

## Chapter 33

### The Teeth and Gums, Jaws, Nose, Ear.

#### Congenital and Developmental Anomalies of the Teeth

**Abnormalities of number and size.-** One or more teeth may fail to develop. The ones most often absent are the third molars and the upper lateral incisors. Many teeth may be missing from both the primary and secondary dentition in partial anodontia or all may fail to develop in total anodontia. Such individuals may also lack adult hair, sebaceous and sweat glands, and are suffering from ectodermal dysplasia.

*Supernumerary teeth* may be either smaller and more simple in form than normal, often conical, or resemble the adjacent teeth of the normal series; so called supplemental teeth. Supernumerary teeth between or behind the upper central incisors (mesiodens) are often conical, those in the lower premolar region tend to resemble normal premolars. Fourth molars may develop behind the lower third molar or behind or lateral to the upper ones. Teeth of the normal series may be smaller or larger than normal and with fewer or extra cusps or roots.

#### Odontomes and Odontogenic Tumours

The more severe developmental abnormalities of tooth form are called odontomes. Dentine and enamel may be invaginated into the pulp chamber and root canal to produce invaginated odontomes like the dens in dente and the dilated odontome.

Invaginated odontomes are important as their pulps may become infected and the source of an abscess. A compound odontome is a hamartoma composed of an encapsulated cluster of small denticles, many of which are minute so that when the tumour is dissected out a surprising number of denticles are uncovered. A complex odontome is composed of an irregular mass of prismatic enamel, tubular dentine and pulp. Many other hamartomas and benign or even malignant neoplasms of dental tissues, the odontogenic tumours, are recognised. Most are uncommon or very rare and require the opinion of clinicians and pathologists expert in this field for their identification, and management. Only the ameloblastoma and odontogenic myxoma will be described here.

**Ameloblastoma.-** This neoplasm develops from the dental lamina. Typically, cords of odontogenic epithelium ramify in a cellular connective tissue. Follicles are formed with stellate reticulum-like cells within and a single layer of columnar ameloblast-like cells at the periphery. These cells undergo reversal of polarity but neither induce the formation of odontoblasts nor deposit enamel. Cysts may form either in the stellate reticulum (epithelium lined cysts) or within the stroma. Thus, this neoplasm may form a solid, soft tissue tumour, a single large cyst with a small solid component, or a multilocular cystic mass which is the typical type. It may enclose the crown of an unerupted tooth and resemble a dentigerous cyst. Many involve the third molar region extending into the coronoid process, angle and body of the mandible. Less frequently the anterior part of the mandible is involved and rarely the maxilla. Patients may present at any age from around 11 years upwards, but most present in the 4th and 5th decade. It is slow growing and painless unless infected. This neoplasm is locally invasive within medullary bone and soft tissues and should be excised with about a

1 cm margin at these sites. Subperiosteal excision is satisfactory where the overlying bone is intact and of reasonable thickness. There is evidence that the tumour cells implant readily and it should not be cut into or fragmented at operation. If an ulcerated surface is handled, gloves should be changed. It does not metastasise to lymph nodes, but will do so to the lungs, usually after incomplete local excision. Long standing tumours can reach a large size. It is usually possible to reconstruct the mandible with an iliac crest bone graft at the time of resection. Diagnosis of histologically typical lesions from a biopsy is not difficult but there are cases presenting problems which require special expertise. Local recurrence can follow enucleation or curettage and has been reported as long as 15-20 years after the initial operation. Recurrence involving the base of the skull or tissue planes of the neck may occur after inappropriate surgery. These neoplasms are not radiosensitive.

**Odontogenic Myxoma.-** These comparatively uncommon jaw tumours also have a predilection for the angle of the mandible though they may be encountered more anteriorly in the mandible or maxilla. They are composed of stellate connective tissue cells in a myxomatous stroma. They are lobulated and produce a bone cavity which radiographically has a well defined polyarcuate periphery and which is crossed by straight ridges or septa. Like the ameloblastoma it can penetrate the adjacent medullary spaces and once it involves the surrounding soft tissues either during natural growth or as a result of fragmenting surgery can be next to impossible to eliminate. If incorrectly handled on the first occasion the patient usually suffers multiple operations for recurrence over many years. These neoplasms also are not radiosensitive.

### **Abnormalities of Structure**

Acute infections during childhood may affect the deposition of enamel and dentine, especially those which disturb nutrition. Enamel defects are seen as hypoplastic pits in the crowns of all teeth which are developing at that time. They form a line across the crown corresponding to the stage of development of the enamel at the relevant age. The condition is called linear hypoplasia. A special abnormality results from congenital syphilis which, while rarely seen these days, remains diagnostically important. The width of the incisal edge of the upper central incisors and often one or more of the lower incisors is less than normal to produce a screwdriver or barrel shape. The enamel in the centre of the incisal edges may be defective, so forming a notch. The notched incisor with a narrow incisal edge is called a Hutchinson's incisor. It must be distinguished with care from acquired notching of an otherwise normal incisor, due for example, to biting cottons to cut the thread. Hutchinson's incisors may be recognised radiographically before eruption. The length and width of the occlusal surface of the first molars is also less than average so that the cusps are closed together, forming the bud molar. If the enamel is also pitted with hypoplastic pits the teeth are described as mulberry molars.

If water which contains 1 part per million of fluoride is drunk during the time of tooth development, enamel is laid down which is less readily attacked by the acid produced by plaque organisms. This amount of fluoride does not affect the appearance of the enamel, but does reduce the incidence of dental caries. Drinking water containing significantly greater concentrations of fluoride during the development of the crowns of the adult dentition produces a whitish enamel with a rough surface which takes up brown stains to produce an unsightly mottled appearance.

Ingesting additional fluoride as well as fluoridated water may produce minor degrees of mottling. Topical fluoride applied to the enamel as fluoridated toothpaste or as solutions or gels by dentists also reduces the incidence of caries and may aid the recalcification of the initial lesions of caries in the enamel.

There are a variety of inherited abnormalities of structure of the enamel or forms of amelogenesis imperfecta or of the dentine; dentinogenesis imperfecta. For example the enamel may be hypocalcified, but of normal thickness or pitted, or consist of no more than a thin layer. The latter form may accompany severe epidermolysis bullosa. In dentinogenesis imperfecta the enamel tends to separate from the dentine which in its inner layer has relatively few dentinal tubules. The pulp chamber and root canals are greatly narrowed even before the apices are fully formed. A similar abnormality of the dentine is seen in osteogenesis imperfecta.

### **Impacted and Unerupted Teeth**

If there is insufficient room in the arch for a tooth, or if it is tilted, impaction may occur. Those teeth which erupt after their neighbours such as canines, premolars and third molars tend to become impacted. Supernumerary teeth may impede the eruption of adjacent teeth of the normal series, notably the upper central incisors, or may themselves fail to erupt. Failure of eruption may result from severe crowding and impaction or from a disturbance of the eruption mechanism such as a failure of alveolar bone growth. Both tooth development and eruption are arrested at a chronological stage of development, corresponding to the onset of the disease, in hypoparathyroidism and a failure of many teeth to erupt in both the primary and secondary dentition is typical of cleido-cranio-dysostosis.

The most frequent tooth to be impacted is the lower third molar. Partially erupted lower third molars cause problems in two age groups: either in teenagers and young adults or in the edentulous or partially edentulous elderly when previously buried teeth are uncovered as a result of alveolar atrophy. Impacted lower third molars may be the site of a pericoronal infection. Caries either of the wisdom tooth itself or of the second molar at the point where the tooth is impacted against it may result from food stagnation and result in alveolar abscess formation. Several types of cyst (see below) or odontogenic tumour such as the ameloblastoma (see above) are associated with lower third molars. Impacted wisdom teeth also create a weakness in the jaw so that during accidents fractures tend to occur which involve their sockets.

### **Pericoronitis**

Once an erupting tooth has penetrated the overlying tissues a potential cleft exists between the enamel surface and the adjacent follicular soft tissues. In the majority of cases the epithelial attachment remains intact and eruption is completed quite rapidly so as to achieve a normal gingival crevice. If this process is disturbed an actual cleft can be established around the crown of almost any tooth which potentially can become infected, though in practice pericoronitis is virtually confined to third molars and lower third molars in particular.

If the lower alveolar process is too short to contain the third molar, or if it becomes impacted, a gum flap or operculum partially covers the incompletely erupted tooth. An acute or subacute infection can involve this hood of gum. Such an infection may be suppurative so that pus oozes from under the flap. A pericoronal abscess can form which may track forward along the buccinator origin to discharge into the buccal sulcus opposite the lower first molar. More seriously the infection may spread into the adjacent tissue spaces such as the buccal space laterally, or the pterygoid and parapharyngeal spaces medially. If it reaches the submasseteric space it may form a submasseteric abscess and cause a superficial osteomyelitis of the ramus of the mandible. Should infection spread to the submandibular lymph nodes these in turn may suppurate and produce a submandibular abscess. Fulminating infections of these spaces are dangerous, particularly a parapharyngeal abscess. If a submandibular infection spreads via the sublingual spaces backwards medial to the hyoglossus to the epiglottis a Ludwig's angina results. Both parapharyngeal infections and Ludwig's angina threaten to obstruct the airway and can cause the death of the patient.

Vincent's acute ulcerative gingivitis may start under a pericoronal gum flap (ulcerative pericoronitis)(see Chapter 32).

Patients with acute pericoronitis present with soreness in the lower third molar region, with pain which often radiates to the ear, swelling of the cheek, limitation of jaw opening and enlarged and tender submandibular lymph nodes. The severity of the illness varies between wide limits.

*Treatment of pericoronitis.*- Mild infections may respond to irrigation under the gum flap with Eusol and the introduction of Povidone iodine under the flap. Treatment is repeated daily together with the frequent use of hot salt water mouth washes. Where there is persistent pain, cheek swelling, lymph node enlargement or limitation of opening, amoxycillin, metronidazole or tetracycline should be given for five to seven days. If there is a pericoronal abscess the flap is incised lateral to the third molar to establish drainage. Often the upper third molar bites on the gum flap. If it does it should be extracted. Extraction of the lower third molar, and certainly one needing surgical removal is deferred until the acute infection has completely subsided. A Vincent's acute ulcerative pericoronitis is treated with metronidazole.

### **Alveolar abscess on a lower third molar**

If the wisdom tooth can be extracted without the surgical removal of bone this should be done straight away. If there is an abscess pointing it is incised. Removal of a third molar which can be accomplished only by bone surgery should be deferred and the infection controlled by an antibiotic such as amoxycillin. If the infection is spreading into the soft tissues antibiotics should be added even if the tooth can be extracted without difficulty.

*Removal of lower third molars.*- Where the bone around the tooth is young and elastic it may be removed with chisels, making use of a predictable direction of split. This requires skill and practice. The bone may be removed precisely in all age groups with No 10 and No 6 round dental burs in a straight dental handpiece. An incision is made *over the external oblique ridge* behind the third molar and a short vertical relieving incision added later to the second molar. Mucoperiosteal flaps are elevated buccally and lingually to the tooth and a Howarth's rougine (W G Howarth, 1879-1962. London otolaryngologist.) is placed under the

lingual periosteum to protect the lingual nerve. Bone is cut away, buccally and behind the third molar and, if necessary, lingually until the greatest diameter of the crown is uncovered. A trough is cut lateral to the tooth down to the bifurcation of the roots. Another through may be cut behind to create space into which the tooth can be tilted to disimpact it. If the crown of a horizontally placed tooth presses tightly against the second molar in front it is cut off using a No 6 fissure bur. If the roots lock the tooth in place the crown is cut off and the roots separated with a fissure bur so that they can be dislodged individually. All cutting with a bur is done under a trickle of sterile saline applied with a syringe. Impacted teeth may be elevated out as shown. The socket is washed with saline and the flaps sewn together.

### **Dental Caries**

Certain oral organisms proliferate on the surface of the teeth and their adhesion is aided by the precipitation of sticky dextrans to form plaque. If sucrose, or refined carbohydrates which are readily converted to sugar by ptyaline are eaten they are metabolised by plaque organisms to produce high local concentrations of lactic acid. The underlying enamel is attacked and eventually penetrated to the dentine. The dentine is invaded by organisms capable of proteolysis which reduce it to a brown, soft, crumbly consistency. The process spreads laterally under the enamel until the force of mastication causes the overlying enamel to fracture, producing an obvious cavity. It also spreads inwards, following the dental tubules until the organisms reach and destroy the pulp. Pulp necrosis may also follow trauma to a sound tooth, mostly a front tooth, either because the crown is fractured or because the blow jerks the tooth and tears the apical vessels. Infection may also reach the pulp via imperfections in enamel in the depths of invaginated odontomes. The latter are rare, but minor degrees of invagination at the cingulum are relatively common in upper lateral incisions.

**Periodontal disease.-** Where it is in contact with the gingival margin, toxins from organisms in plaque can cause inflammation which, by a process which also involves certain immune reactions, results in damage to the epithelial attachment. The underlying periodontal membrane fibres and then the alveolar bone are also attached. First a chronic marginal gingivitis results and then a pocket between gum and tooth as the periodontal membrane is progressively destroyed. The deeper layers of the plaque becomes calcified by calcium salts found in solution in saliva to form calculus. A bacteraemia results whenever teeth affected by periodontal disease are chewed upon. Organisms from the pockets are also forced into the blood stream as an affected tooth is extracted.

### **Alveolar Abscess**

Alveolar abscesses may result from the spread of infection from a necrotic pulp into the periapical tissues to produce a periapical abscess. Resorption of the overlying cortical bone occurs to permit the pus to escape into the adjacent soft tissues and mostly to point in the adjacent buccal or labial sulcus. On occasions organisms produce an abscess apical to the bottom of a periodontal pocket. Such a periodontal abscess forms another type of alveolar abscess which may discharge through the pocket or through the overlying alveolar tissues so as to point on the side of the alveolar process.

**Emergency treatment of toothache.-** Dental caries is not obvious in the early stages as the breach in the enamel, which occurs either in the depths of an occlusal fissure or where

one tooth is in contact with the neck, is quite small. Not until a large dentinal lesion is close to the pulp and where the overlying enamel has broken away does the cavity cause pain and then only when hot, cold or sweet food is in contact with the cavity stimulating the pulp. Such pain passes within a minute or two.

With a double ended enamel chisel, weak enamel around the cavity opening can be chipped away to enlarge it. The soft carious dentine can be scraped out gently with a spoon excavator and a dressing inserted. Zinc oxide powder is mixed with one or two drops of eugenol or oil of cloves using a small knife as a spatula to form a stiff paste and a lump pressed into the cavity.

If the pain resulting from pulp stimulation persists or comes on spontaneously the pulp is likely to be inflamed. After gently excavating caries a soft dressing can be pressed in, made by mixing zinc oxide with carbolized resin and a few fibres of cotton wool. This may well relieve the pain until expert help is available. If the patient has a throbbing pain an antibiotic is prescribed. Should an abscess point in the sulcus it should be incised and drained for which little operation a topical spray with ethyl chloride to freeze the gum will provide sufficient anaesthesia. Analgesic tablets are prescribed for the pain.

### **Spreading Infection from an Alveolar Abscess**

Strangely osteomyelitis of the jaws is an unusual complication of a periapical abscess (see below), but the infection may well spread in the surrounding soft tissues rather than point in the adjacent sulcus. Infection from lower teeth spreading lingually may cause a cellulitis of the sublingual space. From apical abscesses on second and third molars perforation of the lingual plate occurs below the mylohyoid muscle with spread into the submandibular space. Buccal spread from molars in either jaw can involve the buccal space and posterior spread to the pterygoid space between the pterygoid muscles and the medial side of the ramus. Upwards spread from the pterygoid fossa carries the infection into the infratemporal fossa and then by way of emissary veins to the cavernous sinus. Posterior spread from an abscess on a lower molar may also result in a parapharyngeal abscess with swelling of the lateral pharyngeal wall and soft palate. There is difficulty in swallowing and later in breathing. Respiratory obstruction may threaten in neglected cases or the infection may spread via the carotid sheath to the mediastinum. Bilateral sublingual and submandibular space infections constitute Ludwig's angina. Backward spread around the sublingual vessels results in oedema of the epiglottis and respiratory obstruction. Abscesses from upper canines discharging into the cheek beneath the levator labii superioris travel up to point below the medial corner of the eye. If neglected, the infection can cause thrombophlebitis of the angular vein and cavernous sinus thrombosis. The apex of the upper lateral incisor is closer to the palatal than the labial cortex and causes a palatal abscess while pus from a lower third molar may travel back beneath the masseter as a submasseteric abscess. It strips the periosteum from the bone and results in necrosis and even sequestration of the outer cortex of the ramus. The swelling of a submasseteric abscess does not raise the lobe of the ear and results in complete limitation of opening; features which distinguish it from a suppurative parotitis. Lymphatic spread carries infection from the lower incisors and canines or a sublingual cellulitis to the submental lymph nodes where secondary suppuration can produce a submental abscess. Infection from all the other teeth drains to the submandibular nodes where lymph node suppuration can result in a submandibular abscess.

*Signs, Symptoms and Treatment.*- An early pulpitis causes a sharp pain exacerbated by hot or cold food or drink. The pain becomes throbbing and then subsides as the pulp necroses.

With the onset of an acute alveolar abscess a deep, boring, throbbing pain develops with a soft, puffy swelling of the overlying lip or cheek (collateral oedema). The causative tooth becomes acutely tender to percussion and mobile. As pus nears the surface of the bone the periosteum and parosteal tissues become oedematous, firm and tender and there is redness of the sulcus mucosa. When pus bursts into the soft tissues the pain eases for a while but returns as an abscess form in the sulcus. The overlying soft tissues become more swollen and the skin a pinkish red. If a sinus forms, the pus discharges and the acute symptoms subside. Many pulps necrose without symptoms and a chronic periapical granuloma forms about the apex with local destruction of bone apparent in an x-ray. An acute exacerbation of a previous chronic infection results in the early onset of throbbing pain and swelling.

If a cellulitis supervenes in the overlying tissues the swelling becomes more severe and indurated with the skin a brighter red colour, the lymph nodes become more swollen and tender and severe constitutional symptoms appear. The change to a dusky redness of the overlying skin in the centre of the swelling, the appearance of localised increased tenderness and pitting oedema, together with a sharp rise in temperature herald the formation of pus beneath the deep fascia. Incision and drainage should be undertaken at this stage and should not await the appearance of a fluctuant swelling beneath the skin. Amoxycillin, flucloxacillin, tetracycline and metronidazole are antibiotics to which the organisms are likely to be sensitive.

Where there is a swelling of the floor of the mouth, which raises the tongue, or a parapharyngeal swelling, the induction of general anaesthesia should be undertaken by an expert because of the risk of respiratory obstruction, and intubation and drainage are conducted in the head down position. Sublingual abscesses are incised lateral to the lingual plica and pointing parapharyngeal ones may be opened without general anaesthesia. A dental mouth prop is inserted between the teeth, the tongue sprayed with topical anaesthetic and depressed with an angled spatula, the mucosa immediately over the swelling is injected twice with 0.5 mL 2% lignocaine with 1:80,000 adrenaline and the abscess opened with a No 11 scalpel blade and the pus immediately sucked away. In the case of genuine Ludwig's angina a tracheostomy should be undertaken under local anaesthetic before respiratory obstruction threatens. The traditional deep incisions dividing the muscles of the upper neck are damaging and do not help the outcome. Incisions made in both submandibular regions permit the submandibular, sublingual and parapharyngeal spaces on both sides to be drained as necessary by appropriately directed sinus forceps using Hilton's method (John Hilton, 1804-1878. Surgeon, Guy's Hospital, London.). Long corrugated rubber drains are inserted. Submental pus requires a separate transverse skin incision between hyoid and chin.

The causative tooth should always be dealt with unless bone surgery is necessary for its removal. Incised abscesses will continue to drain until the source of the infection has been removed. Chronic apical abscesses may be dealt with by tooth extraction or root canal therapy.

Where the tooth is valuable a dentist may drain an acute apical abscess via the root canal and subsequently root treat and fill the tooth. Attempts to save the tooth should be abandoned if a spreading infection supervenes. Most sizeable periodontal abscesses require the extraction of the involved tooth.

*Poultices and sinuses.*- Poultices do not benefit most face and neck infections and by inducing skin redness and oedema may make the recognition of the signs of pus formation difficult. They are helpful where enlarged lymph nodes associated with an acute infection are slow to resolve despite the continued administration of an antibiotic which controls the pyrexia and constitutional symptoms. Pus should never be permitted to discharge through the skin spontaneously or an ugly scar will result. Where a spontaneous skin sinus has appeared and drained before the patient is seen the relationship to a particular tooth may not be obvious as the facial muscles determine the site at which it points. Often the sinus track can be felt as a fibrous cord beneath the sulcus and leading to the bone. Pus from the lower incisors can breach the bone below the origin of the mentalis muscle. If so the abscess reaches the surface between the two muscles and drains via a sinus in the midline of the chin. Curiously the source of this lesion is often not recognised and it may be mistaken for an infected sebaceous cyst or even a skin malignancy. Concern is heightened as local excision is always followed by its reappearance. Extraction or root treatment of the offending incisor results in the sinus drying up and healing.

An ugly, chronic, facial sinus scar should not be excised for six months after the causative tooth has been dealt with. Many will improve and be more cosmetically acceptable in this time. Those that do not, need skilful excision if the surgical scar is to be an improvement upon the sinus scar. An ellipse of skin around the sinus is removed in the line of skin creases. The fibrous cord is cored out and the track closed with deep sutures. The skin is undermined and closed carefully in layers.

If there is obvious periapical bone destruction due to a chronic abscess or granuloma to be seen in a radiograph, this may enable the causative tooth to be identified. Periapical films are best for this but oblique lateral views may suffice. Failure to identify the tooth by this means does not exclude a tooth as a cause. Tests for pulp vitality or removal of existing restorations may be needed to find the culprit.

### **Surgically Important Complications of Dental Disease**

Loose or grossly carious teeth which may be dislodged or fractured, should an anaesthetist use a laryngoscope, are best removed before an operation and, if the socket is sutured, need not delay it. Once such an accident has happened it may be difficult to prove the extent of the disease which made dislodgement of the tooth likely. Patients should always be questioned about crowns and bridges and the anaesthetist warned of their presence. Replacement of damaged crowns and bridges can be costly.

The need, where appropriate, to introduce Povidone iodine solutions into gingival crevices or pockets and for the administration of antibiotics for the prophylaxis of infective endocarditis is well known. Artificial heart valves may become infected by a bacteraemia not only after extractions but by mastication upon teeth involved by periodontal disease. As well as the risk of infection of a valve post-operatively there is the need to manage extractions in

an anticoagulated patient should an alveolar abscess occur at this time. Diseased teeth therefore should be dealt with pre-operatively before heart valve replacement. Similar risks of haematogenous infection are present where joint replacement has been undertaken and again are best anticipated.

If any patient presents for elective surgery an inspection of the oral cavity may reduce the risk of future problems, particularly if a prolonged anaesthetic will be necessary. Patients with generally dirty mouths and much calculus should be advised to visit a dentist for a scale and polish and to improve their oral hygiene. Those with many carious teeth and marked periodontal disease should be told to seek treatment before they are admitted.

Post extraction bleeding occasions considerable alarm on behalf of the patient. Mostly the haemorrhage is reactionary in nature, starting two to three hours after the extractions. Occasionally it is secondary and occurs several days later, perhaps as an accompaniment of a Vincent's ulcerative gingivitis. In certain instances it is not the socket which is bleeding but an incision in the sulcus, made to drain an alveolar abscess. The mouth is carefully cleaned of clot with gauze swabs and the site of the haemorrhage identified. A folded gauze swab or a roll of gauze bandage is placed accurately on the bleeding socket and the patient instructed to bite upon it continuously for 15 minutes.

During this time an enquiry is made of relatives, if present, for any evidence suggesting a major defect in the haemostatic mechanism. It is not unknown for haemophiliacs in pain to conceal their problem and persuade a dentist to extract a tooth! Mostly the cause is local, not general. 3/0 (3 metric) black silk on a 22 mm half circle cutting needle or similar suture material, is put out, a syringe and needle and 2% lignocaine containing 1:100.000 adrenaline, a needle holder and stitch scissors. If at the end of 15 minutes haemostasis is not complete, and the clot stable, the socket should be sutured, 0.5 mL of local anaesthetic solution is injected into the sulcus laterally and into the floor of the mouth or palate medial to the socket. The adrenaline will reduce the haemorrhage temporarily and the anaesthesia permits deliberate unhurried suturing. A bite of labial or buccal gum and then of lingual gum is taken and the suture tied with a surgeon's knot. The two turns in the first throw will prevent the knot slipping and enable the suture to be tied lightly. Each bleeding socket is sutured in turn and then the patient bites on a swab again. Failure of haemostasis after suturing is rare. Should it happen a small amount of resorbable oxydised cellulose can be inserted in the socket under the suture.

Some patients experience great pain starting in a socket around the third day after an extraction. The gum margin around the socket is red and swollen and the socket empty of clot or filled with debris. The clot has been lost either as a result of infection or by the action of plasmin to produce a 'dry socket'. Daily irrigation of the socket with eusol, a prescription for metronidazole and one for an analgesic is effective though the patient's dentist can provide more rapid relief with socket pastes.

There are some patients in which the maxillary sinus dips down between the roots of the upper premolars and molars. Granulation tissue about diseased teeth may destroy bone between the tooth and the antrum such that a fistula results when the tooth is removed. Sometimes the tooth mechanically grips the adjacent bone which fractures away as the tooth is extracted. In other cases as bone is removed to expose a retained root the sinus is

uncovered. If this is noticed the opening will be closed at the time but infection in the antrum may lead to breakdown of the repair. In the first instance the exodontist may not be aware of the opening until the clot in the socket breaks down. Infection in the maxillary sinus must be controlled then one of a variety of small repair operations is performed dependent upon the defect.

Occasionally a tooth root is displaced into the antrum. Usually the dentist is aware of the accident and takes measures to retrieve it. In certain circumstances he may not know this has happened and the root can be a cause of chronic sinus infection.

### **Osteomyelitis of the jaws**

Despite how often periapical abscesses occur, osteomyelitis of the jaws is not common, and it is the mandible which is usually involved. In the majority of cases, infection spread from a local focus directly into the bone but metastatic spread from osteomyelitis or an abscess elsewhere in the body is not unknown. There is usually some local or general factor which tips the balance. For example, acute osteomyelitis affecting large segments of the mandible is seen more often in members of malnourished communities. Extraction of the offending tooth is the most effective way to both decompress an intra-bony periapical abscess and also to remove the source of infection. Failure effectively to treat an apical abscess on a lower molar tooth can lead to involvement of the mandibular neurovascular bundle interfering with the blood supply to the medullary bone. That this is a factor leading to osteomyelitis of the mandible is suggested by the frequency with which the onset of anaesthesia of the lip marks the change from a localised to a spreading bony infection. A traumatic extraction in the face of an acute infection may also result in osteomyelitis. If a fracture of the mandible, compound into the mouth, is not immobilised at an early stage and the mucosa closed over the bone end, infected saliva is sucked into the wound whenever the bone ends move relative to one another. Infection of the bone ends soon follows. Gunshot wounds produce many devitalised fragments of bone which, if not removed and the adjacent viable ones covered with soft tissue, soon become grossly infected.

*Chronic osteomyelitis* occurs where there is bone sclerosis. Sclerosis may develop at the periphery of a segment of mandible affected by acute osteomyelitis. The local reduction in blood supply impedes the natural defences of the body and also the ingress of systemic antibiotics. Some diseases such as Paget's disease (Sir James Paget, 1814-1899. Surgeon, St Bartholomew's Hospital, London.) result in the production of patches of sclerosed bone which if exposed at the bottom of a tooth socket may become infected. Incurable osteomyelitis of the mandible can follow tooth extraction in marble bone diseases.

*The course of osteomyelitis* of the mandible is similar to that in other bones. Acute osteomyelitis is accompanied by swelling of the overlying soft tissues, a boring, throbbing pain, malaise and pyrexia. The gum over the affected segment is swollen and the contained teeth become mobile and tender. Pus may reach the surface via sinuses which penetrate the cortex and the overlying subperiosteal new bone which forms the involucrum and discharges on to the face or into the mouth. It may also point via the periodontal membranes of the teeth. Once pus has discharged the disease enters a less painful, chronic phase when the patient is comparatively well except during flare ups in the infection. Infection involving sclerotic bone

masses is often chronic from the start. Sequestra composed largely of cortical bone may be discharged spontaneously, but often require surgical removal.

*Management.*- Provided effective antibiotic therapy is started early, for example as soon as mental anaesthesia appears, frank osteomyelitis involving large parts of the mandible will not develop. Amoxycillin and flucloxacillin in combination are given and persisted with for 4-6 weeks unless investigation of the pus, once it is advisable, indicates another antibiotic.

Subperiosteal abscesses should be drained and loose teeth surrounded by pus, or grossly carious teeth which might be a source of infection should be extracted if this can be easily accomplished. Later, once sequestra have separated, they are surgically removed. Chronic osteomyelitis may require saucerisation of the affected bone back to healthy bleeding medulla or the removal of sclerotic bone masses; preferably once they have been demarcated by bone resorption.

*Acute osteomyelitis of the maxilla of infancy* is a well recognised entity. It starts in the neonate in the first few weeks of life and produces gross swelling of the eyelids so that it may be mistaken for orbital cellulitis. Antibiotics should be given promptly. If pus points it is likely to be below the medial corner of the eye or over the as yet unerupted second deciduous molar tooth germ. A small incision should be made to effect drainage. No attempt should be made to remove infected bone surgically unless an obvious sequestrum presents. Bone surgery will lead to unnecessary later deformity. Hypoplasia of the enamel of the related primary teeth may be seen when they erupt and the maxilla may fail to reach the size of the unaffected side. Osteomyelitis of either jaw may be seen in childhood mainly in the poorly nourished and is accompanied by the formation of masses of subperiosteal new bone.

*Therapeutic irradiation* damages the blood vessels within bone and impairs the bone cells' ability to divide and effect a repair in the event of injury. Extraction of teeth from a part of the jaw previously irradiated to therapeutic levels carries the risk of post irradiation osteomyelitis; so called radionecrosis. There is also a risk if the soft tissues are stripped from part of the bone, or if bone is left bare after an injury.

Following irradiation of a tumour of the perioral tissues there is a fibrinoid reaction and ulceration of the mucous membrane in the irradiated field. The patient finds tooth cleaning difficult and the natural cleansing due to chewing is reduced as mastication is painful. Salivary flow is also reduced, sometimes permanently. There is periodontal tissue atrophy and the vulnerable necks of the teeth are exposed. As a result even patients with previously well cared for mouths can suffer rampant caries. Where this is likely extraction of the teeth in parts of the jaws which will be heavily irradiated is advised before treatment begins. Tuberculous and actinomycotic osteomyelitis and gummas involving bone are conditions rarely seen these days.

**Infection of the Temporo Mandibular Joints.**- Suppurative arthritis may occur as a complication of acute osteomyelitis of the mandible, of acute mastoiditis where the air cells involve the eminentia articularis or where middle ear infection, sometimes as a complication of measles, discharges between the cartilaginous and bony meatus into the joint. Much of the growth in length of the mandible occurs in the region of the condyle and infection of the condyle in childhood can lead to restriction in growth of that side of the mandible.

## Lumps on the Gum

Gingival enlargement may be *generalised* (hyperplastic, hypertrophic gingivitis) or *localised*. Discrete lumps on the gums are known by the traditional name of epulides. Where the upper alveolar process is prominent and the upper lip short the patient may keep the lips apart at rest. The exposed gingival margin becomes dry and chronically inflamed. A pocket is formed between the swollen gum and the enamel of the tooth crown (false pocketing). With time a degree of fibrosis occurs rendering the enlargement permanent. Hormonal changes during adolescence may play a part in the onset of hyperplastic gingivitis. Phenytoin prescribed to control epilepsy will produce generalised gingival hyperplasia though there is some evidence that this does not occur if meticulous oral hygiene can be maintained. Nifedipine, a coronary vasodilator, also may cause the condition.

Pale, swollen gums, often with purple patches where there has been submucosal haemorrhage may be the presenting complaint in cases of acute leukaemia. The most dramatic hypertrophy is seen in the familial condition, fibromatosis gingivae where the enlargement may be so gross that the teeth, though fully erupted from the bone are buried in the enlarged gums.

Provided the cause of the gingival enlargement can be controlled or treated the excess tissue can be removed, the false pockets eliminated and the gum recontoured by the operation of gingivectomy. (Marked improvement may occur following the elimination of causative factors and the establishment of effective oral hygiene so gingivectomy should be delayed until no further improvement occurs.) As well as abolishing the pockets, a bevelled gum margin is re-established. The raw wound surface is covered by a special protective paste (Coepack). A gingivectomy may be used to treat false pocketing or cases of periodontal disease with moderate pocketing. Where bony recontouring is also necessary flap procedures with apical repositioning of the mucoperiosteum to eliminate the pockets is used.

**The fibrous epulis** is a localised inflammatory hyperplasia of gum. It arises in response to local irritation, from the sharp margin of a carious cavity or the presence of subgingival calculus. It often arises from an interdental papilla. At first it is soft and red, but becomes firm and pink as more collagen is deposited in the central mass. It is often pedunculated though the pedicle may be so short that at first sight the epulis appears sessile. The majority of fibrous epulides cease to enlarge after attaining about 1 cm diameter. A few become considerably larger, ulcerate as a result of trauma during mastication and can resemble a malignant neoplasm at first sight. Simple excision, local gingival recontouring and the application of a gingival pack is curative provided the source of irritation is also dealt with; otherwise the epulis will recur.

**Pregnancy Epulis and Pyogenic Granuloma.**- In adolescence, during pregnancy and sometimes in older individuals of both sexes soft, rapidly enlarging lumps appear on the gums. Like the fibrous epulis they arise at sites of local irritation, are generally pedunculated and usually start as an enlargement of an interdental papilla. In children they may be related to small, sharp fragments remaining after a primary tooth has been shed. Unlike the fibrous epulis they remain soft, pink and vascular. In pregnant women the irritant may be relatively insignificant deposits of calculus and the unusual response is thought to be conditioned by the hormonal changes in pregnancy. These inflammatory hyperplastic tumours are termed

pregnancy epulis or pregnancy tumours. Indeed the gum margins may be generally red and swollen and, as a result, bleed easily during the last months of pregnancy. The patient should be encouraged to see her dental surgeon and to improve rather than neglect her oral hygiene if she develops a pregnancy gingivitis. Similar tumours occurring in males and non-pregnant women are called pyogenic granulomas. Histologically all these lesions are composed of a cellular, vascular connective tissue and if ulcerated many inflammatory cells are present. Pregnancy tumours tend to regress after the child is born. Because they are vascular and haemorrhage after excision can be troublesome, their removal may best be undertaken with a special dental diathermy machine with a unipolar cutting electrode.

**Giant Cell Epulis.-** These purple, pedunculated tumours are also probably inflammatory hyperplasias. They are less common than the fibrous epulis or the pyogenic granuloma and may arise adjacent to an infected socket or the site of a shed primary tooth. For these epulides local excision should include curettage of the bone surface. Any local irritant must be dealt with. Histologically they resemble the intrabony giant cell granuloma.

**Denture Induced Granuloma.-** (See above.)

**Fibrous hyperplasia of the Tuberosities.-** The mucoperiosteum of the tuberosities is thickened particularly on the palatal aspect. The enlargement may be considerable so that the sessile hemispherical masses almost meet in the midline.

### **Cysts of the Jaw**

A variety of cysts occur in the jaws and those which are likely to be encountered by general surgeons will be described. In most instances the cyst is composed of a fibrous capsule covered on the inner aspect by epithelium. The two layers are together referred to as the lining of the cyst.

**Periapical, Radicular or Dental Cysts.-** These develop at the apices of teeth with necrotic pulps. They are lined by stratified squamous epithelium derived from the epithelial debris of Malassez (Louis Charles Malassez, 1862-1910. Parisian surgeon and pathologist. Described epithelial rests in the periodontal membrane in 1885). Once formed these cysts enlarge slowly causing resorption of the adjacent bone and then expansion of the jaw. If left untreated they can become large, involving the greater part of the body of the mandible, or most of one side, including the ramus. In the maxilla they tend to enlarge to fill the maxillary sinus and inferior meatus of the nose before causing visible expansion and radiographically must be distinguished from carcinoma of the antrum.

**Dentigerous Cyst.-** Dentigerous cysts arise as a result of separation of the reduced enamel epithelium from the surface of the crown of an unerupted tooth and the accumulation of fluid in the interval. They enlarge in a similar manner to periapical cysts except that they displace the teeth to which they are attached. The tooth is normally displaced deeper into the jaw and prevented from erupting by the cyst. The cyst lining is usually attached around the neck of the tooth so that the crown protrudes into the cyst cavity. Sometimes only the side of the crown is adjacent to the cyst cavity. Unerupted upper third molars seen in a radiograph displaced up to the orbital floor are usually involved in dentigerous cysts.

*Treatment.*- Both periapical and dentigerous cysts may be treated either by enucleation of the lining from the bony cavity and primary closure of the surgical wound or marsupialisation. If the cyst is marsupialised a sizeable opening is made in both the thin bone covering the cyst and the lining and the surgical flap of oral mucosa turned in to meet the cut edge of the lining. Initially the cavity is packed. Later a special plug may be needed to keep the opening patent. Once decompressed the cavity will fill in. Large maxillary cysts occupying much of the maxillary sinus can be treated by stripping out the cyst lining and removing the partition between the bony cavity and the antrum. The oral wound is closed carefully.

In the case of periapical cysts the causative dead tooth must be dealt with either by extraction or root treatment. Sometimes it is possible to retain the tooth involved in a dentigerous cyst and encourage it to erupt. There are a substantial number of technical details relating to these procedures with which the operator should be familiar to ensure success.

**Keratocysts.**- Keratocysts have a thin layer of keratinised epithelium on their inner aspect. The lining has a number of distinctive features recognised by the specialist histopathologist. These cysts arise from residual strands of epithelium from the dental lamina. Some arise between standing teeth and others posterior to the third molar, in the base of the coronoid process. Cysts in these locations are also called primordial cysts. Others arise from the epithelial debris of Serres (Etienne Reynaud Augustin Serres, 1786-1868. Professor of Anatomy and Natural History at Jardin des Plantes, Paris.) (ie, dental lamina remnants immediately above the crown of the tooth) and envelop the tooth as they enlarge and so are radiographically indistinguishable from a dentigerous cyst. However, these cysts remain separated from the crown of the tooth by the tooth follicle so are called extrafollicular dentigerous cysts. Keratocysts may arise singly or from multiple foci of dental lamina. They may do this in a group to form a multilocular cyst or individually in several places in both jaws. In just under half there are proliferations of the lining epithelium into the fibrous capsule and daughter cysts may form in this way. There is a tendency for these cysts to recur after operation in about 40% of cases. The lining is often very delicate, easily torn and any left behind will start a new cyst. A daughter cyst may be retained (particularly if microscopic) or new cysts may form from the epithelial rests of an adjacent or another part of the dental lamina. Painstaking enucleation to avoid damage to the lining and to ensure its complete removal reduces the recurrence rate and careful conservative treatment is usually adequate. Any recurrences or new cysts arising in patients afflicted with multiple cysts are dealt with in the same way. Occasionally a patient with a large cluster of keratocysts is seen. Such highly multilocular cysts are not easily dealt with conservatively and resection of the involved segment of jaw and its replacement by a bone graft is to be preferred. Care must be taken not to fragment the specimen as recurrence in bone grafts has been reported, probably from particles of lining which have seeded in the wound.

**Multiple cyst-basal cell naevus syndrome.**- There is a syndrome affecting systems and which is often inherited the first presenting complaint may be of keratocysts of the jaws which start to appear as the adult dentition erupts. Palmar and plantar pits, milia around the eyelids, basal cell naevi and true basal cell carcinomas develop in the skin during later life. Multiple subcutaneous epidermoid cysts may also appear. A short skull base, a prognathic mandible, bridging of the sella turcica, calcification of the falx, cervical spina-bifida and bifid

ribs are some of the skeletal anomalies which may be found. Many other defects have been reported as occasional features.

**Naso-palatine cysts.-** These cysts develop in the incisive canals to cause a spherical bone cavity behind the upper central incisors. They are composed of a fibrous capsule lined on its inner aspect with epithelium, but while in some this is stratified squamous epithelium in others it is respiratory in nature, with columnar cells, mucus secreting cells and ciliated cells. Nasopalatine cysts rarely achieve a large size but may get big enough to produce a swelling in the midline of the palate or bulge the labial alveolar plate. They arise from epithelium of the nasopalatine ducts. They are treated by reflecting a palatal flap and stripping out the lining.

**Naso-labial cysts.-** Arise outside the bone of the maxilla but may cause a depression in its surface. They lift the ala of the nose, flatten the upper part of the naso-labial fold, form a fluctuant swelling in the labial sulcus and bulge into the inferior meatus of the nose. They may arise from the upper part of the epithelial fin or from epithelium trapped between lateral nasal and maxillary elevations as they merge. Like the nasopalatine cysts they tend to be lined by respiratory epithelium and often contain a mucoid liquid. The sac of a nasolabial cyst is dissected out through an incision in the mucosa of the upper buccal sulcus.

**Solitary Bone Cyst.-** These resemble the solitary bone cysts of long bones. Most occur in the mandible and in the premolar-molar region rather than the incisor region or ramus. Initially the bone cavity is round or oval, but it bulges outwards in a lobulated fashion, leaving ridges on the walls. In particular it loops up between the roots of adjacent teeth. Expansion of the jaw occurs late on with the covering sub-periosteal bone also exhibiting ridges on the cavity surface. There is no dissectable lining, but a thin layer of connective tissue over the cavity surface of the bone. Histological examination of this reveals limited bone resorption and even bone deposition. Haemorrhage readily occurs from the thin walled vessels in the wall if aspiration is attempted. Careful aspiration of large examples while a second needle admits air reveals a yellow fluid containing high bilirubin levels. If blood is aspirated and centrifuged the supernatant fluid will be yellow with bilirubin. Unlike the fluid from other jaw cysts it will clot. Removing part of the bony wall and suturing the soft tissue flap back into place will stimulate these cysts to heal in adults, but they may recur during childhood or early adolescence. They may represent a local aberration of bone growth.

*Cyst fluids.-* Aspirations can help to differentiate jaw cysts. Periapical and dentigerous cysts contain a clear or brownish fluid containing cholesterol crystals. Keratocyst fluid is creamy white like pus, but odourless, while that from nasopalatine and nasolabial cysts may be glary or mucoid.

*Complications of Jaw Cysts.-* A swelling of the face or of the jaw inside the mouth will occur as the cyst enlarges. Pressure against the roots of adjacent teeth displaces them sideways. Dentigerous and extrafollicular dentigerous cysts prevent the eruption of the involved tooth, but the eruption of adjacent unerupted teeth may be obstructed by any cyst which develops in the jaws of a young person. The cyst contents may become infected and if the cyst is large this can be potentially serious. Obviously a large cyst weakens the mandible and pathological fracture may occur.

## **Fibro-Osseous Jaw Tumours**

A variety of conditions which produce enlargement of one or other jaw show the histological appearance called 'osteitis fibrosa'. Histological examination reveals replacement of the bone by a cellular osteogenic fibrous tissue. Woven bone is deposited in the fibrous tissue in a three dimensional spongework. Radiographically this has a ground glass, finger print or orange peel pattern, but in histological sections irregular, thin trabeculae are seen likened to Chinese figures. More mature lesions often contain lamellar bone, others a concentrically laminated, basophilic calcified tissue resembling cementum. A thin layer of sub-periosteal new bone covers the lesion where it expands the jaw.

**Fibrous dysplasia of bone** can affect the bones of the jaw as a monostotic lesion, as part of polyostotic fibrous dysplasia, or as a component of Albright's syndrome (Fuller Albright, 1900-1969. Physician, Massachusetts General Hospital, Boston, Mass, USA.). The swelling may enlarge rapidly during the adolescent growth spurt and the resulting deformity cause concern. Surgery at this age may result in recurrence and an enhanced speed of growth. Enlargement usually slows down once skeletal growth is complete when trimming will produce an acceptable result. If surgery becomes essential during adolescence subperiosteal resection of the lesion should be the aim with immediate reconstruction with a bone graft. Some examples of fibrous dysplasia in young patients contain scanty deposits of woven bone and present as a lobulated, largely radiolucent lesion in the bone, with ridges on the inner aspect of the surrounding bone giving it a coarsely trabeculated appearance. Diagnosis prior to biopsy may be difficult as this radiographic appearance is common to a variety of jaw tumours.

**Ossifying and cementifying fibromas.-** Fibrous dysplasia responds to some of the normal controls of bone growth, and normally has an ill defined periphery radiographically in the jaws, except in the case of the markedly radiolucent form. The ossifying fibroma is a benign neoplasm which can resemble fibrous dysplasia histologically or may feature the spherical cementum-like clumps of tissue described above. These lesions remain circumscribed but enlarge progressively. The cementifying variant particularly can reach a large size but is usually easily enucleated, though care may be required to maintain continuity of the jaw.

**Paget's disease** can affect the jaws. Most often it is the maxilla which is involved. In the early stages both the histological and radiographical features of osteitis fibrosa are seen. In more mature lesions the typical cotton wool sclerotic patches appear. These are of more mature lamellar bone and histologically show the mosaic pattern characteristic of the disorder. Hypercementosis of the teeth makes them difficult to extract. The bone is very vascular and a brisk haemorrhage follows surgery, including tooth extraction. As mentioned above dry sockets and infection of the adjacent sclerosed bone, which is only slowly sequestered, is a complication. Facial deformity and difficulty in the wearing of dentures due to enlargement of the ridges can require trimming operations. These benefit the patient for a while but enlargement often recurs. When the maxilla is affected, the lesion progresses through the face into the vault of the skull. Paget's disease of the mandible is less common than of the maxilla and the mandible can be the only bone affected for many years. Paget's disease affecting both jaws is rare. Benign giant cell granuloma-like lesions (see below) and osteosarcoma are other rare but important complications.

## Giant Cell Lesions of the Jaws

**The giant cell granuloma.-** Histologically this lesion bears some resemblance to the osteoclastoma of long bones, but is quite benign. The tumour has a stroma of plump, connective tissue cells, scanty collagen, many thin walled blood vessels and a considerable number of osteoclast-like giant cells. Many histiocytes can be found, scattered through the lesion. Histologically therefore it resembles the giant cell epulis and the brown tumour of hyperparathyroidism. For the most part the giant cell granuloma occurs centrally in the jaw, either in the mandible or in the maxilla. It forms a lobulated tumour which consequently produces a ridged cavity within the bone. Where it erodes through the cortex it is covered by a thin layer of sub-periosteal new bone which, in the occlusal radiograph outlines the lobulated surface, distinguishing the condition radiographically from a cyst which would produce a hemispherical expansion. The roots of adjacent teeth are displaced, or often markedly resorbed. A subperiosteal variant of the giant cell granuloma is seen in children. A biopsy will distinguish it from a malignant neoplasm which often is the first impression of the clinician. It must not be confused with a giant cell epulis as the underlying bone involvement is often greater than at first appears. Removal of the superficial soft tissue mass only will leave much of the tumour behind.

Because a giant cell granuloma is indistinguishable from the brown tumour of hyperparathyroidism and as the latter may develop before the more generalised bone changes are obvious, this diagnosis should be excluded before local surgery is undertaken. Rapid and alarming enlargement of a giant cell granuloma may take place during pregnancy. Careful enucleation of a giant cell granuloma with primary closure of the mucosal flap will normally effect a cure. Gentle curettage of the cancellous bone of the cavity wall will ensure no peripheral lobules are left behind. Unnecessary damage to unerupted teeth should be avoided during curettage of the subperiosteal type.

**Osteoclastoma.-** A malignant osteoclastoma occurs only rarely in the jaws. Diagnosis rests mainly on the histological features. Rapid enlargement and substantial destruction of the jaw can occur both with the giant cell granuloma and brown tumour of hyperparathyroidism.

**Aneurysmal bone cyst.-** Presents with a similar clinical and radiographic appearance to the giant cell granuloma. It forms a soft, sponge-like tumour centrally in the jaw which oozes blood until enucleation is complete. A cellular fibrous stroma forms septa around blood filled spaces with groups of giant cells here and there in the tissue.

**Cherubism** is inherited as a dominant gene with variable penetration. The lesions of cherubism contain many osteoclast-like giant cells and in early childhood resemble the giant cell granuloma. Later they become tough and fibrous, with a gradual reduction in the number of giant cells, then spicules of woven bone appear. Typically, lesions appear bilaterally in the angles of the mandible during the first year of life. In the more severe cases further lesions appear both anteriorly in the mandible and in the posterior part of the maxilla. Both jaws may be diffusely involved and substantially enlarged. The bulging cheeks pull down the lower lids to show the sclera below the cornea, so the child appears to look upwards, hence the name of the condition. During adolescence the maxillary lesions regress and the mandibular ones follow suit by the mid-twenties. Cosmetic trimming only is required. Extensive lesions interfere with tooth development and eruption and even erupted teeth may be lost early.

**Endosteal Haemangioma** not to be confused with aneurysmal bone cyst. Both endosteal cavernous haemangioma and arterio-venous malformations can be encountered. They may produce no more than local gigantism of the affected bone, or a distinct intra bony tumour, which on account of the radiographic appearance may be mistaken for a cyst or giant cell granuloma. Large examples are dangerous and catastrophic haemorrhage can follow extraction of overlying teeth or surgical exploration.

### **Tumours of Bone**

**Osteomas** normally present as pedunculated, rounded lumps on the surface of the jaws. Both cancellous and ivory osteomas are encountered. Multiple osteomas of the mandible, particularly at the angles, are a feature of Gardner's syndrome (E J Gardner, Contemporary American geneticist.) which is inherited as a dominant gene. Other features include: osteomas of the frontal bone, multiplying polyposis of the colon and rectum in middle age with malignant change, leiomyoma of the stomach and desmoid tumours in surgical scars.

Symmetrical rounded sessile exostoses on the lingual side of the mandible in the premolar region and in the mid line of the palate are called mandibular and palatal tori and do not usually enlarge further in adult life; they are developmental rather than neoplastic.

**Osteogenic sarcoma** rarely affects the jaws. A full therapeutic dose of irradiation followed by radical resection is the treatment and carries a better prognosis than osteogenic sarcoma of the long bones.

**Chondrosarcoma** of the jaws is also rare and tends to be misdiagnosed as a chondroma so that the chance of cure by radical surgery is allowed to slip by.

### **Malignant Tumours of the Mandible**

Squamous cell carcinoma is the commonest malignant neoplasm to involve the mandible. It may arise in the mucosa covering the alveolar process when it almost immediately invades the underlying bone. Alternatively advanced carcinoma of the floor of the mouth, cheek or tongue may spread to involve the adjacent bone. Rarely intra bony primary squamous cell carcinoma may develop from cell rests of the dental lamina or the lining of cysts.

*The treatment of squamous cell carcinoma when involving the mandible* used to be entirely surgical because bone reduces the local dose of ortho voltage irradiation below an effective level because high doses give a risk of radionecrosis of the jaw. Mega voltage irradiation however is now much more effective in controlling invading bone, and in skilled hands there is a lower incidence of bone necrosis. For large tumours, combined radiotherapy and surgery is used and surgery alone, of course, for recurrence after radiotherapy.

*Surgical Procedures.*- When carcinoma spreads from an adjacent tissue to involve the mandible superficially, the opposite cortex can be conserved to maintain continuity of the jaw. Once the cancellous bone is substantially involved segmental resection is necessary. In general it must be assumed that spread along the inferior dental canal has occurred and the bone from

proximal to the mandibular foramen to beyond the mental foramen should be excised. Medial spread via the periosteum and mylohyoid muscle can occur so the latter needs to be removed down to the hyoid. Generous removal of soft tissues around the ulcer is essential and a block dissection of neck added if nodes are palpable.

It is not essential to replace the mandible for resections short of the mid-line, but soft tissue repair with appropriate flaps is necessary for acceptable function and appearance. Delto-pectoral (DP) and pectoralis major myocutaneous flaps are often used, sometimes with a de-epithelialised strip to permit repair of both the inside of the mouth and the skin surface. A forehead flap can be combined with a DP flap to repair both surfaces. Where the mental region also is excised replacement of the mandible either by a temporary implant or by bone is essential to permit suspension of the larynx. Free flaps anastomosed by microvascular techniques to local arteries and veins can be used to replace soft tissues and as compound flaps, both soft tissues and vascularised bone.

Secondary neoplasms, such as carcinoma from other sites, or in children from adrenal medullary neuroblastoma are occasionally seen. Lymphomas, 'histiocytosis X' and myeloma may present as central mandibular tumours. Osteogenic and other sarcomas are seen, though not very frequently, as also as a rare occurrence, are malignant odontomes. Treated vigorously with radiotherapy and radical surgery, osteogenic sarcoma has a better prognosis than in the long bones.

### **Malignant Tumours of the Maxilla**

Tumours of the upper jaw are rare in the UK. They include the following pathological types:

1. Squamous carcinoma.
2. Adenocarcinoma. This may occur as an occupational disease in woodworkers.
3. Miscellaneous tumours such as transitional cell carcinoma, tumours of salivary gland origin, sarcoma and melanoma. Burkitt's lymphoma presents as a rapidly growing tumour of the maxilla and mandible (Chapter 9).

*Clinical features.* Initially the carcinoma is symptomless. Obstruction to the ostium and infection of secretions or ulceration of the neoplasm leads to symptoms suggestive of chronic sinusitis. Epistaxis may be an early symptom. In radiographs at this time thickening of the lining is seen, but often no evidence of bone destruction and the seriousness of the case not appreciated. A histological diagnosis can be obtained by intranasal antrostomy or a Caldwell-Luc operation (see maxillary infections below), and all surgical specimens from these procedures must be examined histologically even if the clinical evidence suggests chronic inflammatory disease.

Once the bony walls are breached the clinical presentation depends upon the direction of growth:

1. *Downwards through the floor.* Pain in the related teeth due to involvement of nerves and the loosening of teeth due to destruction of their supporting bone is followed by expansion of the alveolar process, and the fit of dentures may be upset. Necrosis of the tumour leads first to ulceration and then a large sloughing antro-oral fistula.

2. *Medial enlargement* results in fungation into the nose. A blood-stained mucoid or foul-smelling, purulent discharge results. Obstruction of the ostia of the other nasal sinuses leads to a unilateral pansinusitis. Obstruction to the nasolacrimal duct leads to epiphora. All polypoid growths in the nose must be histologically examined to avoid missing a malignancy.

2. *Antero-lateral spread* leads to pain in the cheek with anaesthesia of the cheek and anterior teeth and gums. An obvious swelling of the cheek occurs with ultimate ulceration and fungation of the tumour through the skin.

4. *Superior spread* results in pain and possible epiphora. Involvement of the external ocular muscles causes diplopia and gross invasion of the orbit results in proptosis.

5. *Posterior spread.* This is the most sinister as significant symptoms may not occur until invasion of the base of the skull has occurred. Limitation of mandibular movement due to involvement of the pterygoid muscles (trismus), pain and anaesthesia of cheek, tongue and lower lip, from invasion of the maxillary, lingual and inferior dental nerves and ultimately a bloody post-nasal discharge from ulceration into the nasopharynx all indicate advanced disease.

Metastasis to the regional lymph node occurs comparatively late on. Either the sub-mandibular or upper deep cervical nodes or both are usually involved.

*CT scanning. Tomography.*- Once the bony walls are invaded bone destruction will be seen on x-ray but CT scans in two planes will demonstrate more clearly the acute extent of the tumour. Where CT scanning is not available conventional tomography should be used.

*Treatment.*- Where curative treatment is the aim, preoperative megavoltage radiotherapy is given in full dose. Four to six weeks later, when tissue reaction has subsided, operation is performed.

*Surgical Procedures* (specialist journals should be referred to for detail).- If the tumour is free of the palate and is mainly antroethmoidal it may be successfully removed by a lateral rhinotomy approach. Involvement of the palate or walls of the antrum require a maxillectomy. If the growth extends into the orbit or anterior cranial fossa, an extended procedure is required such as a craniofacial resection (with good cure rates).

*Lateral rhinotomy.*- A vertical incision down the side of the nose exposes the nasal bones and the medial aspect of the maxilla. Removal of the lateral wall of the nose gives excellent exposure of the antrum and ethmoids allowing good clearance of tumours not involving the palate. If the tumour is found to involve the orbit or anterior fossa additional exposure via the anterior fossa allows en-bloc (craniofacial) resection.

*Maxillectomy.*- The maxilla is exposed by raising a facial skin flap along the lower lid margin, down the side of the nose and through the middle of the upper lip and then along the upper buccal sulcus. The intra-oral incision is completed along the midline of the palate and then laterally to separate the hard and soft palates. The maxilla is then freed with osteotomies from the skull base. Medially the hard palate is split in the midline, the frontal process of the maxilla and the lamina papyracea is divided. Laterally the osteotomy through the zygoma extends to the interior orbital fissure. The final osteotomy is made posteriorly detaching the pterygoid plates. The incision is closed and the defect left by the removal of the maxilla is filled with an immediate dental obturator. This results in surprising little deformity either cosmetically or functionally for such a radical resection.

Palliation following maxillectomy can often be prolonged by use of cryosurgery or laser ablation to suspect areas.

*Spread to Lymph Nodes in the Neck.*- Where lymph nodes in the neck are invaded a block dissection is required. If the subcutaneous tissues of the cheek are invaded to any degree it may be wise to include a triangle of soft tissues down to the submandibular nodes, so as to include the lymphatics around the facial vessels.

### **Infection of the Maxillary Antrum**

**Surgical Anatomy.**- The maxillary antrum (of Highmore) (Nathaniel Highmore, 1613-1685. General Practitioner, Sherborne, Dorset, England. Published a treatise on anatomy in 1651.) is rudimentary at birth and attains full development by the age of twelve years. Lined with ciliated epithelium, it communicates with the middle meatus of the nose by a small ostium situated high on its medial wall. The apices of the roots of the second premolar and the first and second molar teeth are in close apposition to the floor of the antrum being separated only by periosteum and mucous membrane. Rarely, the first premolar and the canine teeth are related similarly.

**Maxillary Sinusitis** may be unilateral or, much less frequently, bilateral. Infection occurs as an extension from the nose. One epidemic of acute respiratory infection (common 'cold') brings as an aftermath many cases of infection of the accessory nasal sinuses, of which the maxillary antrum always heads the list; while another epidemic, in all other respects similar, is free from this complication. A less frequent cause is penetration of the floor of the antrum by bacteria from a periodontal abscess connected with a carious teeth; also entry of infected water during swimming.

**Acute Sinusitis.**- As a rule the general symptoms are severe, especially when the pus is confined by occlusion of the natural ostium. Pain and tenderness are present over the affected maxilla; sometimes the pain is referred along one of the divisions of the trigeminal nerve. Breathing through the nostril on the side of the lesion is impaired and often obstructed. If the ostium is patent, which is unusual in acute cases, a flow of pus can be obtained when the head is held downwards and forwards with the affected side uppermost.

Transillumination of the antrum and a radiograph are likely to reveal a relative opacity of the affected antrum.

**Treatment.-** This includes bed rest, analgesics, ephedrine 1% in saline by a nebulizer, menthol inhalations, and systemic chemotherapy if the temperature is over 38°C. Puncturing the antrum is for diagnostic and therapeutic purposes. An antral trocar and cannula punctures the antrum beneath the inferior turbinate bone after application of surface anaesthesia. Pus is aspirated and washouts are performed with isotonic saline.

**Chronic Sinusitis.-** Pain and swelling are often absent, and frequently the only symptom is an intermittent discharge and facial discomfort or headache. Diagnosis is established by x-ray or by fiberoptic examination through a small trocar punch opening either under the inferior turbinate or sublabially in the anterior wall using local anaesthesia.

**Treatment.-** Chronic infection does not respond to antibiotic therapy and needs surgical drainage by an intranasal antrostomy or a Caldwell-Luc operation.

**Intranasal antrostomy.-** Under general anaesthesia the inferior turbinate is slightly elevated and a window created in the medial wall of the antrum in the inferior meatus of the nose.

**Caldwell-Luc Operation.-** This is a good standard procedure, though not advocated in children as dentition may be affected. (George Walter Caldwell, 1866-1946. Otolaryngologist who practised successively in New York, San Francisco and Los Angeles. Described his operation for treating suppuration of the maxillary antrum in 1893. Henri Luc, 1855-1925. Otolaryngologist, Paris. Described his operation in 1889.) Under general anaesthesia, with oral intubation and packing, an intra-oral incision 2.5 cm long is made in the buccolabial sulcus, centred over the canine fossa. The muco-periosteum is elevated and a gouge is used to make an opening about 12 mm in diameter through the anterior wall of the antrum. All the mucous membrane is removed with Luc's forceps and any diseased ethmoid cells can be exenterated. An intra-nasal antrostomy completes the procedure, allowing post-operative washouts.

**Nasal Polypi.-** Nasal polypi (oedematous ethmoid sinus mucosa) are usually multiple and are seen to prolapse from the ethmoid cells in the region of the middle turbinate. They are recognised by their glistening gelatinous appearance when light is focused upon them.

They are practically confined to adults, and patients complain of nasal obstruction, nasal discharge, and some loss of smell. Many of the sufferers are allergic to dusts and pollens which may initiate attacks of sneezing and rhinorrhoea.

**Treatment.-** Single polypi are removed under local surface anaesthesia with a snare and appropriate forceps. Multiple polypi usually require a general anaesthetic for more complete removal. Associated ethmoidal or antral procedures can be then undertaken at the same time. Antihistamines or topical steroids should be used after removal, since recurrence is common.

## **Epistaxis**

**Source and Cause of the Bleeding.-** The bleeding may be arterial or venous. In 90% of cases it comes from a plexus of veins (sometimes varicose) situated in Little's area (James

Lawrence Little, 1836-1885. Professor of Surgery, University of Vermont, Burlington, Vermont, USA.) on the antero-inferior portion of the septum. The most frequent cause is nose-picking - epistaxis digitorum. Other causes include trauma, nasal infection, neoplasms, hypertension, blood dyscrasias, acute specific fevers, nephritis and uraemia.

**Treatment.-** Sit the patient up so that blood does not run down the throat. Blow nose to remove clots. Pinch the nose for ten minutes. An ice-pack or cold sponge applied to the nose is often helpful.

**Cauterising the bleeding-point** with trichloroacetic acid, 50%, or electric cautery, is undertaken if the bleeding-point can be seen.

**Sedation** with diazepam or Omnopon and admission to hospital are necessary if bleeding is uncontrolled. Blood transfusion may be required.

**Packing: Anterior Packing.-** After anaesthetising the mucous membrane with 4% xylocaine, ribbon gauze coated with BIPP (Bismuth Iodoform Paraffin Paste) is inserted so as to fill the nasal cavity, and if the bleeding is from the septum the nasal cavity of the opposite side is packed also. Antibiotics are given to combat infection.

*Posterior Packing.-* The insertion of a cone-shaped gauze tampon moistened with BIPP is the most satisfactory, although a quick method is to insert a Foley-type catheter as far as possible to the back of the nasal cavity and inflate the balloon. The pack can be left in for five days if an antibiotic is administered.

**Arterial Ligation.-** When the above methods fail, or profuse haemorrhage recurs, it is likely that the source of the bleeding is more posterior. Ligation of the anterior ethmoidal artery in the orbit is indicated, particularly in traumatic cases. Transantral ligation of the internal maxillary artery and ligation of the external carotid artery are other methods which are sometimes called upon.

### **Deflected Nasal Septum**

Although the majority of adults do not have a completely straight septum, it is only the grosser deflections causing symptoms which require correction. Septal deformities are caused either by trauma or by disproportionate growth during development. They may produce nasal obstruction or headaches in the region of the nasion.

The submucous resection (SMR) operation consists of the removal of cartilaginous and bony spurs from between the two coverings of mucoperichondrium and mucoperiosteum. This allows the septum to take a more midline position.

**Nasal Bone Fractures** (Chapter 30).

### **The Ear**

Now follows a brief presentation of well-known afflictions of the ear. The reader is referred to the appropriate text-books for fuller accounts and descriptions of operative surgery.

**Surgical Anatomy.-** The ear consists of four parts: (1) Pinna, (2) External auditory canal, (3) Middle ear, (4) Inner ear and Eighth nerve track.

The pinna is cartilaginous, covered by stratified squamous epithelium. The external auditory canal is in two parts: the outer cartilaginous part, which is lined by similar epithelium, tapers to its narrowest part where it joins the bony external auditory canal. This is lined by a thin stratified epithelium containing no glands or hair follicles, and widens medially until closed off by the tympanic membrane at an angle of 60° to the vertical.

The middle ear is part of the mastoid bone. Above, is the attic connected by the aditus to the mastoid antrum and variable air cells. Below, it communicates with the nasopharynx via the Eustachian tube. It contains (a) three ossicles: malleus, incus, and stapes; (b) two muscles - tensor tympani and stapedius; (c) two windows - oval and round; (d) one nerve - chorda tympani. The facial nerve traverses the superior and posterior wall in its bony canal. The horizontal semi-circular canal lies in the attic.

**Congenital Abnormalities.-** These are: partial or complete absence of pinna; partial or complete occlusion of external auditory canal; partial or complete absence of middle ear - the stapes is nearly always present. Treatment is surgical reconstruction when the child is old enough for the precise hearing level of each ear to be established.

### **External Ear**

**Wax.-** This is best removed by syringing with warm water and bicarbonate of soda solution (1%) after appropriate oily drops have been introduced for the previous two nights. An aural syringe with a small nozzle must be used, and directed upwards and backwards. An intact drum is unlikely to rupture if these rules are followed.

**Foreign bodies** easily become wedged in the narrow part of the meatus, whence they can be removed using a hook, sucker or fine forceps under indirect lighting or under the surgical microscope. This should be done by someone familiar with such procedures, especially in children. In difficult cases, a general anaesthetic may be required.

**Inflammation.-** Inflammation of the external canal may be localised (furuncle) or widespread (external otitis).

**Furuncles** are staphylococcal in origin, and produce pain, pyrexia, periauricular swelling and, characteristically, a painful pinna on movement.

*Treatment.-* Decongestive drops or wicks (eg, glycerine and ichthyol or antibiotic hydrocortisone cream). If symptoms are severe or generalised (pyrexia), broad spectrum antibiotics are required.

**External otitis** (syn telephonist's ear, Singapore ear) is often fungal in origin and may start as a dry scaly eczema. Irritation, discharge, and minimal deafness are the symptoms - secondary bacterial infection introduced by scratching the ear leads to further discharge and pain. Antifungal preparations combined with a local antibiotic either as drops or cream are

indicated. The primary cause, scalp or toes, must be treated at the same time and the ears kept dry. Mild sedation, eg, Phenergan 25 mg may be advisable.

**Carcinoma of the Pinna** is of the squamous-celled type, and the condition is often comparatively advanced before the patient seeks relief. However, if treated early, favourable results accrue from excision of a part, or the whole of the pinna. Acrylic and rubber prostheses effectively replace the lost part.

**Bony Exostoses.**- Supposed to afflict those who indulge in under-water swimming.

### **Middle Ear**

*Note: glue ear and acute otitis media in children are conditions which are encountered very commonly.*

'**Glue ear**', more correctly called seromucinous otitis media, has become an increasingly common disorder in children. It usually affects those with recurrent otitis media and often enlarged infected adenoids, presumably because of poor Eustachian tube function. It is especially common in children with cleft palate (even after repair) as the muscles which open the tube have lost their insertion into the middle of the palate and atrophied. Inadequate antibiotics for acute infections is another possible cause. 'Glue ear' is painless, and simply causes conduction deafness, of which it is the commonest cause in childhood. The appearances of the eardrum may be striking (gross retraction, yellow or grey/blue discoloration, increased vascularity), but more often are not. The condition must therefore be suspected from the history.

**Treatment.**- An antihistamine/decongestant mixture is usually tried first, but if this fails to clear the condition, adenoidectomy, myringotomy and grommet insertion are advised. In cleft palate cases the adenoids are never removed, however, lest nasal air escape during speech be worsened.

**Acute otitis media** is secondary to an upper respiratory infection, and presents as severe prolonged earache with pyrexia and systemic illness. In some very acute cases, pus may discharge through a perforation before treatment can be started. More usually, the eardrum is reddened in appearance. Vomiting may be a presenting symptom.

**Treatment.**- Antibiotics are given in full dosage for five days; nasal decongestants may be added. Early antibiotic treatment has meant that myringotomy (incision of the eardrum) is rarely required nowadays.

*Complications.*- Acute mastoiditis and intracranial complications have become rare since the widespread use of antibiotics.

*Recurrent otitis media.*- Frequent attacks of otitis media in childhood are an indication for adenoidectomy.

**Acute Mastoiditis** (now rare).- The symptoms are pain deep in the ear, periauricular swelling, pyrexia. The pinna is not painful on movement (as distinct from a furuncle). Conduction deafness is marked.

**Treatment.**- Pus must be let out. Therefore a simple mastoidectomy is performed, with an adequate course of antibiotics in support.

The operation entails the removal of the infected air-cells through a posterior auricular incision. The middle ear is not disturbed. The wound is drained for twenty-four hours.

*Complications* are lateral sinus thrombosis, extradural abscess, meningitis, cerebral or cerebellar abscess, facial palsy, and, rarely, labyrinthitis.

**Chronic Otitis Media.**- This term simply means that there is a perforation of the eardrum. If the ear is constantly discharging scanty offensive pus through a posterior or superior perforation ('unsafe type'), modified radical mastoidectomy may be required, especially if cholesteatoma is present (see below).

A dry central perforation ('safe type') which discharges intermittently with colds or swimming, is treated by antibiotic/steroid drops when wet, and may be repaired when dry by grafting of the eardrum (myringoplasty) if necessary with reconstruction of the ossicular chain (tympanoplasty).

**Tympanoplasty.**- Operations and procedures under this group are designed to repair or reconstruct the tympanic membrane and ossicular chain of the middle ear. Various tissues have been used including homografts from cadavers and animals but the most widely accepted are autogenous fascia with repositioned ossicles or shaped bone chips.

*Complications.*- Conduction deafness with ossicular chain destruction, labyrinthitis, facial palsy, cerebral meningitis, cerebral and cerebellar abscess. A *cholesteatoma* (skinball), whose origin is debatable, produces otorrhoea and the above complications by pressure necrosis.

*Treatment* is surgical by (modified) radical mastoidectomy.

**Ruptured eardrum** is due to trauma, eg, hard slap on ear or syringing. There is sudden pain and deafness, with tinnitus and bloodstained discharge.

*Treatment.*- Do not touch. No eardrops. No antibiotics required.

**Otosclerosis** produces bilateral conduction deafness. Common in females, it is often hereditary, and may be exacerbated by pregnancy.

*Treatment* by hearing aid or operation - stapedectomy with Teflon (or wire) strut and vein (or fat) graft. This produces improvement permanently in 90% and it has replaced the fenestration operation.

**Serous Otitis Media** is usually found in adults and, in contrast with 'glue ear', the middle ear effusion is thin and watery; like 'glue ear', however, it causes conduction deafness. It may result from viral upper respiratory infection or from nasopharyngeal carcinoma (causes of Eustachian tube obstruction), or from barotrauma (sudden change in atmospheric pressure produced by flying or diving). Treatment is by decongestants and, if persistent, by myringotomy and grommet insertion.

**Malignant Tumours.-** Excluding the pinna, **squamous-celled carcinoma**, though uncommon, can arise in the middle ear or mastoid cells. There is pain and otorrhoea (which may be long-standing), becoming bloodstained. Deafness is variable. Biopsy will confirm. Treatment is by combined surgery and radiotherapy, but the prognosis is poor.

**Glomus jugulare tumours** are rare but of special interest as they resemble carotid body tumours (Chapter 36). Slow growing and locally invasive, they are highly vascular, with nests of epithelial cells and fibrous tissue. Early signs are seventh nerve palsy, pulsatile tinnitus, and free bleeding. Radiotherapy diminishes both size and vascularity. Cryosurgery is effective for small tumours.

### The Inner Ear

**Presbycusis.-** High-tone bilateral perception deafness gradually affects lower tones with advancing years. It is often associated with atherosclerosis. Treatment is by a hearing aid.

**Cochlear Concussion.-** Hearing at 4000 Hz is commonly affected, but if concussion is repeated daily (eg, gunfire, road-driller, or aircraft pilot not using ear defender), the loss spreads up and down. Failure to hear the telephone bell is often the first symptom.

**Ménière's syndrome** (Prosper Ménière, 1799-1862. Physician, Institute for the Deaf and Dumb, Paris. Described this condition in 1861.) is unilateral perception deafness, intermittent true rotational vertigo associated with nausea and tinnitus. It may be associated with hyper- or hypo-tension, intracranial lesions, syphilis, or disseminated sclerosis. But if all these are excluded, and labyrinthine function tests show loss of function on one side, it is true Ménière's disease.

*Treatment* may be (a) medical - Tab nicotinic acid 100 mg tds (vasodilator), Avomine, Stemetil, or (b) surgical - 1. Operations to reduce endolymphatic pressure by a surgical approach to the endolymphatic sac for drainage can prove satisfactory. Also relatively minor procedures such as ultra-sound or cryosurgery applied to the labyrinth may indirectly produce a similar result.

2. Labyrinthine destruction. A labyrinthectomy may be indicated in cases where the hearing is severely impaired and the patient is getting vertigo symptoms not controlled by other means.

**Congenital Deafness.-** This is due to intrauterine viral toxins in the first three months of pregnancy (eg, rubella, influenza, etc).

## Recent Advances

1. *Surgery for acoustic neuroma*, formerly the province of the neurosurgeon, is now often a team operation by otologist and neurosurgeon, using a translabyrinthine or posterior cranial fossa approach.

2. *Middle cranial fossa approaches* to the temporal bone, mainly to decompress or to repair the facial nerve, or to section the vestibular nerve are practised in a few centres.

3. *Cochlear implantation*, for electrical stimulation of the inner ear in total deafness is still at an experimental stage.