitus temporarily or permanently.

Conductive hearing loss (eg, otosclerosis), a dislocated middle ear ossicle, impacted wax in the external ear canal or foreign bodies in the ear canal may precipitate a 'sea shell' effect.

The patient may interpret the Brownian molecular movement onto the tympanic membrane as tinnitus.

Ototoxic drugs

Some drugs (eg, salicylates) may cause temporary tinnitus with the degree of tinnitus proportional to the toxic dosage. On discontinuing the salicylates the tinnitus may completely disappear without any trace of inner ear damage. Other ototoxic drugs may cause permanent inner ear damage with lasting tinnitus.

Old age may also be associated with high tone tinnitus, so-called presbytinnitus, with associated presbycusis.

With clear cut cochlear damage, as with trauma to the petrous temporal bone, tinnitus is confined to the ipsilateral ear. With ototoxicity, presbycusis, as well as with noise induced hearing loss both ears are usually affected.

Tinnitus may be an early warning sign of an underlying inner ear disease or a tumour impinging on the cochlear nerve (ie, an acoustic neurofibroma). Audiological pitch and loudness matching will reveal a loudness of about 35 dB, equal to the intensity of whispering and a frequency of about 3.5 kHz. This roughly matches the pitch of a cicada. Diagnostic audiology may further reveal the site of the hearing loss (ie, in the cochlear or in the vestibular nerve). Brain stem evoked response audiometry is especially useful in this regard.

X-ray studies and a scan may reveal unexpected underlying pathology (eg, a soft tissue intracranial tumour impinging on the cochlear nerve or an intracranial vascular lesion). It should be noted that acoustic neurofibromas constitute a not insignificant minority of all intracranial tumours in Caucasian populations. Any patient presenting with unilateral inner ear pathology should be referred for further investigation.

In the majority of cases most special studies are negative and the cause of the tinnitus may not be identified. The essence of diagnosis is to distinguish cochlear (end organ) from retrocochlear (cochlear nerve and brain stem) pathology that may be treatable.

Treatment

Treatment is tailored to the cause. If X-rays and audiology do not demonstrate a retrocochlear lesion, palliative therapy is prescribed.

- Tranquilizers or a hypnotic drug may occasionally be necessary for a short period to combat insomnia associated with severe tinnitus. Typically, a patient has difficulty falling asleep and cannot go back to sleep once awakened.

- In recalcitrant cases, the patient may need an evaluation by a psychologist or psychiatrist who may prescribe antidepressant therapy. For the right patient this may reduce the tinnitus from an intolerable level to a tolerable one.

- Some maskers are effective, either of the ear canal type or a noise simulator located close to the patient. Different types of noise may be programmed (eg, a waterall, rain on a tin roof, or the splashing of waves on rock). The same effect can be achieved with an 'off-station' bedside radio tuned to be barely audible to the patient. The 'white noise' emitted by the radiomay neutralise the patient's tinnitus and displace the sound source extracranially. This more natural noise is psychologically more tolerable to the patient.

- Fitting of a hearing aid will often have a useful tinnitus masking effect.

- Ipsilateral or contralateral masking depends on the hearing level of the tinnitus emitting ear. If the tinnitus is present with a profound hearing loss on the same ear, it may be better to identify the tinnitus pattern and to apply the masker to the contralateral hearing ear (central masking). Many patients with tinnitus may have normal hearing.

- The masking may adversely, favourably or tepmorarily affect the tinnitus depending on the individual response. Patients may experience a total suppression of the tinnitus, others may experience a summation effect and yet others may experience a temporary relief due to habituation of the masking sound.

- In some patients the tinnitus may abate for hours to days after the maskers have been switched off. The masking techniques of patients with tinnitus calls for patients' experimentation and adjustments.

- Patients with otosclerosis may benefit from a stapedectomy (or stapedotomy) although some patients may report a worsening of the tinnitus following a stapedectomy (or stapedotomy). Cochlear implants may have similar unpredictable effects on tinnitus. Surgery for Ménière's disease may improve the tinnitus.

- Surgical transection of the cochlear nerve or cochlear ablation may worsen the tinnitus and has no place in its management. Psychological reassurance and explanation of the possible causes to the patient may relieve anxiety. Stellate ganglion block may have a favourable effect in some patients.

It must be recognized that all forms of treatment may further aggravate tinnitus. It is a most difficult symptom to treat effectively and all modes of therapy must occasionally be

utilised to relieve the symptoms. Referral to an otologist is usually necessary to explore adequately all treatment regimens and to exclude unexpected underlying causes.

Hearing aids

Hearing aids and their batteries are modern technological wonders. Medical electronics has found many innovative ways to assist man in his quest to restore hearing loss without sacrificing cosmetic appeal.

Hearing aids provide sound amplification and may be used for hearing losses ranging from mild to profound, provided the inner ear has some residual function.

Hearing aids can be grouped into:

- Body aids.

- In the ear aids.

- In the ear canal aids.

- Behind the ear aids (ear level).

- Bone conducting aids.

In the aged and in children, where manual dexterity may be a problem, a body aid or a hand held remote control may be most practical.

Ear level aids have small controls and batteries and need skill to tune the aid and manipulate it into place.

Headband bone conductors and spectacle bone conductors are rarely fitted and only for very special selected patients (ie, bilateral congenital ear canal stenosis) and for patients with severe local ear infection involving mastoid cavities. They are cumbersome and uncomfortable to say the least. The sound quality is also not as good as air conduction aids.

Hearing aid prices may vary greatly depending on the manufacturer's level of technology. The smaller the hearingaid, the higher the price.

What is required from a hearing aid depends on the quality and quantity of residual hearing. Amplification may be over a wide or a narrow selected frequency range depending on the individual requirements. Hearing aids may be fitted to one or both ears. Fitting binaural hearing aids helps the patient hear stereophonically, and ambient background noises are suppressed because of 'focused' hearing.

Some persons with a profound unilateral hearing loss may find the cross routing of sound (CROS) to the better ear valuable, especially in situations such as boardroom meetings.

Patients with minimal to moderate hearing loss may be in a grey area for using a hearing aid and may only need one on selected or special occasions like round a conference table, at the cinema or during church gatherings.

Fitting patients with recruitment may be very difficult.

Hearing aids should be fitted in children as soon as the hearing loss is established, no matter how young. Hearing can be accurately evaluated at a very young age with conventional testing and conditioning or with audiological evoked brain stem responses (ABR) or otoacoustic emission. The hearing assessment does not necessarily need the cooperation of the child: it may be done while the child is asleep. Auditory stimulation is of paramount importance to the developing child to prevent speech impairment, intellectual and educational handicaps. Auditory stimulation in early infancy may reinforce the development of central auditory pathways.

Once the correct hearing aid has been fitted the child may attend a school for the deaf, hard-of-hearing or a mainstream school, depending on the degree of hearing loss.

To achieve optimal hearing from a hearing aid requires expertly fitted moulds which can increase the performance of the hearing aid as well as to minimise the disturbing occurrence of feedback.

Bilateral hearing aids are recommended if both ears are hard of hearing. Binaural hearing allows the sufferer to hear stereophonically and to focus on the sound source thus improving hearing and understanding, especially in noisy surroundings.

Hearing aid performance has to be accurately matched to the hearing loss.

Technical instructions by the audiologist in the care and use of the hearing aid, as well as follow up instruction is invaluable. External parts are controlled by the patient; internal settings are the responsibility of the acoustician or audiologist. Breakdowns or malfunctions of hearing aids should be detected early, especially with children. Because the wearer may not be aware that the hearing aid is not fully functioning, it should be checked regularly (ie, quarterly). Basic malfunctions such as the hearing aid needing a new battery should be excluded first.

Adequate follow up by a medical practitioner and an audiologist to check for impacted wax in the ear canal as well as for changing hearing needs is recommended.

The golden rule before a purchase is finalised is that a hearing aid should be on trial for one month and used under various conditions (ie, silence, conversation, listening to TV, in a shopping mall and in a busy room such as an office or schoolroom). Patients need to realistically evaluate their expectations.

Cochlear implants: Bionic ears

The cochlear implant device converts sound to coded electrical impulses, bypassing a totally defunct cochlea. This contrasts with the conventional hearing aid, which amplifies

sound to a high energy sound level, stimulating a partially functioning cochlea.

Although the present state of the art implant is still comparable to the Model T Ford, it must be remembered that the latter was, even in its simplicity, a much superior alternative to other modes of transport at that time. During the 20th century vast improvements in sound quality and miniaturisation were possible especially due to the USA space programme.

Patient selection

Recipients of cochlear implants may belong to one of three groups:

- Postlingual adults or children (ie, those who could talk) who have lost their total hearing on both sides after speech development (eg, following meningitis, skull trauma and ototoxicity).

- Prelingual children (ie, infants and young children who have never heard before).

- Prelingual adults (ie, adults who have never heard before).

Postlingual patients

Cochlear imlants are usually successful in postlingual patients as these patients are familiar with sound, and sound interpretations, as well as speech pronunciation. Initially, the new sound may be 'metallic' or strange, but these patients soon adapt. Within one month of auditory therapy they may already experience the full benefit of the implant.

The new sounds are sometimes described as 'a radio off the station at the end of a tunnel'. Nevertheless, with the aid of lip reading, the patient may communicate almost normally, and in some cases a simple telephone conversation is possible. The determining factor in this group of patients is how many intact neurons are available in the cochlear nerve. At present this cannot be determined, but clinical tests are being developed to quantify this important aspect in an effort to predict results.

Infants and young children

Although it is difficult to ascertain the exact hearing level of infants and young children, modern techniques are available that can be helpful (eg, evoked brain stem responses and electrocochleography). It is now well established that cochlear implants in postlingual children and infants who have lost their hearing following meningitis are as successful as those in adults who have lost their hearing.

Prelingual deaf children now fall within the application of this technology. The cochlea of a child is the same size as that of an adult, so technically it is no more difficult to perform the procedure in children than in adults.

Children who have never heard before (prelingual children) are in a different category. Ideally, a potential paediatric candidate should receive an implant as early as possible to gain the full benefit of the child's speech development years.

Children must receive auditory therapy for many years, as well as psychological therapy to full exploit the potential of the implanted bionic ear. The role of the family and community in the support and development of the child's full abilities cannot be overemphasised.

The ABC system (anchored bone conduction system)

The anchored bone conduction system is an electronic device that is cost effective and efficient. Designed for hearing amplification, it delivers high quality sound perception via bone conduction. The system consists of a semi-implantable portion as well as a body aid amplifier. The implant is done in two stages over four months.

The ABC system is especially appreciated by hard-of-hearing patients who cannot tolerate conventional ear level hearing aids tue to:

- A 'blocked' sensation in both ears due to mild pressure.

- Occasional embarrassing acoustic feedback leakage at high amplification gain.

- Recurrent ear canal infection due to tight fitting ear canal moulds.

- Allergic canal skin reaction to the ear mould.

- Congenital middle and external ear canal abnormalities.

The system consists of a conventional body hearing aid a modified bone conductor and a titanium osseous implant implanted *postero superior* to the pinna.

Because infection may occur, diabetic and immunocompromised patients are not ideal candidates and should be excluded.

All the components are of South African origin.

The development effects of early acquired minimal hearing loss

Moderate to severe bilateral loss is relatively easily detectable in the paediatric age group. The daunting sequelae of behavioural problems, delays in intellectual and cognitive development, and later, lack of educational progress, is early proof of a communication disorder.

On the other hand, slight acquired fluctuating unilateral or bilateral conductive hearing loss in the previously otherwise normal infant may go undetected for many months before diagnosis is made and therapy applied. Even during a period of medical treatment or awaiting surgical management there is often a casual attitude towards minimal hearing loss "because the child is on therapy".

Lack of progress is often ascribed to social problems (eg, single parenthood, divorce or lower socioeconomic conditions). Although intelligent children may compensate to a

degree, they miss the opportunity to achieve their full potential.

It is often considered that hearing loss in the paediatric age group of 15 dB is 'acceptable' or may even fall within the range of testing limits. A 0 dB hearing threshold is set for adults, yet in the paediatric age group the hearing threshold may be below 0 (eg, -5 dB), which means that hearing loss would have to reach 20 dB to be considered significant.

Minimal hearing loss in most instances is due to mechanical factors in the middle ear (eg, otitis media with effusion (OME) or 'glue ear').

It is well known that in the developing child the auditory system needs perfect input for optimal function and development. Sound deprivation in developing animals reveals a lack of neural tract development between the hearing nuclei and the second stage hearing nuclei in the brain stem. This represents permanent damage to the auditory feedback in humans, a system that allows us to 'focus' on a sound source (eg, speech) in a noisy environment such as at a party.

Auditory processing deficits are commonly responsible for a large proportion of language learning disorders and this is currently receiving much attention in the educational system. Undiagnosed and untreated minimal hearing loss may result in:

- paediatric behavioural problems like attention deficit disorders (ADD), hyperactivity, disobedient behaviour, day-dreaming, trouble making and non-cooperative antisocial behaviour; and/or

- decreased cognitive development resulting from inadequate auditory cortical stimulation.

Later in life, these personality, behavioural and cognitive development disorders may have a ripple effect on:

- academic achievements and ability to compete with peers - although the child may achieve, he or she may not reach full academic potential,

- behaviour and self esteem - antisocial behaviour and lack of self esteem may lead to rebellious behaviour patterns and severe social maladjustment (eg, delinquency and crime).

Early detection

Early detection of minimal hearing loss is essential. The primary health care practitioner is often the first patient contact. A risk children should be identified early, which is a function of an index of suspicion.

Ear infection, especially repeated ear infections, should be followed-up. The vast majority of acute otitis media cases will adequately resolve with proper treatment, but if otitis media with effusion does not resolve spontaneously, prolonged antibiotic therapy is necessary. In a minority of cases, otitis media with effusion persists in spite of adequate treatment, and specialist referral is necessary.

It is estimated that two thirds of children will have experienced an attack of acute otitis media before the age of seven. This makes the 'minority' of cases within this group, those with persistent glue ear, a significant number. It has reached epidemic proportions in certain socioeconomic groups! These cases are at risk and should be carefully followed by their family doctor, explaining to the parents the potential sequelae of hearing loss. These warnings may encourage parent compliance and cooperation.

Electrophysiological devices are available to the busy GP to detect these middle ear abnormalities; a good otoscope with a Siegle speculum attachment is the first line of diagnostic evaluation. A tympanometer and an impedance audiometer can easily and fairly accurately evaluate the middle ear status. If further doubt exists, then otological and audiological assessment may be obtained.

Deeply impacted wax is often a problem and may lead to erroneous interpretation of hearing results: it may be difficult to remove even by a specialist.

Mass preschool screening by community nurses using tympanometry and audiologic screening devices is another line of early detection of minimal hearing loss.

Once a patient at risk is identified and diagnosed there follows a period of conservative management and observation to determine the spontaneous resolution of the hearing loss before surgical management is attempted.

An observation period of three weeks from first diagnosing the condition is often allowed before referring the patient for specialist evaluation. During this time the child is hearing deprived. A practical analogy is to adjust TV volume to a comfortable hearing level then reduce the volume 50 percent and note how difficult it is to grasp the full content of the programme. If a further period of evaluation by an otologist is added, the child is hearing deprived for a large part of his or her academic year.

During the time of treatment special educational provision is necessary to avoid education deprivation. These may include preferntial seating in the front of the class, remedial class and teacher awareness (eg, making eye contact and facing the class while speaking to facilitate lip reading). A letter from the family doctor to the schoolteaher or principal will often encourage teacher cooperation.

The difference between the daunting long term ramification of minimal hearing loss on the child's future on the one hand, and success on the other, is the index of suspicion of the primary healthcare sector and primary educators. Education is primarily a function of hearing.

Acoustic neurofibroma (vestibular schwannoma)

Acoustic neurofibroma derives from the perineural Schwann cells arranged around the stato-acoustic nerves in the internal ear canal. For unknown reasons the schwannoma has a predilection of presenting in the bony internal ear canal.

As the tumour increases in size, it compresses on the seventh and eighth nerves causing gradual and subtle function loss (ie, slow and progressive facial palsy, hearing loss, tinnitus and unsteadiness). With progressive increase in size, the tumour may project and bulge from the internal auditory canal into the cerebellopontine angle. The brain stem, cerebellum, and the fifth nerve may be implicated by the expanding pressure of the tumour with loss of touch sensation and loss of the corneal reflex.

Signs

Hearing tests may reveal a specific pattern of hearing loss. Speech discrimination may be poor in relation to the pure tone signals. This is strongly suggestive of a retrocochlear pattern of hearing loss.

Balance tests may reveal an ipsilateral hypofunction of the horizontal semicircular canal *(canal paresis).* Above threshold audiometry and auditory brain stem response (ABR testing) have typical findings that may quantify the hearing loss as well as indicate the site of the lesion. Upon these clinical findings MRI and CT scans may confirm the diagnosis.

These tumours frequently masquerade as other ear disorders. However, the success in diagnosing them parallels the index of suspicion: the more often they are suspected, the more often they may be positively diagnosed. Missed diagnoses are increasingly becoming a medicolegal risk in certain countries.

Tuning fork tests

Tuning fork tests are a valuable diagnostic tool for the doctor to screen and clinically evaluate the hearing status of a patient. Conducting the tuning fork range of tests also allows the doctor direct clinical contact with the patient to establish the reliability of the audiological test results. It gives an opportunity to detect malingerers because responsibility is not abrogated to a third person.

The most common medial tuning fork employed are the 256 Hz and 512 Hz ones. These frequencies represent the lower end of the speech and vocal frequencies. Properly applied the tests can reliably determine:

- if a patient has normal hearing in one or both ears;

- if a patient suffers from conductive hearing loss (ie, mechanical deafness of some sort in the external or middle ear) in one or both ears;

- if a patient suffers from sensorineural deafness (ie, originating from the inner ear or auditory nerve) in one or both ears;

- if a patient is faking deafness (ie, malingering). Special hearing tests may then be necessary to confirm the suspicion. Shrewd malingerers can fake their response to almost all hearing tests. A neurolept ABR audiogram (ie, a hearing test under a light anaesthesia) may be necessary to remove the cognitive or intellectual component in order to obtain the true hearing status.

The tuning fork tests may corroborate an audiogram performed by an audiologist or other third party.

Three types of tuning fork tests are used.

- A comparison between the patient's hearing and the tester's hearing (ie, the Schwabach test).

- A midline test to establish if both ears hear bone conduction equally (ie, the Weber test).

- A comparison between the bone and air conduction of each ear (ie, the Rinne test).

The Schwabach test

Activate the tuning fork by tapping it lightly on a bony point (ie, the knuckle of the examiner's hand). Hold it about 4 cm from the examiner's ear). Just before the tuning fork vibration completely disappears the tuning fork is placed the same distance (about 4 cm) from the patient's ear).

Interpretation

If the patient also perceives the sound of the tuning fork vibration, the patient's hearing is normal compared to the examiner's hearing: presuming the examiner has normal hearing. It is thus useful for practitioners to establish their hearing levels regularly. This is also true for other clinical assessments such as stethoscopic auscultation.

The Weber test

Activate the tuning fork by tapping it lightly on a bony point (ie, the knuckle of the examiner's hand). Place the tuning fork firmly on the central forehead or on the midline front teeth. Ask the patient on which side he hears the sound (ie, central or to the left or right side of his head).

Interpretation

If the patient hears it centrally the result is normal. If the patient hears it in the deaf ear the hearing loss is conductive (ie, the inner ear is still active but the conduction mechanism is faulty). If the patient hears it only in the normal ear, there is a sensorineural hearing loss.

The Rinne test

Activate the tuning fork by tapping it lightly on a bony point (ie, the knuckle of the examiner's hand). Place the tuning fork about 4 cm in front of the ear for a few seconds and then place it firmly on the mastoid eminence behind the same for a few seconds.

Interpretation

If the patient perceives the sound intensity louder in front of the ear (ie, air conduction), the result is normal and is called Rinne positive. If the patients perceives the sound louder with the tuning fork on the mastoid eminence (ie, bone conduction) it is called Rinne negative. A Rinne negative result suggests a conductive deafness due to a mechanical fault with the tympanic membrane or middle ear ossicles in the presence of a normally functioning inner ear.

To identify malignerers

If both ears are suspected normal (as evaluated by the Schwabach test), the one ear may be occluded with fingertip pressure. The activated tuning fork is placed in the centre midline position on the forehead. The malingerer may reason the occluded ear should be deaf because it is occluded and will claim to hear the vibration only with the ear that is not occluded. He should, of course, hear the sound inthe occluded ear because the inner ear is still active. Examiners can verify this basic principle by conducting this simple but effective test on themselves.

Facial nerve palsy

Facial nerve palsy is a symptom. Partial or total hemifacial nerve palsy causes cosmetic, communicative and masticatory defects to varying degrees. In addition, the cornea may be at risk of ulceration, scar tissue opacities and subsequent loss of vision.

Underlying causes, some of which may be serious, must be excluded. The temporal bone is associated with 95 percent of facial nerve palsies. When the cause is unknown, it is termed Bell's palsy.

Bell's palsy

Bell's palsy is thought, although not absolutely proven, to be caused by viral infections ie, EB virus or herpes simplex virus. The condition may progress to varying degrees of paralysis. Often the patient notices the palsy while shaving or drinking a cup of tea after waking. Concomitant symptoms may include ipsilateral otalgia and facial numbness (polyneuritis).

Hearing is not affected and vertigo is not present

Aetiology is suspected to be ischaemic neuropathy or a central nervous system viremia (ie, Epstein-Barr virus or herpes simplex). As yet no cause has been positively identified.

Tests should exclude an unexpected underlying pathology. Audiology tests (ie, the stapedial reflex) may reveal the level of palsy of the facial nerve. Mastoid X-rays, CT scans and MRI scans may be normal.

Prognostic testing is by comparing the paralysed side of the face with the healthy side.

Prognostic tests include:

- tear producing tests (Schirmer's test) and submandibular salivary flow test, both of which may be performed in the consulting room,

- stapedial muscle reflex test, performed by an audiologist,

- threshold and supra threshold facial nerve excitability tests - again, these may be performed in the consulting room and require special equipment.

Electroneuromyography gives an indication of the degree of deterioration of the nerve function. An EMG (electromyography) may indicate the recovery of the nerve.

If the palsy is incomplete there is a 95-100% recovery rate. Full recovery is less likely in aged patients with diabetes or hypertension.

Treatment

For patients with partial palsy no treatment is indicated other than regular observation. For those with total palsy, the following treatment is recommended:

- Corticosteroids - the patient is placed on a tapering dose of prednisone over a three week period. The corticosteroid may also minimise post healing synkinesis (ie, when the affected eyelid blinks, the ipsilateral mouth corner also moves or twitches involuntarily). This may be the result of crossover neurone regeneration in an oedematous segment of the nerve with concomitant scar tissue formation.

- Eye caps and natural tears - an eye cap is a transparent self adhesive plastic device that protects the eye from dehydration and corneal ulceration. Eye pads may damage the cornea as the upper eyelid levator is not paralysed and opens the eye, allowing the pad to scratch the cornea. At night and during the day additional natural tears applied to the eye will keep the cornea moist and prevent potential complications such as corneal ulceration.

- Psychological support and reassurance - it is important to reassure the patient that recovery is imminent and full recovery may be achieved within four to six weeks.

- Antiviral therapy is gaining support, but surgical management remains controversial.

Facial nerve palsy is a severe cosmetic, functional and psychological insult to the patient. The patient needs ample psychological backup and reassurance from the attending medical staff.

Viral infection: herpes zoster oticus

Compared with Bell's palsy of the facial nerve, herpes zoster facial palsy is worse and the prognosis is poorer.

The causative agent is the chicken pox virus (varicella). This self limiting disease occurs as a second stage viremina in patients with an incomplete immunity against the virus or lowered cell mediated immunity (ie, following ratiotherapy, carcinoma, chemotherapy and in HIV patients). In other sites of the body it is commonly known as shingles.

Strictly unilateral vesicular eruption may occur on the pinna and around the external ear canal, face and in the mouth. The vesicles form crusts and drop off at about 21 days.

Neuritic facial pain may be mild and transitory. In a minority of cases persistent devastating pain may last for months to years. Accompanying facial nerve palsy **(Ramsay Hunt syndrome)** may call for additional eye care.

The swelling of the nerves in the internal auditory canal may cause vestibular and acoustic nerve involvement with total hearing loss on the same side or vertigo and vestibular function loss on the ipsilateral side.

Treatment

Palliative treatment is indicated for pain, fever, and malaise during the onset of the syndrome. The vesicles on the pinna are best left alone. Warm compresses may ease the pain and discomfort over the pinna.

If facial nerve palsy is present, eye care should be implemented to protect the cornea from dehydration and corneal ulceration with possible corneal scarring and loss of vision. Synthetic tears may suffice. A self adhesive eye pad is comfortable for the patient to wear. It creates a small, moisture filled chamber to protect the corena from dehydration and subsequent corneal ulcerations.

Steroids in a tapering dosage may reduce postherpetic neuralgia. However, these are contraindicated if herpes zoster ophthalmicus is present.

Antiviral agents (ie, acyclovir) given during the acute phase may reduce facial palsy and synkinesis and may reduce pain.

In other instances, palsies may be caused by tumours, acute otitis media, viral infections or cholesteatoma. Herpes zoster oticus appears rarely in young adults, and if the patient falls into this age group he or she should be tested for HIV. Extratemporal palsies (eg, those caused by forceps delivery or facial lacerations and parotid gland tumors) are seldom encountered.

Rare cases of facial nerve palsies

Guillain-Barré syndrome

Guillain-Barré syndrome usually starts with an ascending motor paralysis of the lower extremities. Rare bulbar, myelitic and cerebral forms of Guillain-Barré may not affect the lower extremities and may cause selective bilateral facial nerve glossopharyngeal or vagal nerve palsies. This condition is becoming prevalent in HIV/AIDS patients. Bilateral palsy is

rare.

Bell's palsy during pregnancy

A special physiologic neuropathy that behaves like Bell's palsy may appear during the third trimester of pregnancy. It spontaneously clears after partition. Management is difficult because of the risks of using steroids in pregnancy.

HIV/AIDS

The HIV virus is neurotropic and may primarily affect the facial nerve as a demyelinating polyneuropathy. In the later stage, as AIDS develops, immunodeficiency may be responsible for an opportunistic viral infection involving the facial nerve (ie, herpes zoster or herpes simplex).

Lyme disease

Lyme disease is a tick borne spirochetal disease sometimes presenting with neurologic abnormalities such as unilateral or bilateral facial nerve palsies.

The palsy may spontaneously resolve with treatment of the initial disease. This disease may be successfully treated with broad spectrum antibiotics.

Barotrauma

Barotrauma may cause recurrent palsies every time a critical barometric zone is transgressed. Grommets may relieve the problem.

Palsy due to trauma

The trauma may be accidental (ie, motor vehicle accident, sports injuries, gunshot wounds) or may be iatrogenic. Iatrogenic facial nerve palsies may occur during otologic surgery. Congenital temporal bone abnormalities and variations in the course of the fallopian canal (ie, the canal that conducts the facial nerve) place the facial nerve at risk during surgery. Longitudinal or transverse temporal bone fractures may transect the facial nerve along the long and narrow course of the fallopian canal

If the palsy is acute, surgical exploration is indicated after an observation period to allow oedema and the patient's general condition to stabilise. CT scans and hearing tests may indicate the site of injury. A delay of up to three weeks following the injury may still give satisfactory surgical results.

If the palsy has a delayed onset, excellent results may be obtained with non-surgical steroid therapy.

1. Congenital Deafness. Universal hearing screening programmes are available for high risk babies. Safe, noninvasive test procedures using an oto-acoustic emission (OAE) screening device are available to test newborns as early as ten hours after birth. Early

amplification is mandatory for normal cognitive and speech development.

2. Acute vertigo (vestibular neuronitis) may occur within minutes and may last for several weeks. The aftermath of unsteadiness, motion sickness and inability to walk in the dark may linger for many months. The aetiology is uncertain: a viral infection is suspected. A tumour or inner ear membrane rupture should be excluded. While viral and vascular causes are the most common, a concerted attempt must be made to identify life threatening treateable causes (ie, a perilymph fistula).

3. Sudden hearing loss is an acute ENT emergency. Younger patients have a better prognosis than older patients. Early hospitalisation and treatment may result in improved hearing.

4. Perilymph fistula is a traumatically induced tear of the membranous inner ear at the round window. Vertigo may clear up within 10-14 days. However, the hearing loss may be partial or the tinnitus permanent.

5. Benign paroxysmal positional vertigo (BPPV) causes vertigo in certain trigger positions when a dislodged otoconia rolls out of the utricle and lands on an adjacent cupula of a semicircular canal. An acute spinning vertigo may be triggered. A typical position is instilling eye drops when standing - or just rolling over in bed. BPPV may be a self limiting disease over a period of time.

6. The Dix-Hallpike test: The patient is placed in a sitting position at the end of a bed and then jolted backwards while the head is simultaneously rotated to one or the other side. A rotational nystagmus and a sensation of spinning vertigo may confirm a positive test. The test results typically peter out. Retesting may not provoke a subsequent BPPV response. This manoeuvre is a three dimensional body/neck movement. A dislodged otolith may be jolted onto the cupula of the semicircular canal to precipitate the symptoms, confirming the diagnosis.

7. Motion sickness. To maintain the erect body position, information from the visual system, balance organs and proprioceptors are integrated in the brainstem. When conflicting information from any of these systems mismatches with information from the balance organs, the motion sickness syndrome may be provoked. Hence, without balance organs, motion sickness cannot be elicited.

8. Presbycusis - old age hearing loss. Of the five senses, hearing is the first to suffer from ageing. Some octogenarians may be severely handicapped while others experience only a mild hearing loss. Males are more often affected than females. Genetic predisposition to the early onset of presbycusis may account for the individual degree and age of onset.

9. Ménière's disease. The pathophysiology of Ménière's disease is hypothetical. Raised endolymphatic pressure in the inner ear may disturb normal inner ear function. Balance and hearing may be adversely affected by varius degrees.

10. Ototoxic drugs. An informed consent for treatment with ototoxic drugs may be a prudent decision to avoid medicolegal complications. The potential toxic effect on the inner

ear, balance organs and kidneys should be clearly explained to the patient, ie, an informed consent for therapy.

11. Noise induced hearing loss. Severe permanent noise damage may occur within seconds of an explosion close to an unprotected ear. The damage may resolve in a matter of weeks or may be permanent. The severity of NIHL depends on the noise level and duration.

12. Tinnitus may be pulsatile suggesting a vascular lesion. Continuous high pitched tinnitus suggests inner ear or nerve pathology. Causes may vary from ototoxic drugs to tumours or unknown aetiology. Intermittent tinnitus suggests Eustachian tube disorders. Tinnitus in the aged is usually of a continuous high pitched quality simulating the sound of a cicada (roughly 3.500 kHz). The aetiology may be past noise exposure - this is frequently seen in war veterans who have served with the artillery.

13. Hearing aids may be divided into five types: 1) In the ear canal. 2) In the ear. 3) Ear level. 4) Body aid. 5) Bone conduction aid. As a rule the smaller the hearing aid, the bigger the price tag.

14. The cochlear implant is a medical technological wonder. It may assist the totally deaf patient to perceive sound and in some cases to restore speech conversation, even via a telephone.

15. The ABC system (anchored bone conduction system) is an electronic hearing amplification system that delivers high quality sound via bone conduction. 1) Titanium implant joint. 2) Ball and socket joint arrangement. 3) Modified bone vibrator. 4) Body aid sound amplifier. In the correctly selected patient it may be a blessing.

16. Audiometric testing on a patient with suspected serous otitis media or 'glue ear' may reveal minimal to moderate conductive hearing loss. A flat tympanogram is indicative of middle ear fluid. However, it is not always possible to test a rambunctious youngster and clinical examination may be the only diagnostic information. The family doctor, who is the first patient contact, should not hesitate to refer the patient for specialist evaluation. Minimal hearing loss in the preschool age group may lead to speech impairment and may cause a delay in cognitive development. Soft plosive sounds (ie, 'p' and 't'), and high tone fricatives (ie, 's' and 'f'), may be omitted in speech for lack of correct interpretation. Otoscopy and tympanometry are of paramount importance in the evaluation of the status of the middle ear.

17. Acoustic neurofibromas of the inner ear nerve (stato-acoustic nerve) frequently masquerade as other ear disorders. The more often they are suspected, the more often they may be diagnosed. Widening of the bony inner ear canal confirmed on radiology strongly suggests a growth in the ear canal causing bone absorption.

18. Tuning fork tests allow the doctor direct clinical contact with the patient to establish the reliability of other test results and to identify malingerers.

19. Facial nerve palsy. Partial or total hemifacial nerve palsy is a symptom. It may cause cosmetic, communictive and masticatory defects to varying degrees. Underlying serious causes must be excluded. The cause of the vast majority of facial nerve palsies lies within the

temporal bone.

20. Herpes zoster oticus. Incomplete immunity to the chicken pox virus may result in a relapse infection. Serosanguineous vesicles may appear on the concha of the pinna and on the palate. The facial nerve may be affected causing a unilateral facial palsy (Ramsay Hunt syndrome). The eye may be at risk. Inability to close the eyelids may cause drying of the cornea with possible corneal ulceration, resulting in corneal opacities and decreased vision.

21. Lyme diseases is a tick borne spirochete which may cause a facial nerve palsy. The facial nerve palsy clears up with treatment of the tick bite fever.

22. Facial nerve palsy due to trauma. A temporal bone fracture may transect the facial nerve causing an immediate facial palsy. Temporal bone fractures may be due to a motor vehicle accident or sports injuries. The patient's general condition, ie, unconsciousness, in shock and with major organ injury may delay the diagnosis of facial palsy. A bloody or cerebrospinal fluid otorrhoea may draw the attention to the temporal bone skull injury.

Nose

Nose and paranasal sinuses

Epistaxis

Idiopathic or spontaneous epistaxis occurs predominantly from the septum. In the under 35 age group (the vast majority), it is a common and short lasting condition; mostly venous in origin and from the retrocolumellar vein of the septum (the nose-picking zone). In the older group (the minority), the bleeding is mostly arterial in origin and from Kiesselbach's plexus or Little's area on the caudal septum.

Some patients may bleed from the roof of the nasal vault. This is especially associated with hypertension, artherosclerosis or as a result of certain medications (eg, aspirin and anticoagulants). The vessels concerned are the ethmoidal and sphenopalatine arteries.

In older patients with arterial bleeding, epistaxis may be prolonged and extensive blood loss may occur. In the older patient with marginal cardiovascular function, severe epistaxis is poorly tolerated and cardiovascular decompensation may be precipitated.

Contributing factors to epistaxis may be minor nasal trauma, such as nose-picking or forceful nose blowing. Dry, warm air may exacerbate the problem due to mucus crusting on the nasal septum, especially if the septum is displaced into the air stream.

Treatment

The nose pinching option, where pressure is exerted on Little's area of the septum for ten minutes, will usually stop a venous bleed in the younger age group.

Chemical cauterisation with silver nitrate sticks or beads may be performed under topical anaesthesia. Cocaine is useful because of its vasoconstrictive properties.

Electrocautery, either under local or general anaesthesia, may be indicated.

Ultrasonic ablation may cause less mucosal injury and be an alternative to electrocautery.

In inaccessible cases, control hypertension with sedation and do not interrupt antihypertensive therapy. Do not use rapid acting antihypertensives as they may precipitate hypovolemic shock. Monitor the blood clotting factors and platelets and make adjustments where necessary. Tamponade the nasal vault with expanding cellulose tampons or packing. These should be removed within 48 hours to avoid septicaemia and a pungent smell. Lubricating the tampons with an antibiotic cream may reduce bacterial proliferation on the tampons and reduce the smell.

An emergency septoplasty may be necessary to gain access if the bleeding point is obscured by a deviated septum.

Specific vessel ligation (usually of the internal maxillary artery) under general anaesthesia or local anaesthesia is seldom required. Such ligation, when needed, is carried out via a transmaxillary sinus approach. Less common is ligation of the ethmoid artery. Ligating the external carotid artery is usually unsuccessful as nasal blood flow forms an anastomosis between the internal and external carotid systems.

Therapeutic embolization with selective arteriography may be applied under direct X-ray guidance.

ENT sport injuries

Facial fractures

Mid-facial fractures are commonly seen in contact sport injuries. They may involve the maxilla, the nose and the mandible. As the nose is the most prominent facial structure, it is by far the most commonly injured. Much less common are malar bone fractures such as the tripod fracture, so-called because the three fracture lines run infraorbitally, supraorbitally and through the zygomatic arch.

Flying missiles (eg, golf or cricket balls) may be responsible for an orbital floor 'blow-out' fracture. The thin orbital floor literally implodes into the maxillary sinus because of the sudden pressure exerted on it by the compressed globe of the eye. Squash ball injuries, in contrast to those from golf balls, may injure the globe of the eye rather than the bony orbit. Conjunctival tears and abrasions as well as retinal detachment may cause total or partial loss of vision. In South Africa, a significant number of eyes are blinded annually by squash balls. Protective eye gear is mandatroy for squash players.

Cheekbone fractures

The tripod fracture involving the maxilla, the zygomatic bone and the frontal bone causes a 'blue eye'. Usually the soft tissue around the eye and the cheekbone is swollen; the cheek eminence may be flattened. The haematoma may extend subconjunctivally and the eye

may be displaced downwards with resulting double vision, especially on upward gazes.

An acute blunt injury to the eye globe may present with blurred or poor vision. There may be corneal abrasion, subconjunctival haemorrhage, blood in the anterior chamber (ie, hyphaema) or injury to the retina (ie, swelling and haemorrhage). A retinal tear is rare. Late complications may include glaucoma, cataract and retinal detachment.

Anaesthesia of the cheek may occur following damage to the infraorbital nerve, a branch of the second division of the fifth cranial nerve.

Treatment

First, exclude concomitant ocular injury; an ophthalmologic consultation is necessary.

- Unless the patient is dehydrated, an anti-inflammatory analgesic to control pain and swelling is desirable.

- Prophylactic antibiotics may be administrated.

- Avoid nose blowing: this may cause surgical emphysema over the cheek or in the orbit.

- A Watersž view X-ray may be sufficient to delineate the fracture lines.

- Surgical reduction and fixation of the fracture lines is indicated once the patient is clinically stable. The orbital floor should be explored simultaneously for herniation and entrapped orbital contents.

Orbital blow out fractures

The orbital globe is at risk due to the sudden pressure applied to the globe. An ophthalmological consultation is urgent. Symptoms include periorbital swelling and a 'blue eye' as well as conjunctival haematoma. Enophthalmos and limited eye movement cause double vision. Initial orbital swelling can mask enophthalmos and diplopia.

Treatment

Surgical exploration of the orbital floor is necessary to retrieve entrapped orbital structures from the maxillary sinus, after which the floor may be repositioned and stabilised.

Zygomatic arch fractures

Zygomatic arch fractures may occur in isolation through a direct blow to the side of the head. The arch prominence is flattened. Trismus is present because of the locking effect of the depressed zygomatic arch segment on to the coronoid process of the mandible. A submentovertical basal skull view X-ray will demonstrate the flattened arch.

Treatment is by surgical reduction: the segment is 'popped' into place.

Frontal bone fractures

Apart from the cosmetic deformity, the fracture line may pass through the nasofrontal duct or the posterior lamina of the sinus. Cerebrospinal fluid leakage via the nose may cause an ascending intracranial infection.

Nasofrontal fractures are surgically treated to prevent a mucocele with delayed and insidious intracranial complications. CT scans are mandatory to exclude a fracture in this region.

Acute nasal fractures

Because the nose is the most prominent facial structure in the majority of people, it is more exposed to trauma and the nasal fracture is the most common bone fracture in humans, especially in the adult Caucasian male.

Nasal fractures are caused by low velocity blows; high velocity injuries may also affect other mid-facial bones.

The most common cause of nasal fractures is assault, either domestic or in contact sport. Contrary to popular belief, boxing has a low incidence of nasal fractures, but a high incidence of cartilaginous septal fractures which may lead to the unsightly cosmetic defect of a boxer's or 'pug' nose, a supra tip concavity or a saddle nose deformity.

Greenstick bony nasal fractures in children may lead to abnormal nasal septal growth patterns with cosmetic and functional sequelae. Nasal fractures in children and women should always evoke the suspicion of child abuse or wife beating. All nasal fractures are open fractures with bleeding from intranasal mucosal tears.

Symptoms

The nose is painful and tender. Nasal airway obstruction may be present. The patient may complain of double vision which may signify orbital involvement.

Signs

The patient may present with epistaxis, swelling, oedema and ecchymosis of the overlying nasal soft tissue. If soft tissue swelling is severe, it may obscure the degree of nasal bone displacement and malalignment. The nasal septum may also be fractured and displaced.

A septal haematoma must be excluded or, if present, should be drained as soon as possible to prevent complications such as abscesses that may cause absorption of the supporting septal cartilage resulting in a boxer's nose.

X-ray confirmation of the nasal fracture is usually not necessary but may be required as medicolegal evidence. In certain cases keeping a permanent record may be advisable.

Treatment

- Control the epistaxis; most often epistacis can be controlled with an expandable cellulose tampon in each nasal cavity.

- A septal haematoma should be drained as soon as possible under sterile condition.

- Analgesics may be given.

- Prophylactic antibiotic use is controversial. All nasal fractures are comminuted.

- Nasal fracture reduction should be postponed until the nasal swelling has subsided, usually seven to ten days after the injury, to allow for accurate evaluation and positioning of the nose.

- X-rays may confirm the diagnosis as well as identify other facial fractures.

Complications include:

- A boxer's nose or a saddle nose deformity resulting in a cosmetic blemish and often associated with nasal airway obstruction. A septal haematoma forms in the sub-perichondrial space whereupon the cartilage may become devitalised and a septal abscess may result. The septal cartilage may then be absorbed, destroying the nasal bridge support in the supratip area of the nasal dorsum. Subsequent collapse may cause the so-called boxer's nose deformity resulting in a saddle or pug nose.

- A septal abscess with possible intracranial spread. Intracranial spread is possible via the ophthalmic veins as they collect infected blood from the so-called danger triangle. The ophthalmic veins may act as emissary veins, draining extracranial into intracranial structures.

- Cerebrospinal fluid leakage which may clear up spontaneously, but must be monitored accurately and repaired if necessary.

- Diplopia and epiphora may necessitate exploration of the orbital floor or the nasolacrimal apparatus.

- Cribriform plate fracture/s with potential complications.

The jaw and teeth

A jaw fracture is usually the result of a low velocity, high energy impact injury. The teeth may also be involved in the fracture or separately knocked out. Knocked out teeth should be retrieved for reimplantation.

Because the forceful impact may be transferred to the cervical spine and cranium, an associated **spinal injury** and **intracranial injury** should be kept in mind, especially in the unconscious patient. The head should be immobilised in relation to the torso during transportation.

Symptoms

The patient usually presents with a speech defect due to the painful trismus. Examination may reveal an expanding haematoma of varying size over the fracture site, extending into the oral cavity. Haemorrhage from mucosal tears may be extensive.

Treatment

- Remember the ABC of jaw fractures: **A** - airway, **B** - bleeding, **C** - cervical spine.

- The profuse haemorrhage and expanding haematoma may compromise the oropharyngeal ariway and an emergency tracheotomy may be indicated to secure an adequate airway.

- The fractured mandible should be splinted to the upper jaw with a figure-of-eight Barton bandage that supports the jaw below and in front.

- Open reducation and fixation may be delayed for a few days to allow stabilisation of the patient.

- Antibiotics and analgesics are administered as ancillary therapy.

- If knocked out teeth are retrieved, they may be successfully reimplanted. Preferably this should be done within the first hour (the 'golden hour'). If the fracture line intersects a knocked out tooth socket the tooth cannot be reimplanted.

- In a patient with a single knocked out tooth, the tooth should be placed into the socket and manually stabilised while the patient is en route to the dentist. Beware of aspiration of the loose tooth.

- If the knocked out tooth cannot be placed into the socket, have the patient hold the tooth in his cheek and rush him to a dentist for reimplantation. If the tooth cannot be found, a chest X-ray will determine if it has been aspirated.

- Handle the tooth only by the crown: do not touch the root as the very delicate tooth periodontal membrane may be injured, thus reducing the chance of successful reimplantation. Do not wash the tooth in tap water. Ask the patient to lick the tooth clean while gripping the tooth by the crown.

- The dentist will stabilise the reimplanted tooth with an interdental bonded splint.

- An antitetanus injection is mandatory.

Summary

With jaw fractures, rapidly progressive airway obstruction must be anticipated. Support the jaw with a figure-of-eight Barton bandage. Urgently reimplant disloged teeth within the hour.

Nasal airway obstruction

Adenoid facies - the 'long face' syndrome

The long face syndrome is a middle and lower facial skeletal developmental abnormality. The resultant chronic nasal airway obstruction and its sequelae are of concern to the otolaryngologist while the upper/lower jaw and dental maldevelopment concerns the orthodontist and dentist.

The patient appears to have a long face whether the mouth is closed or open. Furthermore, the constant open mouth makes the lower third of the face look longer than usual: hence the term 'long face syndrome'.

The ENT, dental and orthodontic sequelae described below constitute and complete the syndrome.

Rather like the proverbial chicken and theegg, it is unclear whether the shape of the face causes the syndrome or whether the syndrome affects the shape of the face. A strong familial appearance is apparent, suggesting a familial trait.

The constant mouth breathing and open mouth profile brings about a low jaw position. The latter induces abnormal muscle function of the masticatory system (ie, lips, cheeks and tongue), an aggravating factor in malocclusion in the growing child.

The salient abnormalities that concern the dentist and orthodontist are:

- Perpetual mouth breathing may cause dryness of the gums and lips, otherwise bethed in saliva. The resultant dryness may cause chronic irritation with a corresponding low grade inflammatory response of the gums. The unusually hyperaemic and oedematous gums are responsible for the red, puffy gum appearance.

- The constant dryness may alter the oral flora and may result in malodorous breath.

- The constant mouth breathing and resultant dryness of the mouth may reduce the protective salivary coverage of the teeth which may result in increased plaque formation, tooth discolouration and dental caries. The latter may render the teeth vulnerable to early loss, especially of the upper front teeth. This may affect the cosmetic appearance and function of the teeth and may have a detrimental psychological effect on the patient, resulting in a lack of self esteem.

- A short upper lip ('lazy lip') and excessive display of the upper incisor teeth makes contact with the lower lip difficult at rest and may result in lip incompetence with consequent drooling, again decreasing quality of life.

- Unopposed dental pressure between the upper and lower molars may results in over eruption of the molars (ie, they grow too long and cause an anterior open bite). Malocclusion may result due to the premature contact of the over erupted teeth. This may be responsible for a temporomandibular (TM) joint dysfunction syndrome and tenderness over the TM joint,

referred earache, facial and temporal pain, as well as a degree of trismus.

- Narrow upper arches and a high palate may be responsible for overcrowding and overlapping of the teeth, especially the front teeth, which may result in a cosmetic disfigurement.

- Protruding upper front teeth ('buck' teeth) may be induced by the hyperactivity of the lower lip attempting to obtain a lip seal during swallowing.

- The open jaw position has a receding or retrognathic (weak jaw) appearance on profile view.

ENT anomalies

- The nasal blockage may be due to enlarged adenoids and nasal allergy. The sequence of the nasal blockage may precipitate chronic sinusitis and exudative otitis media.

- Psycholinguistic deprivation may hinder scholastic achievement, intellectual development and ability to compete with peers. Antisocial behavioural patterns may emerge.

- Chronic blocked nasal airways take a toll on sleep. Frequent periods of hypopnoea and apnoea (cessatin of breathing for a brief period) interfere with sleeping patterns. Recent studies on abnormal sleeping patterns (polysomnography) reveal these patients become light sleepers with spells of night cries, night terrors, nocturnal myoclonic jerks and sleep walking. Daytime hypersomnolence results in a lack of mental alertness and may affect scholastic achievement.

- Sleep disorder studies have also revealed that antidiuretic hormone (ADH) and growth hormone (GH) are both produced during the slow-wave phase of sleep. If this sleep pattern is frequently interrupted, a lack of ADH and GH may result in enuresis and a lack of growth.

- Lower oxygen tension may lead to a relative hypoxaemia and polycythaemia with a resultant cor pulmonale; it is an absolute indication for an adenotonsillectomy.

Treatment

Ideally, prior to the commencement of any orthodontic therapy, a clear nasal airway passage should be assured. This may in itself ameliorate some of the nasal and ear pathology. In children, orthodontic therapy to correct the occlusion may, in some cases, be successful without surgical intervention.

The initial ENT approach to the management of long face syndrome in children is to establish a clear nasal airway. An adenoidectomy may be necessary and the child may need to be placed on an **allergy protocol** (ie, identification of the allergen and consequent avoidance), and topical intranasal therapy with steroids or antihistamines.

Surgical management of redundant allergic tissue over the inferior turbinates is a last resort in recalcitrant cases. Ventilation tubes may be necessary for associated exudative otitis media if medical management is unsuccessful.

Early identification of the long face syndrome will certainly add to cost-effective, successful treatment. This should be seen as an investment in the cosmetic and functional rehabilitation, as well as the scholastic achievement of the paediatric patient.

In the older patient whose facial bones are already fused, surgical intervention may be necessary to correct some of the deformities. Regular biannual dental hygiene by a dental hygienist, in collaboration with a dentist, may minimise tooth decay.

Seasonal rhinitis (intermittent allergic rhinitis)

Allergic rhinits or hayfever is IgE (immunoglobulin E) mediated and is seasonal. It is a type I immune response (ie, immediate response) caused by contact with pollen and affects a significant percentage of children in South Africa.

Hayfever is a misnomer: hay may be involved as an allergen but fever is not involved. Many hayfever patients may also suffer from asthma. Treating and controlling allergic rhinitis in children may prevent the onset of asthma in later life. Hayfever has become more common in recent years as has asthma.

Symptoms of seasonal allergic rhinitis and conjunctivitis consist of an itching and runny nose, bouts of sneezing, blocked nose and constant sniffing, as well as itchy and runny eyes commonly referred to as 'runners' (ie, an acute histamine release). The congested and blocked nose causes forced nocturnal mouth breathing, dry mouth and pharyngitis sicca. In severe cases the blocked nose may cause anosmia.

The interaction of allergens with the IgE on the surface of the mast cells (a granulocyte derivative) in the nasal mucous membrane leads to the release of various vasoactive, chemotactic and enzymatic mediators resulting in vasodilatation, oedema, mucosal permeability and cellular recruitment. This rapid response can be reversed by antihistamines or prevented by prior use of sodium cromoglycate: corticosteroids do **not** inhibit the rapid response phase.

Perennial rhinitis (persistent allergic rhinitis)

Perennial rhinitis is IgE mediated. It is a year round type I immune response caused by the house dust mite, fungal spores or animal dander. High humidity may increase fungal spores and mould counts and add to the symptoms.

Although caused by the house dust mite and danders from domestic animals (eg, birds, dogs and cats), the symptoms may be exacerbated by pollen during the pollen season. The perennial allergens are concentrated in furniture stuffing, feather pillows, eiderdowns, carpets and curtains.

Symptoms of perennial rhinitis are prolonged periods of blocked, stuffy nose that may be more pronounced compared to seasonal rhinitis: commonly referred to as 'blockers' (ie, an inflammatory response). Children push the tip of the nose upwards to counteract the nasal itchiness causing a crease in the supra tip area, the so-called 'allergic salute'. They may develop nasal speech and loss of smell and taste senses, headaches and disturbed sleep. They often wake at night for a drink. Child sufferers often develop exudative otitis media.

Treatment

In allergic rhinitis and conjunctivitis the nose and eyes are sensitised by a progressive inflammatory response that may cause blocked nasal airways. Irritants such as cigarette smoke, chalk dust, chlorinated swimming-pool water, pollen, strong odours (ie, perfumes), detergents and aerosols may trigger an attack of allergic rhinitis and conjunctivitis. These patients respond well to topical nasal steroids, less dramatically to antihistamines and sodium cromoglycate. Various surgical procedures may alleviate the blocked nasal airways in recalcitrant patients.

Tips for allergic rhinitis sufferers

- Sleep with closed windows to prevent pollen and mould entering the bedroom and allergens in the room from being disturbed.

- Travel with closed car windows.

- Avoid mowing lawns.

- Mattresses, carpets and curtains should be regularly beaten, vacuum cleaned and exposed to the sun to reduce the house dust mite count, as well as to reduce their feeding substrate (ie, shedded skin products).

- Desensitisation to the house duct mite has a limited effect. Notwithstanding the desensitisation therapy, the allergic symptoms may persist due to overwhelming house dust mite counts. Avoiding the house dust mite is the maintstay of perennial allergy treatment.

- Topical nasal decongestant sprays (oxy- and xylometazoline, pseudoephedrine and ephedrine) may only be used for short periods of two weeks. A rebound phenomenon may appear leading to the need for more nose sprays at shorter intervals to obtain the same decongestant effect. For hayfever or intermittent symptoms, long acting non sedatory antihistamines may curtail the nasal as well as the eye symptoms. Topical steroid sprays remain first line treatment to minimise perennial or persistent congestion. Recently developed antihistamines have both an antihistamine and an anti-inflamatory effect on nasal allergy.

Food and drug allergy

Hayfever symptoms may be triggered by certain foods (eg, peanuts and dairy products), NSAIDs, penicillin and aspirin. Avoiding these products is a prudent prophylaxis. Food allergy is most prevalent in the first three years of life.

Non-allergic vasomotor rhinitis (NAVMR)

Non-allergic vasomotor rhinitis is precipitated by an imbalance of the autonomic nervous system and is not mediated by IgE (ie, IgE independent or pseudo-allergic) reactions. It can coexist with allergic rhinitis.

Vasomotor rhinitis presents as swollen nasal mucosa, especially over the inferior turbinates. This may lead to increased nasal airway resistance, blocked nose as well as sinusitis. Forced mouth breathing may predispose to recurrent sinusitis and secondary pharyngitis.

Swollen nasal mucosa may account for the anosmia and hyposmia, as well as the watery nasal discharge. The accompanying itchy nose and palate may be due to varying degrees of turbinate engorgement. Causes may include:

- Psychological conditions such as stress.

- Hormonal fluctuations during pregnancy or when taking oral contraceptives and even during sexual arousal.

- Therapeutic drugs like antihypertensives and aspirin.

- Odours, fumes, air pollution and cigarette smoke.

- Topical nasal mucosal irritants, especially nose drop abuse resulting in rhinitis medicamentosa.

- Change in temperature (too hot, cold, dry or wet).

- Alcohol consumption.

- Nasal polyps, septal deviations and adenoidal hypertrophy in children.

Patient evaluation

An accurate history may determine to a large extent the aetiology, which special tests are required, therapy and the ultimate prognosis.

To differentiate between allergic and non-allergic vasomotor rhinitis, the following tests may be of help in the diagnosis:

- Cytology: A nasal smear of the watery nasal discharge may reveal an overwhelming eosinophilic pattern of inflammatory cells.

- Serology: Immunoglobulins may be elevated in an allergic patient (rule out intestinal parasites which may also be responsible for the elevated IgE titres).

- Skin scratch testing for specific allergens: These may precipitate an acute allergy attack and should be carried out in a situation where one is prepared to deal with anaphylaxis.

- The RAST test is an in vitro test that may confirm the specific allergen involved and is especially valuable with inhalants. It may avoid the complications associated with skin testing.

Rule out concomitant purulent sinusitis, a deviated nasal septum, enlarged adenoids or sinus and nasal polyps.

Treatment

Treatment for non-allergic vasomotor rhinitis is tailored to the aetiology and may require surgical management. With irritants, avoidance is the key concept.

Blocked nose in pregnancy

- Blocked nose in pregnancy: A short course of topical intranasal steroid therapy may be used during the latter stage of pregnancy with the knowledge of the patient's obstetrician. Nasal symptoms usually resolve after pregnancy. If there is no improvement within three months of the infant's birth, anatomical causes of the blocked nose may be treated surgically.

- Non-allergic vasomotor rhinitis following antihypertensive therapy: Switching to another therapeutic agent may ameliorate this condition. If there is no improvement, surgical management of anatomical abnormalities may be needed.

- Rhinitis medicamentosa: These patients are usually psychologically addicted to drops and nasal sprays. Surgical management of anatomic abnormalities may be necessary for lasting success, but it is preferable that this occurs only once the topical medication effects have resolved. More appropriate medication for the mucosal swelling should be used, such as non-sedatory long acting antihistamines in combination with topical steroid nasal sprays or systemic decongestants.

- Ipratropium bromide nasal spray may be effective in controlling symptoms.

Although allergic and non-allergic vasomotor rhinitis may have similar clinical presentations, the aetiology of each is distinctly different. An accurate history and allergic work-up will determine the treatment modality.

Nasal polyps

A pedunculated inflammatory oedematous degeneration of the nasal respiratory mucosa is called a polyp. Polyps may occur in small numbers or as diffuse polyposis. They originate from the nasal mucosa of the ethmoid sinuses and middle turbinates.

The aetiology may be linked to vasomotor instability and nasal allergy. Asthma and aspirin intolerance are not uncommon in these patients. Peak age incidence is between 40 and 60 years.

The clinical appearance is that of a peeled grape. On exposed surfaces the polyps become darkened. The polyps may rarely be single and present as an antrochoanal polyp in the nasopharynx. This should not be confused with a meningocele.

The patients complain of a constantly blocked nose, headaches, mouth breathing and relative anosmia. CT studies may confirm sinusitis and antral and/or nasal polyps.

Treatment

Surgically restore the patency of the nasal airways and drain infected sinuses. Afterwards histological confirmation may exclude tumours such as an inverting papilloma. The polyps may be surgically removed. Antibiotics, oral steroids or topical steroids may be needed postoperatively.

Acute sinusitis

All acute purulent sinus infections (ie, maxillary sinusitis, ethmoiditis and frontal sinusitis) have the potential for **intracranial complications** such as cavernous sinus thrombosis, intracranial abscess and meningitis or intra-orbital complications. The patient with acute sinusitis must be monitored for these symptoms.

If there is deterioration, or if significant improvement is not seenwithin 48 hours of antibiotic therapy, surgical intervention must be considered. If any doubt is present the patient should be hospitalised and placed on intravenous antibiotics. The classic clinical signs of purulent sinusitis (ie, specific sinus tenderness on palpation and headaches) are often absent. More reliable signs of sinusitis are inflamed nasal mucosa on rhinoscopy, and sometimes pus draining from ostia. Sinus transillumination, sinus ultrasound or radiology may help confirm the diagnosis. CT is much more reliable than standard X-ray views. The vast majority of patients with AIDS have purulent sinusitis.

Pathophysiology

Swelling of the mucous membrane lining of the nasal airways and sinuses may cause blockage of the natural drainage ostia. The oedematous swelling of the lining may be due to a viral upper respiratory tract infection (ie, rhinosinusitis) or upper airway allergy.

Oxygen absorbed via the mucous membranes creates a negative pressure in the sinus cavities. This negative pressure is counteracted by fluid extravasation in the cavities. The negative pressure also sucks contaminated material from the nose into the sinus cavity that is simultaneously filled with fluid. This fluid is a good growth medium for bacteria and is quickly changed into pus with the sequelae of an acute infection.

Bacteriology

Acute sinusitis is commonly produced by *Streptococcus pneumoniae, Pneumococcus* or *Haemophilus influenzae.* Less common are *Branhamella catarrhalis, Streptococcus pyogenes* and *Staphylococcus aureus.*

In chronic sinusitis, strains of *Enterobacteriaceae, Pseudomonas aeruginosa* and anaerobes may be encountered.

Treatment

While acute purulent sinusitis may be self limiting, antibiotic therapy speeds up recovery and prevents mucosal metaplasia of chronic disease. Most cases of acute sinusitis are due to non-betalactamase producing organisms and will respond well to antibiotics/antimicrobial therapy. The addition of metronidazole or the use of amoxycillin/clavulanic acid and fluoroquinolones should be considered in chronic sinusitis with anaerobic infections.

In cases where bacterial strains are resistant to all of the above antibiotics, specialist consultation should be sought as a matter of urgency. The duration of antibiotic treatment varies with the severity of the sinusitis, but up to 10-14 days is commonly recommended. The dosage should be higher than that used for soft tissue infections.

Maxillary sinusitis

The maxillary sinuses are present at birth. Maxillary sinusitis is equally common in adults as in children. The sinusitis is usually preceded by an upper respiratory tract infection or nasal allergy. If unilateral sinusitis is present, a dental abscess with erosion into the sinus must be excluded. In children there is a risk of osteomyelitis.

Symptoms

The patient may complain of local tenderness, pain on palpation, and aching or sensitive upper molars. Headaches may be referred to the supraorbital and/or the temporal region. Facial swelling is more commonly due to dental complications, such as a tooth root abscess.

Signs

Examination may reveal a purulent nasal discharge and toxic or feverish patient. Radiology, particularly on the Waters' view, may show an opaque antrum or an air fluid level in the sinus. For detailed information of the sinus ostia, a sinus CT scan is preferable.

Treatment

Administer systemic antibiotics, decongestants and analgesics as well as steam and topical mucolytic inhalants. If medical therapy fails, surgical drainage is indicated.

Functional endoscopic sinus surgery (FESS) is the current trend to accurately restoring sinus physiology.

Ethmoiditis

The ethmoid sinuses are present at birth. Ethmoid sinusitis is common in children. There is the potential for adjacent orbital complications to occur; inflammation of the ethmoid mucosa may lead to infection in any of the other paranasal sinus groups.

Symptoms

The patient may complain of tenderness and pain over the medial canthal area.

Signs

Examination may reveal a purulent nasal discharge. If the infection extends to the orbit, swollen eyelids, restricted eye movement or visual disturbances may occur. Standard radiology is unreliable and CT has taken over as the most reliable radiological diagnosis.

Treatment

Administer antibiotics, analgesics, decongestants and humidification. If medical therapy fails within 48 hours, surgery is indicated. Orbital complications may lead to blindness and require expert monitoring and possible emergency surgery.

Frontal sinusitis

The frontal sinusitis develop from the anterior ethmoids at the age of six to eight years. Fortunately, acute frontal sinusitis is rare in South Africa. It may be complicated by osteomyelitis of the skull with intra- and extracranial extension, the so-called 'Pott's puffy tumour'; this is a surgical emergency.

Barotrauma to the middle ear and sinuses

Barotrauma often occurs with rapid barometric change, for instance when diving or flying in non-pressurised aeroplanes. More subtle changes occur in commercial aeroplanes which are usually pressurised when cruising at high altitudes. The most pronounced effects are on the middle ear, nose, sinuses and upper molars.

Predisposing factors are swollen nasal mucosa, as with an upper respiratory infection, allergy, vasomotor rhinitis, polyps, or a deviated nasal septum.

The pathophysiology of barotrauma is based on the fact that as barometric pressure decreases, air volume increases (Boyle's law) hence gas containing cavities (eg, sinuses, ears and in rare instances the teeth) may be affected. The resultant gas pressure causes soft tissue distortion (ie, vascular engorgement, extravasation of fluid into the adjacent soft tissue, petechiae), and ultimately haemorrhage. Pain is induced because of the stretching of the tympanic membrane and distortion of the mucosal lining of the nose and sinuses.

Serous or blood stained fluid accumulation may occur in the middle ear and sinus cavities to compensate for the loss of gas volume.

The vast majority of barotraumatic dental pain is referred pain from the sinuses to the upper teeth in relation to the maxillary sinuses. True barodentalgia is rare and occurs when air is trapped underneath a filling. It occurs during ascent and the tooth may remain very painful throughout the flight until descent.

Symptoms

The air passenger or diver may experience acute and severe facial pain, earache and dental pain (barodentalgia), especially of the upper premolars and the molars. The patient may also experience a moderate degree of deafness due to haemotympanum. Acute barovertigo may also occur due to inner membrane rupture of the oval or round window membranes. Less severe and short lasting barovertigo may be due to unequal Eustachian tube function resulting in unequal middle ear pressures.

Signs

- Epistaxis.

- Bleeding from the ears indicates a tympanic membrane rupture and moderate conductive hearing loss may be present.

- In divers, facial nerve palsy or palsy of any other cranial nerve may be present.

Treatment for air passengers

- Air passengers on commercial airlines are not exposed to drastic barometric changes. Antihistamines/decongestants may be of value to prevent barotitis and barosinusitis.

- Analgesics may relieve the initial pain of barotrauma.

- The Valsalva manoeuvre (popping the ears while pinching the nose with the forefingers) should be encouraged during descent.

- During descent infants should be sitting upright and swallowing should be encouraged by breastfeeding or bottle feeding to simulate the Valsalva manoeuvre.

- Tympanic membrane perforations are usually small and heal spontaneously. Ear drops are not necessary. Preclude water from the ears until final healing is confirmed.

- For recurrent attacks of barosinusitis, anatomic nasal and sinus abnormalities should be surgically corrected.

- Recurrent barodentalgia secondary to barosinusitis (the vast majority of case) may necessitate no other treatment than nasal decongestants (ie, nose drops) or systemic antihistamines/decongestants.

Treatment for divers

- Divers are exposed to greater barometric changes than air passengers. Those with upper respiratory infection should not dive. Decongestants and antihistamines may not prevent barometric complications (eg, barosinusitis or barotitis).

- Divers should be strongly discouraged from smoking.

- If barotrauma has occurred, rest for 14 days before risking further barotrauma. Recurrent episodes may occur due to insufficient recovery from the barotrauma, predisposing and aggravating subsequent barotraumas. If barotrauma or decompression sickness is suspected, emergency hyperbaric oxygen therapy and facilities are available in South Africa at Diver's Accident Network (DAN) on Tel: 0800-020111 or 012-333-6000 x 5823. In most major centres in South Africa hyperbaric oxygen chambers are available with professional back-up staff.

- **The state of the nose is of paramount importance for safe and enjoyable scuba diving.** Any case of congestion should be corrected as a long term investment for the diver, whether professional or amateur. Causes of nasal congestion are nasal allergy, deviated nasal septum, nasal polyps and the common cold. All these may be successfully treated with medications or with surgical management.

Alleviation of nasal congestion will enhance nasal breathing and should improve Eustachian tube function to equalise pressure differentials in the middle ear cavity.

- Barotrauma should not be confused with decompression sickness where divers are exposed to too rapid decompression, resulting in air embolus formation in the blood and tissues.

Habitual snoring

Snoring is an annoying and often embarassing complaint, seen especially in the older male patient. It is a problem that affects not only the snorer but also the partner.

Although many patients may snore loudly, the partner may become accustomed to it over the years as the couple grow older together! But it can be embarrassing problem in a second marriage or when staying with others.

Sleep deprivation may afflict both the snorer and the partner. Sleep disturbance usually occurs during the early morning hours and deprives the snorer of normal REM sleep. The snorer may also suffer from periods of apnoea that may lead to relative hypoxia causing polycythaemia and early signs of cor pulmonale.

The basic pathophysiology is muscle relaxation of the intrinsic tongue and soft palate muscles. The muscle relaxation may be exacerbated by muscle relaxants, tranquilisers, alcohol, posture and obesity. Nasal airway obstruction may also be a significant contributing factor as may mucosal swelling secondary to infections or gastro-esophageal reflux (GORD).

Treatment

- Explain to both parties (ie, the snorer and the partner) the basic pathophysiology of snoring. They should avoid alcohol or drugs that may exacerbate the snoring.

- Tape record the snoring for prognostic purposes.

- Correct the sleeping position; the snorer should sleep on the side rather than the back.

- Weight control is important, although easier said than done! Sleep apnoea induces increased eating during the day in an attempt to stay awake, while increased weight may cause sleep apnoea. Treating the sleep apnoea often results in weight loss.

- A jab in the side or in the ribs of the snorer may stimulate light sleep and may reduce the snoring to an acceptable level.

- Nasal airway surgery to clear any obstruction present may aid in a large percentage of cases.

- Tonsillectomy may be performed if the tonsils are present. The subsequent scar tissue formation may help to 'stiffen' the soft palate.

- Specific surgical management of snoring (ie, uvulopalatopharyngoplasty (UPPP), works well in selected cases may have temporary or permanent catastrophic results (ie, regurgitation and hypernasal speech). The aim of the surgery is to shorten and stiffen the soft palate and this may cause a velopharyngeal incompetence if incorrectly indicated or too vigorously performed.

- Continuous positive airway pressure (CPAP), where a positive pressure in the upper airway is maintained to prevent soft tissue collapse, is most successful in selected patients. The device, however, is expensive and uncomfortable to wear while sleeping.

The 'Danger Triangle'

Emissary veins connect the venous sinuses inside the skull with the veins on the scalp. They have no valves and blood flows freely between the extracranial and intracranial vessels. There are various emissary veins (ie, mastoid, parietal, occipital or base of skull). The superior and inferior ophthalmic veins are also potentially emissary since they connect intracranial to extracranial veins.

Extracranial infection may potentially spread via this pathway to the intracranial venous sinuses. The triangular area formed by the eyes and the nose collectively drains into the ophthalmic veins. Infection from skin conditions (ie, nasal or skin furuncles), a septal abscess or infected facionasal fractures may pour infected blood into the ophthalmic drainage system resulting in intracranial thrombophlebitis of the cavernous sinus. Thrombophlebitis may lead to cavernous sinus thrombosis and meningitis which are potentially fatal.

The patient may present initially with a mild swinging temperature and only appear mildly toxic. This may be deceptive as to the severity of the condition.

Differential diagnosis must be made between acute meningitis, a brain abscess and intracranial thrombophlebitis. The table below offers characteristics of these conditions. MRI scans and ophthalmic vascular studies are necessary to confirm the diagnosis.

Treatment

The patient presenting with suspected intracranial thrombophlebitis requires:

- Hospitalisation.

- Intravenous antibiotics.

- Monitoring for CNS extension and for the need for a lumbar puncture.

- MRI studies.

- Possible anticoagulant therapy.

Differential diagnosis betweem meningitis, brain abscess and an intracranial thrombophlebitis may dictate the correct course of action and therapy

	Meningitis	**Brain abscess**	**Cavernous sinus thrombosis**
Onset	acute	subacute	subacute
Mening. irritation	severe	rare	none
Focal signs	none	present	none
CSF pressure	raised	normal	normal
CSF protein	raised	slightly	normal
Papilledema	slight	absent	marked
CSF cells	marked	rare	rare
Systemic response	marked	rare	mild
CSF organisms	marked	none	none.

Tumours

Inverting papilloma

This locally malignant growth must be distinguished from allergic nasal polyps. Papillomas tend to recur, have the ability to destroy bone and may transform into squamous cell carcinoma.

The papillomas originate from the mucosa at the junction of the maxillary and ethmoidal sinuses in the lateral wall of the nasal cavity and may invade all paranasal sinuses, the orbit and the intracranial vault. The aetiology is thought to be the human papilloma virus

(HPV).

Radical surgery is necessary to ensure that the growth is totally removed. Untreated patients eventually die from intracranial extension. Irradiation is not indicated.

Malignant

Juvenile nasopharyngeal angiofibroma

Nasopharyngeal angiofibroma is a type of hamartoma affecting predominantly adolescent males in the peripubital period. It is potentially fatal if untreated. On external carotid angiography it has typical appearance. It is extremely vascular and the patient may have a fatal haemorrhage from an ill-considered biopsy or adenoidectomy.

The tumour may regress in some cases. The tumour originates from the junction of the nose and postnasal space and is fed by the sphenopalatine artery. The tumour is locally aggressive and invades the bony cranium to reach the middle cranial fossa.

CT scans and MRI studies confirm the extent of bony destruction and intracranial invasion. Surgical removal is the treatment of choice.

Carcinoma of the external nose

Solar rays may contact the skin at right angles causing maximal solar damage to the skin surface. Carcinoma of the nasal tip may occur simultaneously with skin malignancies on the cheek and the helix of the pinna (the 'sun exposure line'). Timeous surgical removal has excellent results.

Carcinoma of the nasal cavity and paranasal sinuses

The majority of cancers in this area are squamous cell carcinomas; the remainder are melanomas and adenocarcinomas or other minor salivary gland tumours of all the sinuses. The maxillary sinus is most commonly affected. Older males are predominantly affected. Black South Africans have the highest incidence of carcinoma of the maxilla in the world.

Occupational exposure to leather tanneries, furniture manufacturing (ie, wood dust), nickel-plating fumes as well as tobacco exposure and traditional snuff may be implicated in the aetiology.

Clinical findings

Sinus and nasal tumours may be asymptomatic until they reach a stage beyond the confines of the sinuses. Common symptoms are subtle at first (ie, dull toothache, facial pain, epistaxis, nasal airway obstruction, proptosis and diplopia). A high index of suspicion is essential. As the tumour expands, cheek and lip anaesthesia may result with exophthalmos (proptosis) diplopia and cranial nerve deficits. Cervical adenopathy is a late finding.

CT scans may show bone involvement better than MRI for planning treatment.

Treatment

Surgery is the mainstay of therapy and may be supplemented with irradiation. Chemotherapy has a limited role in solid head and neck tumours. The greatest threat is intracranial spread.

Rhinophyma

Rhinophyma is a gross chronic hypertrophy of sebaceous glands on the nasal tip in older males. The disfigurement may be surgically corrected with good results. Care must be taken not to injure the underlying nasal tip cartilages. Rhinophyma is not a tumour of the nasal tip; however, it may cause a cosmetic disfigurement. It is a late stage of acne rosacea. A CO_2 laser is useful in excision of the rhinophyma.

Acne rosacea is a chronic facial dermatitis with four characteristic components:

- A chronic inflammatory dermal response.

- A concomitant vascular flush.

- Sebaceous gland hypertrophy.

- Telangiectasis may be present in the perinasal area. Facial scarring may simulate acne scars.

The severity of the disease may wax and wane. Certain conditions are known to aggravate rosacea in some patients (ie, hot drinks like coffee and tea, spicy foods, alcoholic beverages, extreme heat and cold, excessive sun exposure and stress).

The aetiology is uncertain. Commensal skin organisms have been identified although not proven to be an aetiologic factor.

1. Epistaxis is mostly arterial in origin from Kiesselbach's plexus in Little's area. A number of patients may bleed from the nasal vault, especially those with associated hypertension, atherosclerosis and the use of anticoagulants. Idiopathic or spontaneous epistaxis occurs predominantly from the caudal end of the nasal septum (ie, Little's area or Kiesselbach's plexus). In younger patients the bleeding may be venous, in older patients the bleeding may be arterial.

2. ENT sports injuries. Mid-facial fractures are commonly seen as a result of flying missiles (eg, golf balls). Concomitant eye injuries must be excluded. Squash ball injuries may injure the globe rather than the bony orbit. Protective eyeglasses may prevent the catastrophe of blindness. Delayed complications may include glaucoma, cataract formation and retinal detachment.

3. A boxer's nose is a post traumatic collapse of the supranasal tip area. An infected septal haematoma or abscess may cause absorption of the nasal septal cartilage. The dorsal nasal dip is caused by collapse of the supporting dorsal cartilaginous nasal septum.

4. Mandibular fractures. Direct blows are common causes of mandibular fractures. Handle dislodged teeth by the crown in order not to damage the delicate peridontium. The dislodged tooth may be successfully reimplanted.

5. Adenoid facies - the long face syndrome. Chronically blocked nasal airways and mouth breathing may be aggravating factors in the long face syndrome and associoated ear, nose and throat sequelae (eg, glue ear, chronic sinusitis and pharyngitis sicca). 1) Maxilllary osteum. 2) Pus indicating chronic sinusitis. 3) Allergic rhinitis aggravating the nasal airway obstruction. 4) Enlarged adenoids cause nasal airway obstruction. 5) Eustachian tube dysfunction. 6) High palate. 7) Depressed tongue. 8) Retrognathic jaw. 9) Chronic mouth breathing.

6. Seasonal rhinitis is a hypersensitivity of the nasal mucosa to certain provoking agents that may be allergic or non-allergic. Seasonal rhiitis results in swollen, reddened nasal mucosa especially over the inferior turbinate compromising the nasal airways. Multiple nasal polyps may be present regardless of the aetiology (ie, in allergic or non-allergic patients). The right side is a comparatively normal side without seasonal rhinitis being present with a patent nasal airway.

7. Allergic rhinitis and conjunctivitis. The house dust mite, fungal spores and domestic animal dander are the main allergens responsible for persistent or perennial rhinitis and conjunctivitis. Environmental control is important to minimise the allergic response in sensitised patients (ie, reduce the house dust mite concentration).

8. Scratch testing for allergic antigens. For many years this test has been the mainstay used to identify an allergic antigen causing the patient's allergy, but it may now be supplemented by serological tests such as RAST. Scratch testing may induce a severe allergic response with asthma and anaphylactic shock and should only be performed in doctors' rooms where emergency facilities are at hand.

9. Food allergy: Peanut allergy is on the increase in South Africa. Based on current evidence it would appear that the infant is sensitised *in utero* by the maternal diet. A diet rich in peanuts and breakfast cereals that contain peanuts should be avoided during pregnancy. Avoidance is the mainstay of food allergy treatment; patients cannot be desensitised against food allergies.

10. Rhinitis medicamentosa: Nose drops may result in rhinitis medicamentosa and a constantly blocked nose. The patient may be dependent on the nose drops.

11. Multiple nasal polyps: May be present regardless of the aetiology. An antrochoanal polyp may obstruct the nose and nasopharynx. It may present in the oropharynx.

12. Periorbital oedema is most commonly from an acute tooth root abscess or ethmoidal sinusitis. Unilateral sinusitis suggests a dental infection with erosion into the maxillary sinus. A tooth root may project into a maxillary sinus causing toothache. Many a tooth has been unnecessarily extracted.

13. Dental abscess: Overlying soft tissue inflammatory response may cause periorbital soft tissue oedema. In the so-called 'shallow' sinus (1) the tooth root does not project into the maxillary antrum. In the 'deep' sinus (2) the tooth root projects into the maxillary antrum only covered by a thin layer of mucous membrane and may predispose to unilateral sinusitis.

14. Divers are exposed to harsh barometric changes. Those with an upper respiratory infection should not dive. Decongestants and antihistamines may not prevent barometric complications (eg, barosinusitis or barotitis).

15. Habitual snoring. Snoring may be exacerbated by alcohol consumption and hypnotics taken before bedtime, and by sleeping in the supine position. Sleep deprivation may occur during the early morning hours and may deprive both the snorer and partner of normal REM sleep. The snorer may suffer from periods of sleep apnoea up to 40 seconds. Relative hypoxia may be responsible for polycythaemia that may lead to early signs of cor pulmonale in a marginally compromised cardiovascular patient. Sleep studies (polysomnograms) may confirm the diagnosis. This condition may be managed surgically.

16. Danger triangle. Infection from skin conditions, ie, furuncles, a septal abscess or infected facionasal fractures, may pour infected blood into the ophthalmic drainage system resulting in intracranial thrombophlebitis of the sagittal sinus. Extracranial infection may spread retrograde intracranially via an infected emissary vein connecting the nasal angular vein to the anterior end of the sagittal sinus.

17. Inverting papillomas. Radical surgery is necessary to ensure total removal. Recurrences may be associated with malignant transformation (ie, squamous cell carcinoma). A high percentage of recurrences may occur if surgery is inadequate. An inverting papilloma may initially be mistaken for an allergic nasal polyp. However, an inverting papilloma is more often unilateral than bilateral. Clinically these tumours are locally malignant with bony destruction and local soft tissue infiltration.

18. Juvenile nasopharyngeal angiofibroma. Juvenile nasopharyngeal angiofibroma is limited to adolescent males. It is a locally aggressive infiltrating tumour. The patient may present with a blocked nose and recurrent epistaxis. A CT scan may reveal the bony destruction and extent of the infiltration. A biopsy is contraindicated due to its high vascularity.

19. Intranasal carcinoma. An intranasal carcinoma may remain asymptomatic for long periods before the pathology is diagnosed. These tumours are close to the orbit and infraorbital nerve infiltration may be present upon diagnosis. Because of the proximity of the ethmoid and maxilla to the orbit, their contents may be involved at the patient's first presentation. While the resection (exenteration) of one eye has great emotional impact, the cosmetic and functional defects should not stand in the way of curative surgery. Certain cultures would rather die than lose a non-functional eye. Maxillary sinus carcinomas may remain dormant for long periods of time. Orbital involvement may be present before the diagnosis is established.

20. Rhinophyma. Rhinophyma is a groos chronic hypertrophy of sebaceous glands on the nasal tip in older males. The disfigurement may be surgically corrected with good

results. Rhinophyma is not a tumour of the nasal tip, it is a late stage of acne rosacea.

Throat

Lips

Cleft lip and palate

The upper lip may be affected on one or both sides in the paramedian position (a 'harelip'). The cleft may penetrate partially or completely and may extend from the upper lip in the paramedian direction to the nose. If the cleft reaches the nostrils the palate may also be involved, resulting in a concomitant cleft palate (oronasal cleft). Rarely cleft lips may extend in the direction of the ear (ie, oro-aural clefts).

Treatment

Surgical repair of cleft lip and palate should be delayed to ten weeks after birth (ie, 10 lbs or 4.5 kg weight and a Hb of 10 g/dL). Repair one side at a time leaving a four month interval between operations. An experienced team (ie, ENT surgeon, paediatrician, anaesthetist, and orofacialmaxillary surgeon or plastic surgeon) is essential for success. Speech therapy is a vital part of rehabilitation. The patient should be monitored for signs of otitis media and serous otitis media. Grommets may be necessary to restore hearing and to promote middle ear ventilation and drainage.

Velopharyngeal incompetence may be corrected with a combination of surgery and an obturator placement in the oral cavity. Orthodontic therapy for tooth alignment may be indicated.

Adenoidectomy in a patient with a repaired palatal cleft or a submucosal cleft may cause nasopharyngeal incompetence and should only be undertaken if the indications are very strong.

Aesthetic corrections (ie, cosmetic rhinoplasty and correction of nasal airway obstructions), may be performed after the nasal bones and nasal cartilages are fully grown (ie, after 18 years old for boys and after 16 years old for girls).

Pits

Mucosal lined sinus tracts may present as paired symmetrical pits on the lower lip at the skin/mucosal junction. They may extend vertically into the lower lip to a depth of 2.5 cm as measured with a probe. A mucoid secretion may be present. Surgical excision of the sinus tracts has good results.

Double upper lip

Double upper lip when smiling is the result of redundant mucosa. The latter may be removed for cosmetic reasons.

Lip trauma

Lip lacerations may occur during sports altercations. Primary suturing may have excellent results. If the wound is contaminated with dirt an antitetanus booster is advisable.

In the case of lip avulstions, small avulsions may be primarily sutured. Larger avulsions may require restoration of blood circulation by microvascular surgery.

Dog and human bites may require rabies and HIV precautions respectively.

Tongue

The tongue may be divided into two compartments; the base (ie, in the oropharynx which extends to the circumvalate papillae) and the mobile portion.

Infections

Hairy tongue

A hairy white or a hairy black tongue are harmless conditions in which the papillae become elongated and hyperkeratotic. The tips of the hyperkeratotic papillae may become pigmented with various colours ranging from white to black due to chromogenic bacteria or nicotine stains. Besides the cosmetic dismay, there may be some degree of discomfort and halitosis. This disorder may folow a course of antibiotic therapy.

Treatment

The elongated papillae may be scraped off. Topical lidocaine ointment may lessen the discomfort. The scrapings should be sent for microscopic, culture and sensitivity studies. Smoking should be discouraged as well as consumption of highly coloured foods. Antifungal oral gel application may assist in ameliorating this condition.

Granuloma

A granuloma of the tongue may simulate a malignancy. The aetiology may be a pyogenic granuloma or a TB granuloma aggravated by chronic irritation (ie, a sharp tooth edge or a rough tooth filling). A punch biopsy may reveal the aetiology of the granuloma. Lung X-rays may confirm or rule out the presence of TB.

Fungal infections

Candida albicans and *Candida nigra* - oral thrush - may follow a prolonged course of antibiotic therapy. The mouth may be diffusely inflamed and painful; scattered patches of fungal colonies may be seen on the tongue and oral mucosa. Topical antifungals may successfully eradicate the infection.

Trauma

Tongue lacerations may occur during sports activities or may result from a motor accident. Lacerations on the side of the tongue may have been caused by the patient biting his own tongue. Primary suturing has excellent results.

Congenital/hereditary abnormalities

Geographic tongue

Geographic tongue (migratory glossitis) often occurs in older females. It consists of oval or rounded tender areas, superficially denuded of papillae, measuring about 1-1.5 cm in diameter. Topical steroid cream mixed with a topical anaesthetic cream may minimise the pain. Spontaneous healing may occur within a few days. This has minimal known clinical significance.

Scrotal tongue

Scrotal (fissured) tongue is an impression on the tongue surface that simulates the scrotal skin (ie, peaks and troughs). The troughs are deepithelised and may be ulcerated, causing discomfort. Aetiology is uncertain and the condition usually heals spontaneously. Topical steroid cream mixed with a topical anaesthetic cream may subdue the discomfort and hasten healing.

Angioedema

Angioedema is an allergic response to bee or wasp stings, antibiotic allergy and certain seafood allergies (ie, shell fist such as molluscs and crustaceans). In addition milk, eggs, nuts, strawberries or spices may cause a similar reaction. In the oral cavity a rapidly progressive oedema of the tongue, lips, hypopharynx and larynx may ensue. A widespread itchy urticarial rash may appear all over the body.

This is a medical emergency. Intravenous steroids should be administered and the patient placed under observation for airway monitoring. Subcutaneous or inhalation adrenaline may be necessary in severe cases with anaphylactic shock and a fall in blood pressure. Intubation or tracheostomy may be necessary.

Angiomatosis of the tongue

Angiomatosis of the tongue may be due to a congenital haemangioma or a lymphangioma either in the tongue musculature or superficially on the tongue surface. Management is difficult, as it is diffuse throughout the tongue musculature. Tongue resurfacing using a CO_2 laser may reduce the tongue to a functional size.

Macroglossia

Macroglossia may be idiopathyic (ie, mongolism) or the tongue matrix may be expanded due to an intrinsic haemangioma or a lymphangioma. In selected cases bulk

reduction can be carried out.

Tumours

Benign

Lymphangioma, haemangioma and cystic hygroma

Lymphangioma, haemangioma and cystic hygroma are benign lesions and may vary in size from a few millimetres to a massive enlargement of the tongue (macroglossia). A spectrum of surgical techniques is available depending on the degree of the defect.

Leukoplakia

Leukoplakia (erythroplakia) may vary in size from a few millimetres to a few centimetres in diameter. Treatment is primary excision and biopsy with a skin graft if the wound cannot be primarily closed. A small percentage of excision biopsies may reveal a carcinoma *in situ;* this makes leukoplakia a premalignant condition.

Midline thyroid (ectopic thyroid)

This condition appears exclusively in females. The lingual thyroid may vary in size and may be the only thyroid tissue present. Adequate thyroid hormone levels are essential for proper decision making. Removal of this source of thyroid hormone may result in hypothyroidism. A thyroid isotope neck scan is essential to confirm the suspicion of an absent neck thyroid gland. The mass is situated on the border of the oral tongue and the tongue base. If partial or total surgical removal is indicated, it may be done via an oral approach.

Ranula

Ranula is a unilateral mucocele that originates as an occluded sublingual gland. It may displace the tongue into the oral cavity compromising the oral airway. Surgical removal is indicated. Revision surgery is difficult.

Carcinoma

Upper aerodigestive system

According to the South African Cancer Association, head and neck cancers in South Africa account for about 10% of all cancers. For clinical documentation and staging purposes the tumour and nodes are separately described using a universal staging system - the TNM system.

T - describes the tumour and the size of the lesion.
N - describes nodal metastasis if present. Subclassification into N1, N2 and N3 describes the size, number of nodes, fixation and laterality of the node(s).
M - indicates distant metastases of the tumour.

This classification allows for accurate patient documentation and serves to monitor the patient's progress. At present, head and neck cancer affects older males three times more frequently than females, but incidence in females is increasing.

With head and neck malignancies the skin of the head and neck is the most commonly affected organ, especially in the sunny African climate. But, of non skin malignancies, about 40% occur in the oral cavity and pharynx, about 40% in the larynx and 10% in the thyroid, with other sites accounting for the remaining 10%. Carcinogens (eg, sun, alcohol, tobacco) and poor oral hygiene may have a direct carcinogenic affect on these organs.

Leukoplakia and erythroplakia in the head and neck area present as white or reddish areas due to hyperkeratosis. They may represent precancerous lesions or may reveal early invading carcinomas. A biopsy is warranted in every suspected malignancy of skin or mucosa - it is the single most important diagnostic tool.

Surgery versus radiotherapy versus chemotherapy

Surgical resection offers the best chance to ablate tumours in head and neck cancers. A shorter recovery period is possible. Surgically resected specimens are also easier to study for cancer clear margins.

Because of the high cosmetic and functional morbidity, quality of life is of particular concern in head and neck cancer patients. A multidisciplinary approach to effective rehabilitation may include speech therapy, social workers, rehabilitation, psychological counselling and prosthodontic evaluation.

The postsurgical cosmetic implications and physical disabilities should be made clear to the patient when obtaining **informed** consent.

Radiotherapy is indicated in high risk patients with tumour involved margins, vessel invasion and extracapsular spread as combined therapy. It is the treatment of choice in certain tumours.

Radiotherapy has the advantage of preserving anatomic structures. However, injury to surrounding structures and tissue may be unavoidable (ie, salivary gland damage with xerostomia, bone necrosis, blindness, radiation mucositis or tooth decay). Most importantly, radiotherapy may induce secondary tumours.

Chemotherapy has a minor role in solid head and neck tumours, except for specific conditions such as lymphoma. Immunotherapy must still be considered a research tool for the present.

Regular follow up visits for the rest of the patient's life are essential for effective management. Other primary tumours may develop in the region in up to 30% of cases. In the first year patients should be seen every month. Thereafter, every second month for a year and then every six months for five years, even if they are symptom free. Head and neck patients are at high risk for second primary tumours in the upper GI and respiratory systems.

Rehabilitation

Early diagnosis is the most important part of rehabilitation. If tumours can be resected transorally there is often minimal postoperative disability. Cosmetic and functional surgery will enhance rehabilitation. Prosthetic appliances may assist with satisfactory rehabilitation.

Prognosis depends on the size of the tumour and its site in the oral cavity or on the tongue. Older males are affected four times more frequently than females.

Carcinoma of the oral cavity

About 10% of all ENT cancers present in the oral cavity (ie, buccal mucosa, lips, alveolar ridge, tongue, soft palate and oropharynx). Oral cancers are on the increase and are more common in men than in women. Smoking and alcohol abuse are strongly implicated etiological factors. In certain populations, the use of chewing tobacco is a major cause of buccal tumours.

The patient may present with a long standing ulcerative lesion, an exophytic granuloma, leukoplakia, or an area of erythroplakia. Small lesions less than 1 cm may be asymptomatic and may only be coincidentally discovered on routine examination. Larger lesions are associated with various degrees of local and referred pain to the jaw and ear. Bimanual palpation may confirm the size of the lesion as well as the surrounding indurated oedematous area.

The diagnosis may be confirmed by an incisional biopsy along with adjacent healthy tissue.

Treatment

Treatment is predominantly surgical. If the mandible is involved, segments including the mandibular canal need to be removed surgically. In certain tumours irradiation can be used as primary therapy or in planned combined therapy. Reconstruction is carried out during initial surgical removal.

Except for small lesions it is usually necessary to include neck nodes in the resection.

Carcinoma of the nasopharynx (NPC)

The incidence of carcinoma of the nasopharynx is rare in Africa. It occurs predominantly in middle aged southern Chinese (ie, from Canton, Hong Kong and Taiwan). There is a strong relationship to the Epsterin-Barr virus. Serology titers increase with the advancing tumour. Lymph node metastasis occurs early and may be the first sign of NPC.

Local infiltration is aggressive to the base of the skull with multiple cranial nerve involvement (ie, the third to the twelfth cranial nerves), as well as involvement of both Eustachian tube orifices.

The patient may present with serous otitis media and conductive hearing loss, blocked nose, epistaxis, headaches and cranial nerve deficits. A nasopharyngoscopy with a rigid or flexible scope may confirm the diagnosis of an exophytic mass in the nasopharynx. A biopsy under local or general anaesthesia may, in the majority of cases, confirm a carcinoma with varying degrees of differentiation. In the minority of cases a lymphoma or a melanoma may be present. A CT scan may reveal the extent of local infiltration.

Carcinoma of the oropharynx

Cancer of the oropharynx extends from the plane of the hard palate to the level of the hyoid bone (ie, comprising the base of the tongue, tonsils and soft palate including the uvula). A squamous cell carcinoma is the most commonly encountered tumour in this region. Lymph node metastasis to the upper jugular node may occur early.

Clinically, pain is a presenting symptom resulting in a sore throat, difficulty in swallowing and radiating pain to the ear via the fifth, seventh and ninth cranial nerves. Persistent sore throat, notwithstanding medical therapy, should suggest further investigation to exclude an underlying malignancy.

Bimanual palpation of the mouth and floor of the mouth is essential along with biopsy of the primary mass. A flexible fibre rhinoscopy may confirm the extent of the tumour. Radiology may reveal the extent of infiltration.

Treatment

Treatment of choice is surgical excision followed by radiation in certain cases. If glands are positive, an *en bloc* neck dissection is indicated. Immediate reconstruction is advisable using various flaps. A temporary tracheostomy may be required and a postoperative feeding tube may be necessary. If radiation is part of the treatment plan, dental caries must be controlled before treatment.

Carcinoma of the hypopharynx

The vast majority of carcinomas of the hypopharynx are squamous cell carcinomas. Early metastasis may occur via the rich parapharyngeal lymphatic supply to the base of the skull and mediastinum. The patient may present with weight loss due to painful and difficult swallowing (odynophagia and dysphagia). Referred ear pain is a common complaint (via the fifth and ninth cranial nerve).

Evaluation of the patient must include an endoscopic examination and radiology. An exophytic and or ulcerative lesion may be present. Close association with the larynx means the voice and the airway may be involved by tumour extension. A tracheostomy may be indicated. A speech therapist may render rehabilitation of voice and swallowing. Hypopharyngeal lesions are usually locally advanced and a concomitant laryngectomy may be required.

Carcinoma of the larynx and vocal cords

The vast majority of laryngeal malignancies are carcinomas. Males are affected more often than females. There is a strong correlation between the disease and alcohol and tobacco use. Notwithstanding medical treatment, patients may present with persistent hoarseness as an early symptom. As the tumour advances dysphagia, odynophagia, stridor and coughing may be present.

Referred ear pain via the vagus nerve is typical. Lymphatic drainage is highly specific resulting in late node involvement with vocal cord tumours, early neck involvement with supraglottic tumours and mediastinal secondaries with subglottic tumours. This affects therapy and prognosis significantly.

Treatment

Laryngoscopy with biopsy should be undertaken. X-rays and MRI studies may reveal soft tissue and cartilaginous infiltration.

The malignant lesions may occur on the vocal cords, supraglottic or subglottic. The lesions may be exophytic or ulcerative and may fix on one or both of the vocal cords. The location, size and nodal metastasis will dictate the extent of the surgery. Small lesions may be endoscopically removed sparing the voice. Larger lesions may require partial laryngectomy or total removal of the larynx. Radiotherapy may be useful in selected smaller lesions.

With lymph node metastasis a concomitant lymph node neck dissection is indicated.

For smaller lesions excellent results and successful rehabilitation may be achieved. For larger lesions good cure rates can be expected but rehabilitation is more difficult.

Persistent hoarseness in the adult is the only symptom of early vocal cord carcinoma at a curable stage.

The solitary neck swelling

The solitary neck tumour poses an interesting challenge to the attending doctor because of the vast possibilities of the aetiology of the neck tumour. It may represent:

- chronic inflammatory diseases (ie, TB or a fungal infection;

- a benign lesion (ie, lipoma or a cyst), or

- a malignant metastasis.

The malignant metastasis may originate from the head and neck area (ie, the larynx, pharynx or tongue), the thoracic cavity, the abdominal viscera or from the perineum. In a small percentage of cases the site of the primary tumour may never be established - the so-called occult primary.

The doctor should first exclude a malignant metastasis, as opposed to chronic granulous disease that may simulate a malignancy. In order to establish a differential diagnosis the tumour must be clinically evaluated. Painless nodules should be considered malignant until otherwise proven.

The consistency of the tumour may vary from a rubbery solid squamous cell carcinoma nodal deposit to a soft doughy feeling representing a benign lipoma or a cyst. A cyst may arise from ectopic branchial cleft epithelium. Chronic granulous infections (ie, TB or a fungal infection) may initially feel rubbery and indurated but may break down to a cyst. A solid, bony, hard swelling may represent an exostosis from the transverse process of a cervical vertebra.

The position of the tumour in the neck is of special importance. A tumour in the anterior triangle of the neck may represent a malignant thyroid deposit in a node. The thyroid itself may be enlarged by a malignancy, a degenerated thyroid cyst or an irregular thyroid hypertrophy. A swelling underneath the sternocleidomastoid muscle may represent a benign branchial cyst or a squamous cell carcinoma deposit, as most of the vertical deep chain of the neck glands are underneath the sternocleidomastoid muscle. A nodal metastasis in the upper two thirds of the posterior triangle (ie, tongue, pharynx or larynx) most likely represents a nodal metastasis from the head and neck area. A palpbale swelling or node in the lower one third of the neck (ie, just above the clavicle) may represent a Virchow-Trossier gland (ie, a distant metastasis from the abdominal viscera, pleural cavity or even from the perianal area or testis) spreading to the neck via thoracic duct drainage.

Pain: Chronic granulous disease (ie, TB or a fungal infection) may cause a dull pain on palpation. Malignant deposits are painless.

Mobility: A nodal deposit may initially feel more mobile but as the disease progresses the infiltrating malignancy may fix the lymph node. Chronic inflammatory diseases are mostly fixed due to the surrounding inflammatory response. Benign cysts are non tender, mobile and painless on palpitation. They often transilluminate and are associated with an abscess.

Workup of a solitary neck tumour:

- *For a nodal metastasis in the upper two thirds of the neck.* A thorough ENT examination may reveal the primary tumour in the head and neck area. There is a small chance of not finding the primary lesion (the so-called occult primary).

For a neck nodal metastasis a chest X-ray may reveal lung pathology. If no obvious primary lesion is apparent, often a thorough ENT general examination and a fine needl biopsy may reveal an unexpected primary malignancy melanoma or a lymphoma. A subsequent lymph biopsy is warranted to tissue type the lymphoma. If the node is a squamous cell carcinoma it should not be removed, but should be included in either a radical neck dissection or a total neck radiation.

- *For a nodal metastasis in the lower neck (ie, supraclavicular).* For a low neck node metastasis (ie, a Virchow-Trossier gland) a fine needle biopsy is indicated. If no cellular differentiation is possible to indicate from which organ the nodal metastasis originated, a

lymph node biopsy is warranted. Needless to say, if fine needle biopsy facilities are not available, an excision biopsy is warranted for adequate tissue diagnosis.

The prognosis for a patient with a neck node metastasis with an occult primary is about 50% three year survival. The larger the neck node, the poorer the prognosis.

An unwarranted randomised neck node biopsy may jeopardise the patient's prognosis and life expectancy.

Carcinoma of the thyroid gland

The thyroid gland consists of two lobes spearated by an isthmus. Anatomical variations may include an absent lobe or an absent isthmus. Occasionally one lobe may extend in the direction of the hyoid above the so-called pyramidal lobe. Islands of accessory thyroid tissue may be present along its course of descent via the thyroglossal duct.

Rarely the thyroid does not descend at all and may remain as a lump on the tongue (the so-called lingual thyroid). This may be the only source of thyroid hormone! The two pairs of parathyroid glands are rice grain size, located on the posterior surface of the thyroid gland in close relationship to the left and right recurrent laryngeal nerves.

The lymphatic drainage that extends between the two lobes and exits the gland close to the recurrent laryngeal nerves has a significant bearing on the symptoms and signs of thyroid cancer and its spread. Carcinoma of the thyroid gland has a familial tendency.

Symptoms

Pain, dysphasia and hoarseness are early symptoms of infiltration of local thyroid carcinoma.

Signs

Carcinoma of the thyroid gland may present as a solitary nodule. Malignant nodules may be **indistinguishable** from benign nodules, hence a thyroid nodule should be considered malignant until proven otherwise.

The presence of hoarseness, a palpable thyroid nodule and an ipsilateral neck node is **pathognomonic of a thyroid malignancy.**

Confirmation of the diagnosis is best made with a fine needle biopsy: accurate in the vast majority of cases. A core biopsy may be necessary to distinguish between the subtypes of thyroid malignancies and avoids an open biopsy.

Treatment

The treatment for thyroid carcinoma is dictated by the histology.

Papillary adenocarcinomas usually present as solitary nodules in a younger patient. The minimum surgery in such a case would be an ipsilateral lobectomy including the isthmus. If the histology reveals a unilateral mixed papillary and follicular carcinoma or a pure follicular carcinoma, a total thyroidectomy is recommended because the clinically negative contralateral lobe may be microscopically seeded with tumour cells.

Pure follicular carcinoma spreads via the blood. Metastasis may be treated with radioactive iodine. Thyroid stimulatory hormone suppression in well differentiated thyroid cancers is advisable.

Medullary carcinoma may be familial and necessitates a total surgical thyroidectomy. Anaplastic carcinoma occurs in older patients. Surgery is contraindicated; radiotherapy with chemotherapy is the treatment modality of choice. These patients should receive thyroid hormone replacement therapy to prevent hypothyroidism.

Nodal metastasis of well differentiated thyroid tumours may necessitate a modified or radical neck dissection. 'Node plucking' is not advisable. Elective neck dissection in clinically negative necks does not enhance the prognosis. Thyroid stimulatory hormone suppression in well differentiated thyroid cancers is advisable.

Melanoma of the head and neck

Most melanomas of the head and neck occur from visible or exposed sites (ie, the skin of the head and neck, especially the scalp and the oral cavity). The incidence of skin melanomas is increasing woldwide at a rate faster than any other human malignancy. Most melanomas arise from existing nevi. Melanoma arising from mucosa is more aggressive than that arising from skin. Internal or obscure sites are less common (ie, the nasal cavity sinuses, pharynx or larynx).

Melanomas are **capricious** in behaviour. They may remain dormant for years, may be non pigmented, may spontaneously resolve, or may disseminate locally or systemically to any organ in the body. They occur equally in males and females over 40, but in HIV infected patients with compromised immune systems, they may occur at a much earlier age. Melanomas may occur in the neck nodes as occult metastasis arising from head and neck primaries and from primary abdominal organs.

Melanomas originate from a melanocyte in the basal layer of the skin.

Factors predisposing to malignant transformation in the visible and exposed areas may be chronic infection, exposure to solar radiation and mechanical trauma (ie, friction and burns). Redheaded and freckled people are more prone to melanomas as their skin is not as well protected from solar radiation. Change in colour, shape, irregularity, thickness and **itchiness** of the melanoma are early warning signs of malignancy. Thickness determines the metastatic risk, lesions thicker than 0.75 mm are more prone to metastasise.

Treatment

Radiotherapy is of questionable benefit; melanomas may be radiation resistant. Surgery is the modality of choice. Wide local excision with 1-2 cm margins has the best result. Local electrocautery ablation for small, thin oral and lip lesions may have good results. Mucosal melanomas are surgically removed. An elective neck dissection is warranted for occult neck gland metastasis if the primary lesion is thicker than 0.75 mm.

Five year survival rates with metastasis to the neck lymph nodes from an occult primary lesion is warranted in selected cases.

Aphthous Ulcers

Aphthous ulcers appear as shallow grey ulcerated plaque usually in the buccal cavity. The ulcers may appear as single entities or as part of a systemic disease syndrome (ie, AIDS or Behçet's disease), in which case they may also appear on the genitalia or anus. The ulcers may measure 4-10 mm. Rarely they may be larger, reaching 10-20 mm: the condition is then referred to as Sutton's disease. In immunocompromised patients, the ulcerated surface may be extensive, may be multiple and may coalesce or recur. In these patients the ulcerated eroded area is also deeper and hence excruciatingly painful, leading to prostration, dehydration and weight loss.

The aetiology is still a matter of conjecture. An autoimmune disorder is frequently mentioned in the literature with secondary oral infections (ie, strains of *Streptococci* and fusiform bacteria or yeast organisms such as monilial infections). Herpes simplex is implicated as a possible aetiologic factor; however, it is more often diagnosed than proven. The Herpes simplex virus is a commensal oral virus and may occur normally in people without oral herpes infections, symptom free carriers of the virus. Physical and mental stress may be predisposing factors.

Treatment

Treatment is purely palliative. Local lidocaine anaesthetic cream lightly dabbed onto the ulcers may provide temporary anaesthesia lasting 12-15 minutes. This allows the patient pain free eating and drinking, thus preventing dehydration and weight loss. A steroid cream applied to the ulcerated plaque helps to control the local inflammatory response and secondary pain. An oral mouthwash with 1% steroids four or five times per day may aid with oral hygiene during the acute phase. Topical saliva barriers keep saliva, which is a digestive enzyme, out of the unprotected tissue (ulcer) thereby reducing pain and facilitating healing.

Systemic analgesics and anti-inflammatory medication will subdue the local discomfort. The area of ulceration usually heals within 10-14 days, except in immunocompromised patients in which case the ulcers may last for many weeks or months.

Topical antiviral creams may prevent secondary herpes infections. The use of systemic antiviral therapy is yet to be proven as a treatment for aphthous ulcers.

1. Harelip. 'Harelip' is a misnomer. The upper lip in a hare is split in the centre, not paramedian as in humans. The cleft lip may extend into the nose and palate.

2. Pitted lower lips. The pits may extend into the lower lip to a depth of 2.5 cm. A low grade infection may be present resulting in a mucoid purulent secretion.

3. Carcinoma of the lower lip. Carcinoma of the lower lip occurs frequently in older Caucasian males with outdoor occupations. Small lesions may be surgically successfully removed.

4. Carcinoma of the anterior two thirds of the tongue. Tongue and oral cancers may clinically appear as exophytic masses or ulcerative craters. Areas of leukoplakia and erythroplakia may represent a carcinoma in situ or invading carcinoma. Poor dental hygiene, nicotine and alcohol abuse in the older male patient may be related to aetiological factors. Neck gland metastasis occurs relatively late to the upper ipsilateral chain of lymph nodes. The presence of a lymph gland metastasis greatly reduces the long term prognosis.

5. Carcinoma of the nasopharynx. Carcinoma of the nasopharynx appears mostly in middle aged Chinese. The first symptom may be conductive hearing loss due to Eustachian tube involvement.

6. Carcinoma of the oropharynx. Carcinoma of the oropharynx in the older male patient may present with dysphagia, odynophagia and radiating pain to the ear. Emaciation to various degrees may be present due to negative nitrogen balance.

7. Carcinoma of the hypopharynx. Carcinoma of the hypopharynx sufferers may present with referred ear pain, weight loss and drooling as a result of painful and difficult swallowing.

8. Graphic documentation of a left transglottic vocal cord carcinoma (ie, a carcinoma involving the left vocal cord extending to above and below the vocal cord). Persistent **hoarseness** in the adult is the **only symptom** of early vocal cord carcinoma at a **curable stage.** If, despite medical treatment, hoarseness continues for longer than two weeks the sufferer should be investigated for evidence of a malignancy.

9. The solitary neck swelling. An unwarranted randomised neck node biopsy may jeopardise the patient's progress and life expectancy. In the vast majority of cases, where a thorough search for the primary has been unsuccessful, a fine needle biopsy may assist in the diagnosis. Cytologists may experience great difficulty finding the primary site, especially with thyroid and salivary gland tumours.

10. Well differentiated thyroid cancer may present in young patients with a solitary neck nodule. In older patients a fixed thyroid nodule with hoarseness may represent an anaplastic carcinoma.

Acute Airway Obstruction

Congenital posterior nasal choanal atresia

Congenital airway obstruction is of special importance in infants because of their relatively small airway diameter. Infants are obligate nose breathers and can easily suffocate through nasal obstruction. Posterior nasal choanal atresia may be bony or membranous, unilateral or bilateral.

Signs

In bilateral atresia the infant suffers from severe respiratory distress and cyanosis at birth. Typically the cyanosis **improves on crying.** Blocked nasal airways (unilateral or bilateral) are associated with a glairy nasal discharge.

The diagnosis is confirmed by nasal endoscopy or the inability to pass a catheter from the nostrils and nasal cavity into the postnasal space. The lesions are well demonstrated by a CT scan performed with contrast media drops added into the nasal cavity.

Treatment

- In bilateral atresia, an oral airway should be established immediately.

- The use of an oral airway or open mouth teat is usually sufficient until the defect is surgically corrected; this should be done as soon as possible.

- Unilateral atresia, by contrast, is seldom dangerous and is often only recognised later in life.

- Surgical repair of unilateral atresia may be delayed until structures are larger.

Acute airway obstruction due to hereditary angioedema

With hereditary angioedema there is a strong familial history. Attacks of hereditary angioedema usually start during childhood and may become more frequent and severe during adulthood. Attacks may occur spontaneously or may be precipitated by minor trauma (eg, sports contact injuries, dental extractions and minor surgical procedures).

Symptoms and signs

The patient may present with non itchy, blanched, urticaria like hives on the face and extremities lasting for two to three days. Concomitant swelling of the mouth, pharynx and larynx may lead to rapid, potentially fatal airway obstruction. Gastrointestinal cramps may mimic appendicits or even peritonitis. Serology tests should be marked for C1 esterase inhibitor (quantitative and functional) as well as for C2 and C4, which will confirm the diagnosis. **All relatives should be tested.**

Treatment

- Adrenaline, cortisone and antihistamines may have erratic effects on the relief of the airway obstruction.

- Intubation and tracheostomy may eventually be necessary.

- Prophylactic therapy with fresh frozen plasma and danazol 200 mg to 600 mg daily in three divided doses 24 hours prior to surgery or dental procedure.

- A **MedicAlert** bracelet should be worn.

Acute partial airway obstruction by a foreign body

Young children and elderly people are particularly prone to foreign bodies in the hypopharynx. Children tend to swallow coins, buttons, marbles and parts of toys. The elderly may accidentally swallow dentures, fish bones or poorly masticated sinewy meat, especially the edentulous patient consuming a large carnivorous meal with copious amounts of alcohol - the so-called 'restaurant coronary'!

The patient presents with a bewildered facial expression and may have dysphagia, odynophagia, drooling, hoarseness, aphagia and a variable degree of inspiratory stridor. The laboured breathing efforts cause intercostals, suprasternal and substernal retraction.

Cyanosis due to anoxia indicates severe or imminent airway obstruction leading to CNS damage within a few minutes.

Treatment

In cases of partial obstruction, the patient should be transported to a hospital and X-rayed to determine the site and type of foreign body. The patient should be accompanied to hospital by a qualified medical or paramedical person capable of providing an emergency airway.

Ideally the foreign body should be removed by direct laryngoscopy or rigid bronchoscopy or oesophagoscopy performed under general anaesthesia by an otolaryngologist.

In cases of total obstruction, emergency life-saving measures should be applied. The patient may be comatose and cyanotic. Often no air movement is evident and violent respiratory efforts are apparent.

Acute total life-threatening airway obstruction

In an airway emergency the immediate management is one or more of the following (within four minutes):

- Clear the oral cavity and pharynx with a finger and place the patient on his back.

- Perform the Heimlich manoeuvre, a forceful sudden jerk on the upper abdomen to increase the abdominal pressure. The sudden increase in abdominal pressure is propagated to the thoracic cavity, expelling the foreign body from the laryngeal introitus.

- If necessary, undertake a cricothyrotomy with a cricothyrotomy cannula and trocar.

- Oral endotracheal intubation or tracheostomy may be necessary.

1. Heimlich manoeuvre. The Heimlich manoeuvre is a forceful jerk on the upper abdomen to expel a foreign body trapped above the vocal folds. The insert shows the position of the hands on the upper abdomen. The lower hand is fisted, the upper hand flat to add force to the jerk. An artificial airway (ie, a cricothyrotomy, tracheostomy or an endotracheal intubation) may be necessary.

Trauma

The larynx and trachea

Laryngeal and tracheal fractures are surgical emergencies. If one is to prevent long term problems with airway stenosis (ie, glottic, supraglottic and infraglottic) they should be corrected within 24 to maximum of 48 hours of the injury.

Blunt trauma to the larynx, such as a blow from a fist or boot, may cause laryngeal injury with life threatening airway obstruction.

Some examples of blunt trauma to the larynx or trachea are:

- Motor vehicle accidents where the larynx impacts on the steering wheel or dashboard.

-Incidents where the cartilaginous larynx impacts with a suspended cable.

- Assault such as direct fist or blunt instrument blows to the larynx.

Penetrating trauma may be sustained by a gunshot wound or stab wound to the neck, and may penetrate into the larynx. In addition to the cartilaginous and soft tissue trauma, acute surgical emphysema may follow which may further compromise the airway.

Associated injuries (eg, spine, blood vessels, nerves or central nervous system lesions) must be excluded. **Neck injuries can be fatal without marked external signs due to a haematoma or anatomical distortion obstructing the airway.**

Symptoms

The patient may be distressed and bewildered or may be in a state of shock. The patient may experience local tenderness and pain. Airway distress may be sudden or progressive. Odynophagia (painful swallowing) may also be present.

The voice may vary from mild hoarseness to aphonia, which may prevent the acutely distressed patient conveying the precise accident particulars to the attending doctor.

Signs

Ecchymosis of the skin may appear where the blow struck the larynx. Pain and tenderness may be noticed on palpation of the larynx. Surgical emphysema of the subcutaneous tissue may be prominent as well as loss of anatomical landmarks, with crepitus on palpation of the cartilaginous fragments of the larynx. These give an indication of the severity of the trauma.

Associated skin lacerations of various degrees, bleeding, as well as mucosal lacerations of the tongue, larynx and pharynx may also be present.

Indirect laryngoscopy, if possible, may reveal reduced vocal cord mobility, reduced airway patency and anatomical distortions.

X-rays of the neck may reveal subcutaneous air in the soft tissue, or displacement of the cricoid or trachea below their normal position. Spinal fractures, as well as laryngeal fractures may also be present on the X-rays. Direct laryngoscopy under general anaesthesia is done at the time of the corrective surgery.

Treatment

In injuries of this nature, valuable time may be lost due to the aphonia. The attending doctor must be aware that severe **life threatening** airway obstruction may be present with minimal external signs of bruising, lacerations or bleeding. Persons attending trauma cases must be constantly aware of the possibility of laryngeal injury.

Pinhead sized entrance of a sharp instrument or a blunt blow may cause catastrophic airway obstruction.

Intubation, perhaps already performed by paramedical personnel who attended the patient at the site of injury, may disguise or further compound the laryngeal injury.

Maintaining an airway is of lifesaving importance:

- Airway obstruction may necessitate emergency tracheostomy, cricothyrotomy or intubation.

- It may be possible to intubate the patient, but attempts to intubate a distorted larynx can lead to sudden death.

- Direct laryngoscopy and urgen exploration of the larynx with repair of the laryngeal or tracheal cartilages may be required.

- Excellent results are usually obtained with early repair while results are poor if repair is delayed.

- Systemic antibiotics and antitetanus therapy are required.

Any sportsperson who has suffered a contact sport impact to the larynx that has resulted in even the **slightest** of airway obstructions should be urgently evaluated by a specialist laryngologist. This professional will decide whether the patient should be transported to an emergency room, or simply observed. Long term complications of neglected laryngeal injury may result in permanent hoarseness and airway problems.

The jaw and teeth

A jaw fracture is usually the result of low velocity, high energy impact and may arise from a sports altercation. Teeth may also be involved in the fracture or separately knocked out. If teeth are lost, they should urgently be retrieved for reimplantation.

Because the forceful impact may be transferred to the cervical spine and cranium, an associated spinal injury and a contracoup injury should be kept in mind especially in the unconscious patient. The head should be immobilised in relation to the torso during transportation.

Symptoms

The patient usually presents with muffled speech due to the painful trismus. Examination may reveal an expanding haematoma of varying size over the fracture site, extending into the oral cavity. Haemorrhage from mucosal tears may be severe.

Treatment

- Remember the ABC of jaw fractures: **A** - airway; **B** - bleeding; **C** - cervical spine.

- The profuse haemorrhage and expanding haematoma may compromise the oropharyngeal airway and an emergency tracheostomy may be indicated to secure an adequate airway.

- The fractured mandible should be splinted to the upper jaw by a figure-of-eight Barton bandage that supports the jaw below and in front.

- Open reduction and fixation may be delayed for a few days to stabilise the patient.

- Antibiotic and anti-inflammatory anaglesics should be administered as ancillary therapy.

- A knocked out tooth, once retrieved, may be successfully reimplanted, preferably if done within the first hour (the so-called 'golden' hour!).

- The tooth should be placed into the socket and manually stabilised en route to the dentist.

- Beware of aspiration of a loose tooth or tooth fragment.

-If a knocked out tooth cannot be placed into the socket, have the patient hold the tooth in his cheek and rush to a dentist for reimplantation (within the hour). If the tooth cannot be found, a chest X-ray is mandatory to exclude the possibility it has been aspirated.

- If the fracture line intersects a knocked out tooth socket the tooth cannot be salvaged.

- To clean the tooth:

1) Handle the tooth only by the crown; do not touch the root or the very delicate tooth periodontal membrane may be injured, thus reducing the chance of successful reimplantation.

2) Do not wash the tooth in tap water. Request the patient to gently lick the tooth clean while gripping the tooth by the crown.

- A dentist will stabilise the reimplanted tooth with an interdental bonded splint.

- An antitetanus immunisation is mandatory.

Summary

- With jaw fractures, rapidly progressing airway obstruction must be anticipated. Support the jaw with a figure-of-eight Barton bandage.

- Urgent reimplantation of knocked out teeth should be undertaken within the so-called 'golden' hour.

1. Larynx fractures (ie, from motor vehicle accidents). Blunt trauma to the larynx, such as a blow from a fist, may cause laryngeal injury resulting in life threatening obstruction, with only superficial abrasion visible over the larynx. Airway obstruction may necessitate an emergency trachestomy or a cricothyrotomy. Forceful blunt trauma to the larynx may result in fractures of the laryngeal framework, resulting in acute life threatening airway obstruction.

2. Mandibular fractures. Direct blows are common causes of mandibular fractures. Dislodged teeth may be reimplanted. Handle the dislodged tooth by the crown in order not to damage the delicate periodontium.

Allergic Airway Emergencies

Bee sting

There may be more than an "ouch" to bee stings! Bee sting angioedema of the nasal, oral and laryngeal airways is an ENT emergency.

Types of reactions

Bee sting venom may cause a local stinging sensation due to the bee toxin. A systemic reaction depends on the individual patient's immunologic/allergic status to the bee toxin. Older patients may be more severely affected due to previous exposure to bee stings.

The local response may consist of local urticaria, oedema and pain lasting approximately 24 hours, due to the bee toxin. Acute systemic responses in sensitised patients may include angioedema (ie, swollen tongue, pharynx and larynx). In severe cases, acute bronchospasm and cardiovascular collapse (ie, anaphylactic shock) may ensue.

Pathophysiology

The bee venom contains hyaluronidase as a spreading factor. Enzymes, peptides and amine complexes as well as the haptens of the bee toxin may act as allergens in sensitised patients.

Treatment

If possible, remove the sting by scraping it out with a knife blade, otherwise the residual venom in the sting may be squeezed into the stung spot. Locally applied ice cubes and a pressure bandage may retard the venom absorption and reduce pain. **Folklore remedies** that call for the application of all kinds of local antidotes are a waste of time.

If a systemic shock syndrome is looming, adrenaline, steroids and antihistamines may prevent a full blown anaphylactic reaction in a previously sensitised patient.

Long term prophylaxis to sensitised patients should take the form of desensitisation to bee stings as a prudent precaution against an unexpected sting. **The immunologic memory to bee stings is lifelong!** The monthly desensitisation maintenance injections should be continued for at least five years.

Some words of caution to sensitised patients:

- Refrain from walking barefoot on the lawn.

- Avoid gardening during the pollination season.

- When eating outdoors, be aware that sweet foods and soft drinks may attract bees. **Drink with a straw!**

- Brightly coloured clothing may lure bees as may sweet smelling body odours and perfumes.

- Remember that certain homeopathic remedies contain bee pollen.

Be 'bee-wise', always have an emergency (anaphylaxis) kit at hand consisting of a preloaded syringe of adrenaline, antihistamines and steroids for the unfortunate patient.

Food allergy

The prevalence of food allergy in atopic individuals in South Africa is estimated at around 10%. Adverse reactions to food may be divided into immunoglobulin E (IgE) or non-IgE mediated reactions. The symptoms are caused by IgE bodning to the mast cells causing degranulation of the mast cells and release of cell mediators. Non IgE mediated reactions are mediated by vasoactive amines and may follow the same pathway as IgE mediated allergic reactions (ie, degranulation of the mast cells lining the respiratory system and release of cell mediators). The two types of allergies differ only in their triggering mechanisms.

Allergens may be traced to some fast foods (ie, hamburgers, breakfast cereals, pastries), ice cream, soy milk and soy products, legumes such as peanuts, Chinese and Indian foods, peanut cooking oil, marzipan and some sea foods (eg, crustaceans such as crayfish).

Adverse reactions may include the upper aerodigestive system (ie, swollen lips, blocked nose, swollen tongue and laryngeal oedema). In severe cases angioedema may cause acute asthma, lowered blood pressure and unconsciousness (ie, anaphylaxis). If this occurs it is an allergic emergency.

Sensitised atopic patients may develop symptoms within minutes after eating, touching or merely smelling the offending foods.

There is increasing evidence that maternal diet during pregnancy may influence and modulate the immune system of the developing foetus. Certain allergens in the maternal diet (ie, peanuts and products that contain them, such as peanut butter, breakfast cereals, health bars and biscuits) should be avoided during pregnancy.

Treatment

Specific food avoidance should be strictly adhered to by keeping a food diary of the offending foods.

Sensitised patients should wear a bracelet warning of the fact and carry an emergency kit with a preloaded syringe with 1/1000 adrenaline. Adrenaline aerosol inhalers are also available.

In the future, genetically modified foods may 'breed-out' naturally occuring allergens that may cause inadvertent allergenicity.

Latex allergy

Latex allergy has become the most common cause of occupational rhinoconjunctivitis and asthma among healthcare workers. Latex is obtained from the Brazilian rubber tree, and latex allergy is caused by a protein (isoprene) incorporated into the latex.

Allergic (sensitised) individuals develop itchy and runny noses and eyes, sneezing, coughing, wheezing and asthma. A drop in blood pressure and unconsciousness (ie, anaphylaxis) may result.

Pathogenesis

Repeated exposure to minute amounts of latex proteins in the powder that is used to coat latex surgical gloves may result in the formation of IgE antibodies. The mast cells may degranulate and release cell mediators and histamine. Some highly allergic individuals may develop anaphylaxis by merely inhaling surgical glove powder. A RAST test may confirm the latex allergen.

Treatment

Avoidance is mandatory to prevent progression of the latex allergy. Desensitisation to latex is not possible. Latex free gloves are available. Latex contaminated products in the theatre may also trigger an allergic response.

Many hospital products that may contain latex, include: anaesthetic masks, breathing bags, blood pressure cuffs, breathing circuits, catheters, cervical dilators, dental appliances, elastic bandages, endotracheal tubes, injection adaptors, eye dropper bulbs, face masks, feeding tubes, gloves, haemodialysers, hot water bottles, implants, instrument mats, IV injection ports, nasal airways, orthodontic elastics, rubber sheeting, syringe stoppers, tooth protectors, tourniquets, ultrasound cover, urine bags, ventilator tubing, warming blankets, wheel chais and wound drains.

Unfortunately, certain domestic products may also contain latex and may trigger the allergic sequelae. These include: bandages, baby bottle teats, balloons, condoms, diaphragms, douche bulbs, elastic in clothes, diapers, erasers, eye droppers, hot water bottles, stress balls, paints (waterproofing), rubber grips on racquets, bicycle tools, rubber bands, rubber toys and shoes (Professor Potter, UCT RSA).

Sensitised patients should wear a bracelet warning of the fact and carry an emergency kit to prevent an allergic seizure progressing to anaphylaxis. Sensitised health workers should request latex free gloves and a latex free hospital environment.

Drug allergy (ie, antibiotics and anti-inflammatories)

Symptoms

Within 30 minutes the patient feels a tingling sensation in the face and mouth, a sensation of warmth, and shortness of breath. The patient's condition may deteriorate rapidly;

the chest pain and shortness of breath simulating an asthma attack or myocardial infarction.

Signs

The earliest signs are of the upper airways: sneezing, watery nasal discharge, blocked nose, hoarseness and sweating. Oral signs follow with swelling of the lips and tongue. Pharyngeal and laryngeal oedema give rise to hoarseness and an inspiratory stridor. Pulmonary signs are inspiratory wheezing and cyanosis. Cardiovascular signs are rapid, feeble pulse with hypotension followed by severe arrhythmias. Cardiopulmonary collapse, coma and eath may follow.

Differential diagnosis

Anaphylactic shock should be distinguished from syncope. In syncope:

- The patient is pale, not cyanotic.

- The pulse is full and regular, not rapid, feeble and with arrhythmias.

- There is severe hypotension.

- There are no concomitant pulmonary complications (eg, bronchospasm).

Treatment

The following treatment should be considered:

- Parenteral adrenaline.

- Intravenous corticosteroids and antihistamines.

- Oxygen and aminophyline.

- Intubation and tracheostomy may be necessary.

Known allergies should be avoided and recorded on a **MedicAlert** bracelt. People often say they are allergic to penicillin after experiencing vague symptoms of nausea. **Their allergy should be verified by lab tests before this valuable medication is permanently withheld.**

Respiratory Tract Infections

Corticosteroids are indicated in acute airway obstruction only when a viral infection or allergy is the cause. When caused by a bacterial infection, corticosteroids are not indicated and large doses of intravenous antibiotics are necessary. At all times a tracheostomy set should be available.

Ludwig's angina

Ludwig's angina is a bacterial cellulitis of the floor of the mouth, mainly involving the submandibular and sublingual spaces. The portal of entry is via a dental abscess. Severe swelling below the tongue pushes the tongue backwards and upwards compromising the oropharyngeal airway.

Symptoms and signs

Patients with Ludwig's angina experience severe tenderness and pain the floor of the mouth. Dysphagia and odynophagia are also present. The patient sits with mouth wide open due to the pain and swelling and drools due to the inability to swallow. Cellulitis, inflammatory oedema and inflammation of the floor of the mouth are evident.

The patient may be pyrexic, toxaemic and dehydrated. There may be an inspiratory stridor, indicating the severity of the airway obstruction.

Retropharyngeal abscess

Retropharyngeal abscess is a bacterial cellulitis with abscess formation in the retropharyngeal lymph glands. It involves the retropharyngeal space. It occurs mainly in young children and is a life threatening airway emergency. The posterior pharyngeal wall is pushed forwards, compromising the hypopharyngeal/laryngeal airway. In AIDS patients this condition is much more prevalent.

Symptoms

The patient experiences neck pain, dysphagia, odynophagia and drooling from the mouth. The patient may be pyrexic and toxemic. Dehydration occurs due to the inability to swallow.

Signs

An inspiratory stridor indicates the severity of the compromised airway. Posterior pharyngeal swelling and fluctuation may be present. A lateral neck X-ray will reveal a widening of the retropharyngeal space and confirm the diagnosis. Opisthotonos may be present due to the involvement of the neck muscles, and it may simulate meningitis.

Croup (laryngotracheobronchitis)

Laryngotracheobronchitis occurs in young children, especially those between two and seven years, and is preceded by an upper respiratory tract viral infection.

Symptoms and signs

The patient presents with a night time barking cough and an inspiratory and expiratory stridor. The patient is hoarse due to the severely swollen vocal cords, may be feverish, but is rarely toxemic. No drooling is present as there is no odynophagia or dysphagia.

Treatment

- Hospitalise the patient, humidify the inspired air and administer intravenous rehydration fluids and corticosteroids.

- Provide sympathomimetic vaporised inhalation if the patient is in a controlled environment (ICU) in case of rebound mucosal swelling.

- Antibiotics are rarely necessary.

- The parents should be present to help calm the child.

- It may occasionally be necessary to intubate or tracheostomise the patient.

Epiglottitis

This occurs mostly in younger children due to *Haemophilus influenzae.* The onset is usually rapid. A compromised airway may result, with inspiratory stridor due to the acute airway oedema causing breathing obstruction.

Signs

Examination may reveal a toxic patient with high fever, a 'hot potato' voice and drooling. The patient prefers to sit upright in a chin forward position for maximum airway patency. At radiology, examine the patient in a sitting position. Lateral X-rays of the neck will confirm the diagnosis, but should only be carried out with tracheostomy or intubation facilities immediately available.

Treatment

- Hospitalise the patient with intravenous antibiotics and rehydration.

- Humidification of the airway is necessary.

- The patient should be intubated for 72 hours.

- Sedation with assisted ventilation may be necessary. A standby intubation or tracheostomy set is mandatory from the moment of admission.

- A tracheostomy may be required for longer periods of ventilation or in the case of difficult intubation.

1. Bee sting venom may cause a local stinging or a systemic reaction if the individual is sensitised. Acute angioedema of the airways may lead to acute bronchospasm and cardiovascular collape (ie, anaphylactic shock). Sensitised patients should be desensitised as a precaution against unexpected bee stings.

2. Crustacean sensitised atopic patients may develop symptoms within minutes of smelling, touching or eating the offending food. The most severe reaction to food allergens is anaphylactic shock (ie, drop in blood pressure, loss of consciousness and an acute attack of bronchospasm and laryngeal oedema).

3. Latex allergy. Repeated exposures to minute quantities of latex proteins may result in the production of harmful IgE antibodies in some sensitised individuals. Histamine release from degranulated mast cells in the upper and lower airway mucosa may result in acute bronchospasm, laryngeal oedema, drop in blood pressure and anaphylaxis.

4. Epiglottitis. The position of a patient with acute epiglottitis, the so-called 'air hunger' position. The patient is in agony and prefers to sit upright in a chin forward position for maximummm airway patency. A rapid and fatal airway obstruction may follow.

Tonsillitis and Peritonsillar Abscess

Acute tonsillitis

Acute purulent tonsillitis is, in most cases, due to *beta-haemolytic Streptococcal* infection and is prevalent in the paediatric age group.

Pathogenesis

In acute purulent tonsillitis, the lymphoid follicles in the tonsillar tissue are transformed into small abscesses within the tonsillar matrix and discharged into the tonsillar crypts. Pus draining from the crypts is seen as white spots; termed follicular tonsillitis. If the infection is severe enough these spots coalesce into a confluent membrane; this is termed membranous tonsillitis. The distinction bears no clinical significance.

Symptoms

Children with acute purulent tonsillitis suffer from throat discomfort. Referred ear pain and, rarely, odynophagia (painful swallowing) may be experienced. Children usually appear toxaemic and have a fetid breath. Cervical lymphadenopathy often occurs in children and presents as bilateral tender nodes at the angle of the jaw; the so-called jugulodigastric gland which is situated in the 'V' intersection between the digastric muscle and the internal jugular vein.

Adults, on the other hand, experience agonising throat pain. Odynophagia and referred ear pain are also often present. Adults are usually less toxaemic than children.

Treatment

The antibiotic of choice is synthetic penicillin for one week. If the patient is allergic to penicillin, cotrimoxazole, fluoroquinolone or cephalosporin are equally effective. Treatment should be commenced before swab microbiological results are obtained. Anti-inflammatory medication may help for discomfort and fever. A soft food diet, high fluid intake and bed rest are recommended for the toxaemic stage.

If no improvement is achieved within 24 hours, infectious **mononucleosis** should be suspected and a blood sample taken for serologic tests and full blood count. An opportunistic infection must be borne in mind, and a HIV test should be undertaken in suspected cases.

Surgical management of recurrent tonsillitis

Although the adenoids are usually removed during tonsillectomy in children, the indications for adenoidectomy and tonsillectomy should be on their individual merits.

Patients with palatal incompetence, such as cleft palate and especially submucosal cleft palate, should be approached with circumspection as they may have nasal speech or regurgitation after adenoidectomy.

Adenoidectomy may form part of the treatment of recurrent sinusitis, rhinitis or ear infections.

Removing the tonsils is indicated:

- If there is a history of repeated attacks of acute tonsillitis causing systemic upset, or combined with febrile convulsions.

- Following a peritonsillar abscess.

Enlarged tonsils may cause airway obstruction and possible cor pulmonale, an absolute indication for an adenotonsillectomy. It is important to nurse these cases in intensive care postoperatively until cardiopulmonary stabilisation is achieved.

Adequate documentation from the family doctor is necessary to distinguish between viral pharyngitis and acute bacterial tonsillitis. If uncertain, it is recommended that the otorhinolaryngologist personally monitor a number of infections before embarking on surgery.

Peritonsillar abscess (quinsy)

Peritonsillar abscess or quinsy usually occurs unilaterally in adults who do not necessarily have a history of recurrent tonsillitis. The patient usually complains of severe unilateral throat pain, fever, dysphagia and odynophagia. Peritonsillar abscess has become a common presenting condition ni patients with HIV/AIDS.

Signs

Patients usually present with acute tonsillitis, may be toxemic and are often significantly dehydrated. They have a 'hot potato' voice due to immobilisation of the soft palate or base of tongue. Typically they have trismus (inability to admit two finger widths between the incisor teeth). They have a unilateral prominent tonsil with the uvula pushed from the midline.

Treatment

Peritonsillar abscess is a potentially life threatening disease. It is advisable to hospitalise these patients, providing adequate intravenous hydration and large dosages of intravenous antibiotics. If they have not responded significantly within 12 hours, surgicaldrainage should be performed. As the toxaemic state can lead to problems during anaesthesia, it is advisable to control this before a general anaesthesia is administered. Surgical drainage can then be performed under general or local anaesthesia.

An abscess should be drained at the point of maximal fluctuation, usually over the superior pole to the tonsil. The tonsil 'floats on a sea of pus', which leaves a well dissected area between the tonsil and the tonsillar bed, allowing easy access to proceed to a quinsy tonsillectomy on the affected side. The remaining tonsil may be removed with considerably more effort due to the acutely oedematous infected tonsil and excessive bleeding. There is no value in incising an area of cellulitis until the pus becomes localised and fluctuates. A tracheostomy set should be available during the procedure.

Treatment summary

- Hospitalise the patient and administer intravenous fluids and antibiotics.

- Intravenous penicillin, amoxycillin/clavulanic acid or a cephalosporin combined with metronidazole are effective.

- Monitor the airway.

- A standy intubation/tracheostomy set should be available.

- Incision and drainage should be performed when fluctuation occurs in the area of inflammation.

1. Acute tonsillitis. Removal of tonsils is indicated if there is a history of repeated attacks of acute tonsillitis causing systemic upset, and enlarged tonsils cause airway obstruction.

2. Peritonsillar abscess - quinsy. A peritonsillar abscess is potentially life threatening.

Voice Disorders

Voice disorders may vary from a voice that is unpleasant for the listener to ineffective communication. They may be functional or organic.

When no organic aetiology can be established, the voice disorder is functional. It may have emotional and psychological causes.

Organic voice disorders are secondary to congenital or acquired anatomical disorders.

Voice disorders may be categorised according to quality, resonance, loudness and

pitch:

- Quality may be affected by vocal cord pathology resulting in hoarseness, harshness and breathiness.

- Resonance may result in hyper- or hyponasality (ie, nose/sinus or nasopharyngeal obstruction).

- Loudness (or softness): Autophonia due to a conductive deafness may result in a too soft voice while presbycuses may result in the speaker using a too loud voice because he does not hear himself. Control of excessive loudness of the voice is important in patients with vocal nodules.

- Pitch: A narrow pitch range may result in a monotonic voice such as in a totally deaf patient.

Clinical evaluation

Listen for the above voice qualities while the patient reads, counts or repeats selected words.

Take a history of the duration of the voice disorder (ie, acute or chronic), the sufferer's habits (ie, screaming), professional duties (ie, teacher, auctioneer), smoking, alcohol abuse and previous intubations for anaesthesia or GORD.

Physical examination should include a general ENT examination with emphasis on the nasal airways, sinuses, hard and soft palate and adenoids in children. Nasopharyngoscopy and laryngoscopy are mandatory during phonation in adults to exclude malignant tumours.

Children may have to be examined under anaesthesia to exclude a recurrent respiratory papillomatosis.

A reliable stroboscopic examination is only possible in older children and adults. Computer assisted voice analysis may help with complex voice disorders.

Speech and voice therapy, if required, should be undertaken by a specialised therapist and may last for many months.

Vocal nodules in children are due to misuse and abuse of the vocal folds (ie, 'screamer's nodules'). In recalcitrant cases surgery may be necessary to remove the vocal cord nodules. Microsurgical excision or a CO_2 laser may be used. Long term results depend largely on the patient following speech therapy to prevent a recurrence.

Hoarseness

Hoarseness in infants

Laryngeal pathology causing hoarseness in infants may also be associated with stridor due to the small size of the larynx. This is usually due to congenital abnormalities especially laryngomalacia. Aggravating factors include an upper respiratory tract infection.

The patient should be placed under observation. Rapid deterioration of the laryngeal airway may follow within hours.

Infants with stridor should undergo endoscopy to establish the correct diagnosis and prognosis.

Hoarseness in children

Transient hoarseness is caused by an upper respiratory tract infection or acute vocal abuse. Chronic hoarseness is caused by:

- Repeated habitual vocal abuse, which may result in vocal nodules; so-called 'screamer's nodules'. The condition may be corrected with changes to the child's behaviour pattern.

- Laryngeal papilloma are like skin warts and may spread in the larynx and trachea, causing airway obstruction. Repeated removal by CO_2 laser is the therapy of choice. Intubation or tracheostomy may disseminate the condition. Subglottic spread and loss of control of the disease may ultimately be fatal. The care of these patients is highly specialised.

Hoarseness in adults

In adults, hoarseness may be transient or chronic. Transient hoarseness is due to acute vocal abuse or a viral respiratory infection causing inflammatory oedema. Chronic hoarseness may be caused by the following:

- Chronic vocal abuse such as with singers straining their voices in the wrong vocal range, or with auctioneers. This results in bilateral recurrent submucosal haemorrhage of the vocal folds, which is resolved by fibrosis and hyalinisation. Repetitive trauma results in vocal nodules, or 'singer's nodules', an occupational hazard. On viewing the vocal folds, these nodules appear as two small opposing triangles on each vocal fold at the junction of the anterior one third and the middle one third. Vocal rest may improve the condition. Microsurgical removal with follow up speech therapy is usually the only cure for long standing cases resilient to conservative therapy.

- Intubation granulomas may follow two to three weeks after a traumatic or 'crash' intubation and appear as a large sessile unilateral vocal cord polyp, usually situated posteriorly on the arytenoid. Surgical removal may have to be repeated before the condition is controlled. Gastro-oesophageal reflux and infection need to be controlled.

- Reinke's oedema (polypoid degeneration) follows long standing chronic irritation, especially smoking, but also drinking or vocal abuse. Elimination of the causative agents may result in amelioration of the condition, but microlaryngeal surgery is usually required.

- Vocal cord palsies may be transient due to a viral infection. Every case requires a full work up, even in an otherwise healthy individual, to exclude a primary or secondary malignancy along the pathway of the entire recurrent laryngeal nerve (ranging from the base of the skull to the thyroid gland), or an aortic aneurysm on the left side.

- If hoarseness lasts for more than two weeks in patients on medical therapy, an otolaryngologist must perform a laryngoscopy on these patients. **Persistent hoarseness in the adult is the only symptom of early vocal fold carcinoma at a curable stage.**

- Iatrogenic hoarseness (dysphonia) may be induced by topical inhaled steroids leadingto a degree of dyskinesia of the voluntary vocal muscles that determine the vocal cord tension.

- Spastic (adductor or abductor) vocal dystonia.

Vocal nodules (singer's nodules)

This is a chronic benign condition of the membranous vocal folds more frequently encountered in children (as screamer's nodules), females and auctioneers.

Symptoms

A vocal capability test questionnaire may reveal the following symptoms:

- Voice quality and hoarseness (roughness) that may vary from day to day.

- Loss of the ability to sing high notes comfortably.

- Short initiation period of aphonia especially with soft high notes.

- Voice fatigue (ie, less vocal endurance).

Pathophysiology

Vocal nodules are due to chronic repeated vocal abuse. The impact of the membranous folds on collision against each other may cause small haemorrhages and oedema in the submucosal (Reinke's space). The chronic and repeated episodes of vocal trauma and subsequent resolution eventually result in nodule formation on each vocal fold at the point of maximal impact (ie, between the anterior one third and posterior two thirds of the membranous vocal fold). The vocal nodules may vary in size, contour, colour and symmetry.

Diagnosis is easily established by direct or indirect laryngoscopy performed in the consulting rooms. Children may not be cooperative and may need to be evaluated under anaesthesia.

Treatment

Medical management may focus on secondary contributions from allergies and acid reflux (GORD).

Voice therapy for a three month period should focus on changing speech behaviour and vocal abuse. Voice therapists are mandatory for professional singers if they seek to avoid permanent vocal damage that may endanger their singing careers. With therapy the nodules may regress.

If the voice remains unacceptable, microsurgical removal becomes an option. Precision microsurgical removal has excellent results. One week of postoperative voice rest is advisable. Postoperative voice training by a voice teacher, especially for professional singers and entertainers, is indicated to prevent a recurrence.

Intubation polyps

Intubation polyps are a rare post-intubation complication of general anaesthesia. These polyps may follow endotracheal anaesthesia in adults. Females are more prone to this complication due to a smaller larynx.

The aetiology may be a direct intubation trauma or pressure necrosis over the arytenoid process due to an over inflated endotracheal cuff. Excessive fibroses healing over the site of the injury may become covered by mucous membrane to form a sessile or a pedunculated polyp.

Treatment is surgical removal. A recurrence may be likely and the patient should be followed up.

Tuberculous laryngitis

Tuberculosis of the larynx may accompany pulmonary tuberculosis or may occur independently. It is on the increase, especially in patients with lowered immune responses (ie, those suffering from AIDS).

Weight loss may precede persistent hoarseness, dysphagia and odynophagia.

Clinical examination of the larynx may reveal a solitary granulous lesion on a vocal cord or multiple affected sites. A solitary granuloma on a vocal cord may easily be confused with a malignancy until otherwise proven. Chest X-rays and sputum tests may assist with the correct diagnosis.

Treatment is by medical management of the tuberculosis.

Laryngeal papillomas

Laryngeal papillomas are epithelial lesions caused by the human papilloma virus (HPV). There are more than 100 variants of HPV although only a few may be responsible for laryngeal lesions. The virus may also be related to the inverting nasal papilloma, a local aggressive lesion with a high recurrence rate. The HPV is also related to malignancies of the bladder and anogenital region. The virus may be a promoter of carcinogenesis to squamous cell carcinoma of the larynx and oral cell carcinoma of the larynx and oral cavity. HIV infected patients are at greater risk of HPV complications (eg, malignant transformation).

Papillomata may occur at all ages. Papillomata in younger patients are often numerous sessile pedunculated lesions that may recur frequently. The papillomata appear to be hormone sensitive: spontaneous remission may occur after puberty and during pregnancy. The papillomata may affect the glottic, supraglottic and infraglottic areas as well as the tracheobronchial tree. This may cause hoarseness and may compromise the lower airways.

A tracheostomy is not indicated in view of the high incidence of recurrence and implantation, notwithstanding complete removal.

Treatment

Endoscopic CO_2 laser vaporisation is the treatment modality of choice. Minimal scarring and webbing of the trachea and larynx result with laser therapy. Repeated removal may be necessary. If the peripheral bronchial tree is affected, an airway obstruction may be fatal.

The term 'juvenile papillomas' is inaccurate, this condition should be referred to as 'recurrent papillomatosis'.

Gastro-oesophageal reflux disorder (GORD)

Gastro-oesophageal reflux syndrome is a subjective symptom complex, consisting of a sensation of having a lump in the throat which causes a choking sensation in the lower pharynx. It may be associated with hypopharyngeal, substernal and epigastric discomfort.

The cause of GORD is gastric reflux into the oesophagus, which may reach the lower pharynx. The acid refluxes into the oesophagus due to an incompetent lower oesophageal sphincter: a high pressure zone controlled by the interaction of the sympathetic and parasympathetic nervous system.

The condition may be responsible for many ENT complaints such as laryngitis, tracheitis, pharyngitis, rhinits or sinusitis. The patient may complain of heartburn in the substernal area.

GORD occurs most often in middle aged to older patients, is slightly more common in females and occurs much more frequently in children than commonly thought. Pregnant women may suffer severely from acid reflux during the last trimester.

Aggravating factors may include:

- Increased intragastric pressure following large meals.

- Smoking and alcohol consumptionm that may relax the lower oesophageal sphincter, allowing gastric juices to escape into the oesophagus and hypopharynx.

- Specific foods (eg, spiced foods).

- Increased intra-abdominal pressure due to obesity or pregnancy, especially in the last trimester.

Chronic reflux has long been accepted as a major cause of Barrett's oesophagus (a chronic peptic ulcer of the lower oesophagus, often with stricture) and has been linked to a substantially increased risk of adenocarcinoma of the lower oesophagus.

Symptoms

Oesophageal reflux is diagnosed largely by the patient's history. The patient may complain of a choking sensation at the level of the hypopharynx (sixth cervical vertebra), heartburn, epigastric discomfort and a bitter taste when in the prone position.

Special diagnostic tests

Barium meals may be disappointingly negative and may reveal a coincidental hiatal hernia. The latter may still be present long after the patient is asymptomatic. The value of a barium meal is to exclude organic pathology. Gastroscopy, along with biopsy, are the diagnostic tests of choice. An inflamed oesophagus and lower hypopharynx may be apparent on gastroscopy. Oesophageal pH monitoring may also produce many false negative results. An inflamed upper trachea, especially in infants and children, is common.

Treatment

Most patients respond favourably to conservative treatment, becoming asymptomatic within weeks. The aim of treatment is to reduce the gastric acid and minimise the reflux. The following advice should be given to the patient:

- Elevate the head of the bed by a brick's height instead of using more pillows.

- Avoid large meals within two hours of retiring for the night.

- Avoid abuse of alcohol, especially spirits, which tend to relax the lower oesophageal sphincter, allowing acid to escape into the oesophagus. A beer or glass of wine is permissible.

- Keep a food diary. This will enable the identification of food or beverages that may aggravate symptoms.

- Curtail coffee intake and smoking.

- Lose weight. A concerted attempt should be made to achieve goal weight.

Pharmacotherapy

- Antiemetics before meals will promote gastric emptying and may avoid increased intragastric pressure.

- In mild cases, an antacid before bedtime may be very effective.

- In severe cases, an H_2-antagonist or proton pump inhibitor may be necessary, but can be replaced by more cost effective antacid therapy as the condition improves.

- In pregnant women with acid reflux the attending gynecologist should be consulted before any medication is prescribed.

Although acid reflux causes discomfort, it can usually be managed conservatively. Surgical management should be reserved for the recalcitrant younger patient. Rarely, a patient presents with agonising discomfort, which may mimic a myocardial infarction, but the presence of cardiac enzymes and a normal cardiograph shyould exclude this. GORD often explains symptoms experienced by patients who suffer from the so-called globus hystericus syndrome, a condition that may be cured if oesophageal reflux is controlled. The misnomer may lead to a wrong diagnosis and treatment.

Functional Aphonia

Functional aphonia (conversion aphonia) is one of multiple psychological disorders in which the patient mimics and fakes a loss of function, ie, loss of voice. The patient usually presents with a whisper.

It is common in young adult females. The cause is stress related for financial gain, for sick benefit or other reasons.

Examination reveals normal vocal cords on direct fibre optic laryngoscopy except for a bowstring of the vocal cords during an attempt to vocalise. When asked to cough, the patient has a normal cough which confirms the diagnosis.

Treatment

Refer to a psychologist or psychiatrist.

Caustic Ingestion

The damage caused by caustic ingestion may vary from mild to severe, and is often more extensive than initially estimated. Organ perforation may lead to shock and sudden death.

Incidence

In children, caustic ingestion is usually accidental, while in teenagers and adults it is often the result of a suicide attempt. In the past, South African cases were mainly from rural areas where alkaline solutions were used to clean milk cans and milking machines. Today, at least 50% of caustic ingestion cases are from urban areas. Frequently the victim is a child who was confused by dangerous substances having been stored in containers such as soft drink bottles.

Types of caustic agents

There are four categories of caustic agents:

- Detergents and cleaners (eg, soaps, bleaches and ammonia) that cause mild damage.

- Alkaline products such as drain and toilet bowl cleaners, which usually cause rapid and severe damage. A commonly ingested caustic agent is automatic dish washing detergent in granular form.

- Acid products like hydrochloric, nitric and sulphuric acids, which may also produce rapid and severe damage.

- Small batteries, such as those used in hearing aids.

General principles

A child found with an open container of any corrosive substance should be assumed to have ingested some of it until proven otherwise. If there is nothing left of the contents, the container should be examined by a poison centre. The poison centre should also be given a brief description of the patient's condition. Some centres will have rapid response vehicle available with paramedics, IV transfusion equipment and drugs on board. An airlift may also be available. In South Africa, the **Netcare poison centre** (tel: 082 911) is on 24-hour standby.

- Normal appearance of the mouth and the pharynx does not exclude the ingestion of a corrosive agent and subsequent damage.

- Do not induce vomiting or attempt gastric lavage because these could lead to fatal aspiration. Encourage (bud don't force) the patient to drink water or milk in the acute stage to dilute the caustic agent.

- Do not administer neutralising agents; these may cause more damage.

- Always admit the patient to hospital for observation.

- The patient's acid balance should be monitored.

- A chest X-ray should be taken to exclude aspiration.

- Contrast fluoroscopy studies of the oesophagus and stomach should be performed to evaluate the extent of the damage.

- Oesophagoscopy by a skilled specialist should be performed when the patient is stabilised, usually within 24 hours.

Treatment

- Administer fluids intravenously. When the patient can swallow his/her own saliva, a clear fluids diet may be prescribed.

- Administer intravenous antibiotics. Corticosteroids are appropriate in certain cases.

- Intubation and tracheostomy may be necessary in case of acute airway obstruction.

- For severe complications a multidisciplinary approach is necessary: an otolaryngologist should evaluate the upper digestive system and a general surgeon and/or thoracic surgeon assist with dilation and/or surgery and long term rehabilitation.

- Control the acid/base imbalance.

Survivors of caustic ingestion may become physically and psychologically handicapped. It is of great concern that innocent children should suffer this fate because of the negligence of adults who store corrosive chemicals in accessible places. **Caustic agents should never be stored in containers that may be mistaken by children for a pleasant cold drink.** In addition, pressure should be brought to bear on manufacturers and government, demanding childproof packaging for corrosive agents, whether in powder, liquid or granular form.

Salivary Glands

The major salivary glands are paired (ie, the parotid, submandibular and sublingual glands). The minor salivary glands are scattered throughout the oral cavity and upper respiratory tract.

The physiological function of the salivary glands is to produce adequate saliva to lubricate the food bolus for easy mastication and swallowing. Approximately 1 to 1.5 litres of saliva is produced over 24 hours. The messenger stimulation for secretion is via a parasympathetic reflex. Saliva consists 99.5% of water, the balance comprises glycoproteins, electrolytes, digestive enzymes as well as antibacterial and antifungal agents. The mucinous submandibular gland secretion is somewhat different to the serous parotid secretion.

The **paired parotid glands** are the largest of the paired salivary glands. They are bilobed and roughly pyramidal shaped, measuring 6x4x2 cm. The superficial lobe is subcutaneous. The gland is situated in the preauricular area just below the zygomatic arch onto the masseter muscle. The secretion is conducted via the parotid duct (Stenon's duct) that measures about 5 cm in length. It opens opposite the second upper molar on the buccal-oral surface.

Acute viral infection

Seasonal epidemic parotitis (mumps) is an acute febrile painful parotitis with potential, although rare, complications (ie, orchitis and pancreas involvement). The prodromal symptoms are malaise, fever, chills and anorexia.

Signs are a swollen tender parotid gland/s and a submandibular gland/s. The ear lobe is lifted forwards in the case of a parotid swelling. The parotid glands are partially embedded behind the ear lobes or singularly or in combination with the submaxillary gland/s. A single infection may leave lasting immunity. Rarely, a recurrent infection of one of the glands may recur.

Acute bacterial infections *(Kussmaul's disease)* may occur in older, dehydrated, debilitated patients with poor oral hygiene.

Pathophysiology: The saliva may become inspissated in the glands having a 'plug like' effect. Ascending bacterial infection may precipitate a purulent suppurative parotitis (or submaxillary adenitis). Rarely, a localised abscess may occur.

Chronic granulomatous lesions of the salivary glands

Sarcoidosis, tuberculosis and actinomycosis may all affect multiple organs in addition to the salivary glands. Chest and lung X-rays, sputum cultures, skin tests, smears and a fine needle biopsy of a lymph gland may assist to establish the diagnosis and the appropriate therapy. If needle biopsy facilities are not available, an incision biopsy may be warranted for diagnosis.

Benign lymphoepithelial sialadenopathy (BLS)

BLS is most common among middle aged females. Symptoms are recurrent swollen, tender salivary glands, either singularly or bilaterally. Secondary purulent sepsis may occur due to salivary stasis and pooling.

Histologically, the salivary gland/s are destroyed by infiltrating lymphocytes that replace glandular tissue. There is markedly reduced saliva secretion, hence the sicca syndrome (ie, dry eye or keratoconjunctivitis sicca) and a dry mouth (xerostomia). The aetiology is thought to be an autoimmune disease. BLS is often associated with other collagen diseases (ie, rheumatoid arthritis, scleroderma, polyarthritis and polymyositis).

Treament is palliative to combat pain and secondary infection. Surgical removal may be indicated where the gland is totally destroyed, non functional and with recurrent purulent infections.

Sjögren's syndrome was described as BLS in conjunction with lacrimal gland enlargement, keratoconjunctivitis sicca and rheumatoid arthritis (ie, a syndrome complex due to a diffuse autoimmune collagen disease involvement of multiple organs).

HIV parotitis

Patients infected with HIV/AIDS develop a similar clinical syndrome (ie, non-tender parotid enlargement along with enlargement of the salivary glands). A coexisting sicca syndrome may be apparent. Multiple loculations may be present in the parotid glands and are typical on a MRI scan.

Treatment is to correct the fluid balance, establish oral hygiene and to administer systemic antibiotics. Massaging of the gland in the direction of the duct may dislodge the inspissated mucous plugs and establish normal drainage. Surgical incision and drainage may be necessary if an abscess has developed.

Chronic adenitis is the end stage disease of recurrent purulent infections. The collecting duct systems become lobulated as seen on sialography with marked delay of mucous drainage. Usually a single gland is affected.

Therapy is conservative (ie, physiotherapy and antibiotics). Surgical removal of the affected gland may be necessary when other therapeutic measures fail.

Sialolithiasis (salivary stones)

By virtue of the nature of submandibular gland secretion (ie, a high calcium content), most salivary stones form in the submandibular gland. Symptoms are a swollen, tender gland. The pain is exacerbated by meals.

Signs

The stones may be palpated with bimanual palpation. The sialolith may be present on X-ray if the calcium content is high. Contrast sialography may confirm the diagnosis (ie, a filling defect caused by the blockage by the calculus).

Treatment is to remove the stone in the duct. Removal of the gland may be necessary if it is diffusely affected.

Tumours of the major salivary glands

The aetiology is unknown, however, familial, genetic and environmental factors may be of paramount importance. The Epstein-Barr virus and smoking are also implicated as possible causes.

Benign mixed tumours

Benign mixed salivary tumours are the most common tumours of salivary glands. The parotid gland is the most frequently affected.

A mixed salivary tumour is a slow growing, firm, painless tumour. It does not involve the facial nerve until very late. The tumour may arise from any lobe of the parotid gland. Histologically, the mixed salivary tumour has a pseudocapsule with tumour 'tentacles'

protruding through it. These are responsible for the high recurrence rate following surgical enucleation as opposed to wide excision.

Treatment is by surgical removal allowing a wide margin beyond the pseudocapsule. Recurrent tumours are difficult to remove and recur frequently.

Malignant tumours

A small percentage of head and neck cancers are malignant salivary gland tumours. Both genders are equally affected. Parotid gland malignancies account for the vast majority of major salivary gland tumours, followed by the submandibular gland. The sublingual gland is rarely the seat of malignancy. The patient may present with an asymptomatic parotid swelling.

Pain is a late symptom. Facial nerve dysfunction and neck gland metastasis are pathognomonic signs of a parotid gland malignancy. Diagnostic investigations include MRI studies and fine needle biopsies. Subclassification into histologic type of carcinoma is less important than the histological fact that the tumour is high- or low grade malignant.

Treatment

Surgery is the treatment of choice. Every effort should be made to spare the facial nerve. If the nerve has to be sacrificed, a nerve graft should be done at the time of surgery. Postoperative radiotherapy is indicated for most patients. A neck dissection is indicated only in patients with palpable metastatic neck nodes. Advanced tumours may require resection of the skin, manidble and adjacent temporal bone, which may result in considerable morbidity.

A temporary or permanent tarsorrhaphy may be necessary to protect the cornea if the facial nerve is sacrificed or grafted. Cosmetic reconstruction may restore symmetry. Survival rates are better with parotid tumours than with the other major salivary glands. Tumours of the parotid gland are histologically less aggressive.

1. Vocal fold nodules usually present at the point of maximal impact between the two vocal cords (ie, between the anterior one third and posterior two thirds of the vocal cords).

2. Multiple viral papillomas. Papillomas in younger patients are often numerous, sessile pedunculated lesions that may recur with frequency after surgical removal. Spread to the trachea and bronchi may be fatal. CO_2 laser vaporisation is the treatment of choice.

3. Reinke's oedema (chronic hypertrophic laryngitis). Reinke's oedema follows longstanding chronic laryngeal inflammation by vocal, alcohol and cigarette abuse. Vocal cord microsurgery may relieve the oedema.

4. Left vocal cord palsy. Unilateral vocal cord palsy may be transient due to a selected mononeuritis. However, a malignancy must be excluded along the pathway of the recurrent laryngeal nerve.

5. Functional aphonia. Functional aphonia results in a whispering voice. Laughing and coughing is normal and is diagnostic of this condition. Psychotherapy may ameliorate the situation. Indirect laryngoscopy reveals a bowstring closure of the vocal cords.

6. Vocal dystonia. Spastic vocal cords may produce a choking or strangulated voice quality. Intralaryngeal botulinum toxin may relieve the situation and a normal but softer voice may result. The effect may only be temporary.

7. Intubation polyp. An intubation polyp may follow subacute trauma to the arytenoid cartilage as resulting from prolonged intubation or a 'crash' intubation. A recurrence after removal is likely and the patient should be monitored.

8. TB laryngitis. TB laryngitis appears as shallow irregular granulous lesions with a necrotic base. Typically these lesions appear on the posterior larynx and on the inter arytenoidal fold. A TB granuloma may appear as a solitary lesion and may simulate a malignancy.

9. GE reflux. The acid refluxes into the oesophagus due to an incompetent lower oesophageal 'sphincter', a high pressure zone controlled by interaction of the vagus and the sympathetic nervous system. Chronic reflux is linked to a substantially increased risk of adenocarcinoma of the oesophagus.

10. Gastric lavage. Do not promote vomiting; fatal aspiration or rupture of the oesophagus may occur.

11. Salivary glands. Of the three main paired salivary glands the parotid glands are the largest. The submandibular glands are the second largest and the sublingual are the smallest.

12. Acute viral parotitis (Mumps). This is an acute painful parotitis with the potential, although rare, complications of pancreatitis, meningo-encephalitis and orchitis in males.

13. Sjögren's syndrome. The parotid glands are the most commonly affected with painless enlargement on both sides. The facial nerve is not involved. The eyes are 'dry' due to involvement of the lacrimal glands and a loss of tear production. Systemic involvement (ie, rheumatoid arthritis) may follow.

14. Submandibular salivary stones. These are frequently due to high calcium content of the gland secretion.

15. Benign salivary tumours. Benign mixed salivary tumours are the most common salivary gland tumours. The parotid gland is the most frequently involved. The facial nerve may only be involved at a later stage. The tumour is painless.

16. Carcinoma of the parotid gland. A parotid tumour associated with facial nerve palsy and a neck gland is pathognomonic of a parotid malignancy. Surgical excision is the therapy of choice; additional radiotherapy may be warranted in selected cases. Cosmetic

reconstruction may be considered at a later stage.

Miscellaneous

Geriatric ENT Conditions

Of the five senses, hearing is the first to suffer from ageing. Presbyacusis is the natural failure of hearing with advancing years and is caused by degenerative changes in the inner ear. In youth, the inner ear can readily perceive frequencies above 10 kHz. Starting in the 20s this function is progressively curtailed so that by the 60s it may be difficult to perceive sound at 6 kHz and above.

This loss of high frequency perception takes its toll on everyday communication. Words are only partially heard and hence are not fully understood. In particular, the soft non vocal consonants, such as S, T, P and V (plosives and fricatives), which are of high frequency (between 4.5 kHz and 6 kHz) are not heard. Because plosives and fricatives delineate the beginning and end of words, the geriatric patient will perceive 'stop street' as 'top treat'. This confuses the patient and the patient starts to miss out on information. Embarrassment, social isolation and depression may follow.

Consciously or subconsciously these patients begin to depend on lip-reading and body language to supplement speech discrimination. This maybe a disadvantage if they cannot see the speaker's face (eg, in poor light or in a car).

The following points may help with old age hearing loss, ie, presbycusis:

- A hearing aid fitted and correctly tuned may dramatically improve hearing.

- When talking to the person, keep in mind to face them and clearly enunciate words - don't shout!

- Consciously attempt to speak in a lower register.

Presbytinnitus

Tinnitus in the aged is usually of a continuous high pitch, simulating the sound of a ciccada (roughly 3500 kHz) or a hiss. The aetiology may be earlier noise exposure, frequently seen in war veterans whose service was with the artillery.

More serious causes of tinnitus such as tumours or cerebral lesions must be excluded. Once the patient is assured that the tinnitus is not a sign of something dangerous, it is often more easily accepted.

Tinnitus maskers may be helpful in treating this distressing problem. They can be ear level maskers or in-the-room maskers (eg, noise generators). Ambient noise generators may displace sound in the head to an extracranial position; psychologically more acceptable to the patient. Commercially available tinnitus maskers feature a selection of sounds (eg, waterfall, rain on a tin roof, or splashing waves). However, if the patient wishes to record other suitable

sounds, these provide an equally effective solution. All that is needed is that they be adjusted to a comfortable audible level, especially when going to sleep.

A hearing air or an ear level tinnitus masker may also be beneficial and should be selected to suit the needs of the individual user.

Tinnitus habituation seems to be most promising in otherwise refractory cases. The worst that can be said to the patient is that "nothing can be done".

Presbyvertigo

This is an annoying sensation of an instable gait accompanied by a falling and whirling sensation. The integration of peripheral information from the vestibular and visual systems needed to maintain balance, as well as from the proprioceptive organs, may be centrally mismatched giving rise to insecure gait, especially in the dark. Falls and other accidents may give rise to a variety of injuries or even fractures. Treatment may include psychotherapy and labyrinthine sedatives.

Patulous external ear canal

The external ear canal may become patulous. This leads to poor wax excretion and wax accumulation which often makes it difficult to fit a hearing aid. This can be corrected surgically.

Wax accumulation

Over the years, wax may become impacted deep in the ear canal and even onto the tympanic membrane by do-it-yourself attempts to remove it with a cotton bud. Wax forms in the outer third of the ear canal and is moved outward by the movement of the jaw and hairs in the outer ear canal. This is counteracted if the wax is impacted into the ear canal.

Wax may easily be removed by using a wax curette. For more deeply impacted wax it may be necessary to syringe the ear canal. People should be discouraged from using ear buds. Hearing aid use also interferes with the movement of wax out of the ear and may lead to wax impaction. This, in turn, interferes with the effectiveness of the hearing aid.

Hearing aid users should routinely have their ear canals checked for wax impaction which may lead to insidious hearing loss.

Iatrogenic afflictions

Large mastoid cavities often accumulate wax and harbour low grade infection with a smelly discharge. They may be difficult to keep clean and dry and will need regular cleaning by an otologist.

Malignancies

Skin cancer

The sun's ultraviolet rays strike the skin on the ear helix and nasal bridge at right angles, delivering maximal radiation damage. Hence, years of solar exposure may introduce basal and squamous cell carcinomas of the pinna and nose. A small carcinoma may be successfully treated with simple exicion and closure. The specimen must always be forwarded for histological examination. Larger excisions may necessitate skin flaps and skin grafting, usually from behind the ear for colour and texture matching.

Upper respiratory tract tumours

Years of smoking and abusing alcohol may induce malignancies in the upper respiratory tract and oral cavity. **Persistent hoarseness may be the first and only symptom of a curable vocal cord carcinoma.**

Pharyngeal carcinoma

This may present with odynophagia, dysphagia, emaciation and referred ear pain via the ninth and tenth cranial nerves. Hence, the old, emaciated male who presents with drooling from the mouth and cotton wool in his ear to combat the earache shows pathognomonic signs of an upper aerodigestive carcinoma. At a later stage, involvement with metastatic neck nodes may occur.

Early referral may be voice- and life-saving for many sufferers of pharyngeal or laryngeal carcinoma. The earlier the referral, the less morbidity and the less the mortality. **Unnecessarily prolonged conservative therapy for such cancers misdiagnosed as 'a sore throat' is a waste of a golden opportunity for early eradication and control of the disease.**

Blocked nasal airways

Hypertensive therapy may induce vasomotor rhinitis causing a chronic blocked nose and result in a chronic sinusitis syndrome.

Nasal polyps may also be present, compounding the issue of a blocked nasal airway. The blocked nose is usually more severe in the recumbent sleeping position. Common complaints are: mouth breathing, dry lips, dry mouth, dry throat and snoring. Pharyngitis sicca and frequent attacks of purulent sinusitis may also be prominent. Psychological nose drop abuse may aggravate the condition leading to rhinitis medicamentosa.

The following points may help the patient with blocked nasal airways:

- Change the antihypertensive agent (eg, from a betablocker to a calcium blocking agent).

- Gradually discontinue the use of nose drops. Psychological dependence may already have established itself. Treatment of the underlying nasal obstruction is necessary.

- Refractory cases may dramatically improve with nasal surgery.

Epistaxis

Epistaxis is predominantnly from arterial bleeding of the septum caused by forceful nose-blowing. Dry, warm air accentuate the problem due to crusting of the nasal septum.

A number of older patioents may bleed from the roof of the nasal vault, especially if associated with hypertension, atherosclerosis and certain medications (eg, aspirin).

Epistaxis from the anterior septum may be chemically cauterised with silver nitrate under topical anaesthesia. Electrocautery is rarely necessary.

For inaccessible bleeds, control the hypertension with sedation. Control the blood clotting factors. Use expandable cellulose tampons. Specific vessel ligation under general or local anaesthesia is occasionally required. Percutaneous embolism of the bleeding vessels may be performed under direct X-ray visualisation.

1. Cottonwool in the ears usually means otalgia and, when associated with drooling, is suggestive of a tongue or pharyngeal malignancy and this possibility must be excluded.

Medical Lasers

Laser is an acronym for Light Amplification by the Stimulated Emission of Radiation. Albert Einstein in 1917 postulated the theoretical foundation of laser action. In the early 1960s laser became a reality and was commercialised. A laser beam delivers the highest intensity of a mono-colour light beam known to man. The size of the spot, intensity and penetrating depth can be accurately controlled. There are various types of lasers (ie, gas, crystal or semiconductor lasers) for different applications.

Commercial applications of lasers are in supermarket bar code scanners, lecture pointers, telecommunications and compact disc players.

Medical lasers are divided into two classes, surgical and therapeutic lasers. In therapeutic lasers the beam is 'scattered'. The laser energy is spread over a larger area and penetration may be between 1-4 cm. Therapeutic lasers are used in the ENT field to speed up wound healing and 'bio-stimulation' may assist to control pain when applied to inflamed temporomandibular joints, may reduce trigeminal neuritic pains, or may speed up recovery from Bell's palsy and in the treatment of chronic sinusitis.

In surgical lasers, the beam is focused on a small spot to produce heat of several hundred degrees Celsius. The intense heat casues tissue evaporation at the spot of the interaction with body tissue. A small crater is produced with a few flakes of carbon debris.

The lateral spread of the heat is limited by using short, intense bursts of laser energy. Tissue haemorrhage, oedema and scar tissue formation is minimal. Surgical lasers are used in the ENT field for the removal of skin tumours, upper aerodigestive system tumours (benign and malignant), middle ear ossicular reconstruction (ie, stapedotomy) and recurrent laryngeal papillomata.

Universal safety precautions

Hospitals that offer laser surgery should appoint a laser safety officer and a laser safety committee. Universal safety protocols should be strictly adhered to by all theatre staff and attending doctors. The laser surgery staff should be educated about the following potential dangers:

- The eyes of the surgeon, anaesthetist, theatre staff and of the patient are at risk if laser use is not controlled. The laser beam may reflect or bounce off any metallic surface and injure the eye. Yet, the exposure maybe painless. Spectacles are adequate protection frmo a CO_2 laser and should be mandatory for all present in the theatre. The patient's eyes should be protected with wet pads or drapes.

- Teeth may be damaged and should be protected by wet gauze covers.

- Facial skin should be protected by moistened double layer saline-saturated surgical drapes. The drapes must be kept moistened to prevent accidental ignition.

- The endotracheal tube should be protected with moistened gauze to prevent accidental ignition of the tube or mouthpiece. The oxygen content of the anaesthetic gasses should be as low as possible. Special laser resistant endotracheal tubes may provide extra protection.

- The most important safety factor is that surgical and theatre staff should be aware of the properties and dangers of laser.

1. Surgical lasers minimise tissue trauma by limiting lateral heat spread. Tissue oedema and scar tissue formation are minimal.

HIV/AIDS in ENT

AIDS is a comparatively recent manifestation in medicine and was first recognised as a human disease syndrome as recently as the early 1980s. The daunting facts are that the ripple effects of infection will only be felt in the early part of the 21st century with major implications on global manpower and health costs.

The virus is classified as a retrovirus. Retroviruses have the capacity to integrate into the host cell genetic material (chromosomes) **causing lifelong** infection in the host.

Pathogenesis

The virus has a tropism for cells bearing the CD4 receptor molecule. These cells are T-helper lymphocytes and macrophages. The predominant organ system involved is the lymphoid system (lymph nodes) and over time there is a progressive loss of CD4 lymphocytes, both in the peripheral blood and lymph nodes.

The outcome of the depletion of the CD4 lymphocytes is a depressed cell-mediated immunity that allows the occurrence of opportunistic infections and opportunistic diseases, especially TB. The reason for this is the reactivation of the dormant TB focus (Ghon's focus) that is otherwise held in check by normal cell mediated immunity.

In addition the virus is found in nerve tissue (eg, the brain), and various brain dysfunction syndromes may be encountered (eg, muscle palsies and dementias).

Transmission of HIV is by blood and blood products, sexually, and from mother to child either during the birth process (intrapartum) or by breast feeding. A small percentage of cases of mother to child transmission are transplacental.

The predominant global transmission is by sexual contact. A **major breakthrough** in the reduction of mother to child transmission is the use of antiretroviral therapy given during the last few weeks of pregnancy, during labour and for some weeks postpartum. This has been shown to greatly reduce the transmission rate.

In mothers who are HIV positive and when socio-economic circumstances permit, it is advised to substitute formula feeding for breast-feeding because of the increased risk of viral transmission through breast milk. So far mosquito transmission of HIV has not been shown to occur.

Opportunistic diseases and infections that occur as a result of immunodeficiency in the field of ENT include:

- Fungal disease (ie, *Candida, Cryptococcus* and *Histoplasmosis* of the sinuses, oral cavity, pharynx, larynx and trachea).

- Recurrent bacterial upper respiratory infections (eg, otitis media, sinusitis, pharyngitis and stomatitis).

- Chronic **drug resistant infections** of the sinuses, middle ears, pharynx and oral cavity with overwhelming fungal and TB infections.

- Viral infections (eg, Herpes simplex, Herpes zoster or papilloma viral disease in the respiratory tract).

- Parasytic infestation (ie, toxoplasmosis of the soft tissue of the head and neck, especially the tongue and neck or intracranially).

- Tumours (ie, AIDS-related Kaposi sarcoma of the oral cavity, sinuses or pharynx).

- Hodgkin's and non-Hodgkin's lymphomas of the head and neck area.

Any of the above lethal conditions can impact on the organs mentioned at **atypical ages.**

Prevention

Because of the increased prevalence of HIV infection, universal precautions should be implemented when treating patients in whom HIV infection is suspected. Needless to say, sterilisation of instruments must be stringently adhered to, for the protection of patients.

Healthcare workers

Healthcare workers should be protected by wearing glasses and or a full-face shield or face mask. The ENT surgeon is placed in a vulnerable position in front of a patient during an examination.

The eyes may easily be infected by an opportunistic infection when accidentally exposed to a cough, sneeze or even when the doctor is speaking to an infected patient. HIV infection may take place when blood splashes in the eyes.

Opportunistic infection may occur through droplet spread. Liquid droplets from the mouth may spread several metres where they may be inhaled by susceptible bystanders and possibly spread TB.

Occupational hazards for doctors have increased alarmingly since the advent of AIDS. In institutions where HIV patients are treated, healthcare professionals and workers should annually be screened for TB.

Treatment

For adults or children infected with HIV, multiple drug treatment may prolong life. The viral load may be suppressed below 50 virus particles / mL of blood, thus improving the patient's well-being and quality of life. A moral issue remains regarding sexual transmission by those already infected. Although the patient's general health condition may be improved with therapy, the patient may potentially still transfer the HIV/AIDS virus and infect a sexual partner.

An effective vaccine has not yet been developed. Paradoxically the AIDS virus targets and destroys the very cells that are responsible for immunity (ie, the CD4 lymphocytes).

The CD4 cels are helper T-lymphocytes (CD4T cells) and form part of the cell mediated immunity system.

1. The ENT Surgeon is placed in a vulnerable position in front of a patient during examination. Healthcare workers must protect their eyes when examining HIV infected patients. The eyes are vulnerable when exposed to an accidental cough or sneeze by a patient infected with an opportunistic infection.