Chapter 32

The Mouth. The Cheek. The Tongue.

Stomatitis

Stomatitis is a general term applied to inflammatory, erosive and ulcerative conditions widely affecting the mucous membranes which line the oral cavity. Gingivitis (Chapter 33) refers to inflammatory, erosive and ulcerative conditions which are confined to the mucoperiosteum (gums) covering the alveolar processes. Some inflammatory conditions of the gums may spread to involve other parts of the oral mucous membrane, in which case the term gingivostomatitis is used.

The mucous membrane which covers the alveolar process, the retromolar region and the hard palate is keratinised, firmly attached to the underlying periosteum and bone and is relatively resistant to injury. It is described as the masticatory mucosa. The papillated mucosa of the dorsum of the tongue is also keratinized and tough and firmly attached to the underlying muscle. Certain diseases have a characteristic distribution in relation to the different types of mucosa lining the oral cavity.

Infecting Organisms.- Many organisms are to be found in the oral cavity where, under normal circumstances, the majority are harmless to the individual. The number of such organisms is controlled, and their ability to cause harm is reduced, by a variety of factors such as:

1. The regular desquamation and replacement of surface cells. By this means microscopic damage to the surface is repaired and many organisms are carried away on the shed cells.

2. The constant washing of the oral cavity by saliva so that organisms are swallowed and destroyed in the stomach.

3. The mild antibacterial activity of the saliva.

4. The health and integrity of the lining epithelium. Certain oral commensals are *facultative pathogens* and will take advantage of any weakness in the defences of the oral mucosa to produce a localised or generalised infection of the mouth. Certain other organisms produce specific infections are *true pathogens.*

The surface of an ulcer in the oral cavity, irrespective of its cause, is soon colonised by facultative pathogens such as oral streptococci, staphylococci and occasionally Vincent's organisms (Jean Hyacinthe Vincent, 1862-1950. Professor of Epidemiology, Val-de-Grace Military Hospital, Paris.). There is a resultant non-specific, acute, inflammatory reaction in the tissues forming the floor of the ulcer. For this reason a microbiological examination of swabs from oral ulcers and a biopsy is not often helpful as a means of establishing the diagnosis. Where necessary, secondary infection of oral ulcers may be treated by an 0.2% aqueous solution of chlorhexidine gluconate which is used as a mouthwash four times a day.

Predisposing Factors.- 1. *The patient's general health and nutrition* are reflected in the state of the oral mucosa. Anaemia associated with vitamin B_{12}, folic acid and iron deficiency all lead to thin, atrophic, epithelia and loss of the papillae from the dorsum of the tongue. The mucosae are easily damaged during mastication so that the patient suffers from recurrent ulceration of the insides of the lips and cheek. Lack of several other B vitamins can lead to loss of the papillae on the dorsum of the tongue. All such patients may present with a complaint of a red, burning tongue and angular cheilitis. Both the sore tongue and the angular cheilitis are due to secondary infection by candida albicans. Severe vitamin C deficiency ('scurvy') also causes ulceration of gums and buccal mucosae by interfering with collagen synthesis. A similar condition of the mouth is seen in severe protein deficiency (kwashiorkor = The name means the 'red boy' or 'the disease the child gets when the next baby is born' in the language of Ghana.) and in sprue. Cancrum oris is another serious condition associated with malnutrition.

2. *Other factors affecting the health and efficiency of epithelium* may lead to recurrent ulceration, or chronic candida infection. Several inherited conditions, such as epidermolysis bullosa result in abnormalities of both skin and oral mucous membrane. Epidermolysis bullosa leads to bullae which, in the moist environment of the mouth, soon rupture to form ulcers. In severe forms, gross scarring occurs with adhesions which obliterate the sulci. Hyperkeratotic patches, due to any cause, may be invaded by candida albicans. Cytotoxic drugs affect the ability of the epithelial cells to reproduce so that the epithelium is abnormally thin and easily damaged. Repair is also slow because epithelialisation of the resultant ulcers is delayed.

3. *A reduced ability to deal with secondary infection* may lead to recurrent ulceration or the persistence of ulceration of the oral mucosa which is the result of minor trauma. Cyclical leucopaenia, agranulocytosis, aplastic anaemia and hypogamma-globulinaemia are conditions which predispose to recurrent oral ulceration in this way. Patients who are taking adrenal cortical steroid drugs tend to develop thrush.

4. *An immune or auto-immune mechanism* is believed to underlie a variety of conditions affecting the oral mucous membranes. Damage to epithelial cells can result in a slough which separates to form an ulcer. Such a mechanism has been involved to explain recurrent aphthous ulcers. Damage to cell membranes and intercellular cement substance can lead to intra-epithelial bullae as in pemphigus. If the basement membrane is damaged subepithelial bullae or blood blisters may result as in benign mucous membrane pemphigoid. Damage to submucosal vessels leads to thrombosis and a deep-penetrating ulcer as in *Behçet's disease (Hulusi Behçet, 1889-1948. Dermatologist, Istanbul, Turkey.)* Indirect immunofluorescence studies with serum from patients with pemphigus may demonstrate antibodies in the intercellular region in the epithelium of tagged monkey oesophagus. Direct immunofluorescence studies on biopsy material will demonstrate antibody on the basement membrane in benign mucous membrane pemphigoid.

If a biopsy is used in the diagnosis of these conditions a specimen should be taken which includes the adjacent mucous membrane. The fragment of tissue should be handled with particular care as the epithelium will separate readily from the underlying tissues, rendering it useless to the histopathologist.

Lichen planus, for which an auto-immune mechanism forms a less certain explanation, may also produce widespread oral erosions, submucosal bullae and frank ulceration in addition to the more familiar lace-like hyperkeratotic lesions.

An immune reaction often in response to a drug such as sulphonamide or phenobarbitone underlies the severe bullous and ulcerative stomatitis which is seen in erythema multiforme and usually occurs in association with the characteristic target-like skin lesions.

5. *Certain chemicals* predispose to, or give rise to, a stomatitis. Inhalation of mercury vapour results in mercury poisoning, the ill effects of which include redness and swelling of the gums, and loosening of the teeth. Necrosis of the bone may follow. Chronic lead poisoning results in the appearance of a blue-black line at the gingival margin due to the deposition of lead sulphide in the tissues. The sulphur which reacts with the circulating lead to form the insoluble sulphide comes from bacterial activity in food debris at the gingival margin. Now bismuth is not used to treat syphilis, the similar black or purple bismuth line is no longer seen. In each of these conditions a secondary Vincent's gingivitis is prone to arise. The excessive ingestion of iodides leads to a sore mouth and excessive salivation.

Particular Types of Stomatitis

Aphthous Stomatitis.- This term is now applied to three specific entities: recurrent minor aphthous ulceration, recurrent major aphthous ulceration and herpetiform aphthous ulceration.

Minor aphthae appear as crops of between one and many ulcers on a cyclical basis. The ulcers are up to 0.5 cm across, round or oval in shape, with a yellow base and a red erythematous margin. They are distinctly painful, occur in the unkeratinised mucosa of the cheek, lips, soft palate and floor of the mouth and normally heal within 10-14 days. The frequency of the ulcerative episodes varies considerably. They occur more frequently in women than men and may then coincide with the second half of the menstrual cycle.

Major aphthae are in many ways similar, but are usually larger and deeper. As a consequence they may enlarge to involve the keratinised mucosae, take longer to heal and do so with scarring.

A chlorhexidine gluconate mouthwash will help many sufferers. Major aphthae can be coated, after the use of the mouthwash, with triamcinolone acetonide (Adcortyl in Orabase); a pectin material which adheres to the moist surface of the ulcer and protects it. Choline salicylate gel can be applied to minor ulcers to relieve pain. The disappearance of pain from the ulcers heralds rapid healing.

If the patient has genital and conjunctival lesions as well as severe oral ulcers, the possibility of *Behçet's syndrome* should be considered. In *Reiter's syndrome (Hans Reiter, 1861-1969. President of the Health Services and Honorary Professor of Hygiene, Berlin.)* there is urethritis, arthritis, periarteritis nodosa, conjunctivitis and oral ulcers. The latter may either resemble minor aphthae or the erosions seen in bullous lichen planus. They are noticeably less painful than aphthous ulcers and respond to tetracycline.

Herpetiform aphthous ulcers are quite small; only 1-2 mm in diameter. They occur in crops of many ulcers. They are probably of a different aetiology to minor and major aphthous ulcers. Despite their name they are *not* caused by the herpes simplex virus. 0.2% chlorhexidine or a tetracycline mouthwash will lead to rapid healing.

Herpes Simplex Infections

For most individuals the primary infection with the herpes simplex virus is subclinical. In others it results in a marked gingivostomatitis which usually occurs in infancy or childhood, but may also occur during adolescence or early adult life. Many small vesicles appear and rapidly break down to form small, yellow ulcers with bright red margins. They occur on the gingivae, cheeks, lips and tongue. Lesions may also occur around the nostrils and even on the skin of the cheek. The patient is unwell, febrile and has markedly swollen submandibular lymph nodes. A soft diet, plenty to drink, an analgesic elixir and the gentle swabbing of the ulcers with 0.2% aqueous chlorhexidine is all that usefully can be done until the child recovers naturally in about 10-14 days.

Herpes labialis, which appears in response to cold winds, bright sunlight, or febrile illnesses is due to periodic reactivation of the virus. Parents who suffer from herpes labialis should avoid kissing their children when they have active lesions for obvious reasons. Doctors, dentists and nurses should be careful how they handle the lips and mouths of affected patients, lest they develop a herpetic whitlow or transfer the virus to their eyes to produce a herpetic keratitis.

Other Virus Infections.- Intraoral bullae and painful ulceration can occur in herpes zoster. Small bullae and ulcers also appear in the mouth, though mainly on the soft palate, in chicken pox. Ulceration of the soft palate and fauces occurs in both herpangina (due to coxsackie group A3 virus) and hand, foot and mouth disease (coxsackie A16 virus) but the latter is not to be confused with the foot and mouth disease of cattle which only rarely affects humans. Small bullae are also found on the palms of hands, soles of the feet and gingivae in hand, foot and mouth disease which tends to occur in localised outbreaks affecting several individuals. A large sloughing ulcer on the faucial tonsil or posterior third of the tongue may be an early cause of complaint in glandular fever.

Monilial Stomatitis.- Several clinical forms are seen of oral infection with candida albicans.

Acute pseudomembranous candidiasis or thrush occurs classically in debilitated infants. Small, thin, soft, moist, creamy-white plaques form on the mucous membrane, looking like adherent curds of milk. If the white patch is wiped off a bleeding erosion is revealed. Thrush may be seen also in the chronically ill, the elderly, or at any age during treatment with adrenocortical steroids or cytotoxic agents. 1% aqueous gentian violet applied direct to the lesions is the best treatment for the very young and amphotericin B lozenges to dissolve in the mouth four times a day for older patients.

Acute hypertrophic or hyperplastic candidiasis resembles the acute pseudomembranous form except that the tongue is usually involved and a thick, confluent, white mass of mycelia and keratin coats the dorsum.

Acute atrophic candidiasis is most often seen as a complication of treatment with broad spectrum antibiotics. Again it is the tongue which is most affected. The lateral part of the dorsum is smooth and red and the patient complains of a burning sensation.

Chronic atrophic candidiasis or denture sore mouth, produce a red oedematous mucosa over the area covered by an upper denture. Sometimes there is a papillary hyperplasia of the mucosa of the hard palate. Despite the name 'denture sore mouth', it is usually painless. The patient's denture hygiene is usually poor and needs to be improved. Further, the patients are often wearing the dentures at night, and this they should not do. Amphotericin B lozenges are sucked to get rid of the candida in the palatal epithelium, or Nystatin cream may be applied to the inside of the dentures before they are worn.

Chronic Hypertrophic or Hyperplastic Candidiasis and Speckled Leucoplacia (see candida leucoplacia below).

Angular Cheilosis (Syn: Angular Stomatitis).- There are moist, infected and crusting cracks at the angles of the mouth. The cause is a leak of saliva at the corners of the mouth and the moist skin becomes infected by candida and staphylococci. It may be seen in children who suck a finger, when it is called *perleche,* or in the middle-aged and elderly. In the older patient the face sags and wrinkles to produce a moist fold. A similar deepening of the crease at the angle of the mouth tends to occur in the edentulous person, particularly where atrophy of the ridges under the dentures permits overclosure. Loss of the canine eminence also permits the angles of the mouth to sag. The local infection can be treated with nystatin cream and fusidate ointment, or alternatively miconazole cream, which is effective against both candida and staphylococci. The denture wearer should go to the dentist for new dentures to correct the overclosure of the jaws. Any atrophic candidiasis under the upper denture should be treated at the same time.

Vincent's Acute Ulcerative Gingivitis and Stomatitis (Syn: acute ulceromembranous stomatitis).- The *Borrelia vincentii* and the *Fusiformis fusiformis* are always to be found in large numbers in smears from slough over the lesions in these conditions. On this evidence they are likely to be the aetiological agents.

However, experiments in which material from the surface of active lesions has been packed into the interdental spaces of volunteers have failed to produce the disease. The *Borrelia vincentii* is a mobile spirochaete with three to four loose spirals while *Fusiformis fusiformis* is a large rod-shaped organism with pointed ends. They are both anaerobic and Gram negative. They may be demonstrated in smears of slough from the lesions stained with carbol fuchsin, but are very difficult to culture. Acute ulcerative gingivitis starts on the crests of the interdental papillae and progresses to form a deep crater covered with a greenish grey slough which is composed largely of necrotic tissue. The organisms are found only in the slough and do not penetrate into the underlying tissue.

In some patients the infection starts around a partly erupted wisdom tooth and in others in the crypts of the tonsils. Tonsillar infection is called Vincent's angina. In severe cases it may spread over the adjacent tissues. The ulcers bleed readily and the patient may complain of spontaneous gingival haemorrhage. In develop countries the disease is not seen

in young children, but is not uncommon in adolescents and young adults. It does not affect the edentulous mouth. There is a characteristic musty foetor oris.

In acute cases the patient is unwell, has a pyrexia, complains of a persistent, severe ache in the affected part and salivation. However, subacute infections may also occur with little constitutional disturbance. The incidence increases during the winter months and during war time, so that other intercurrent illnesses, or a mild degree of malnutrition may be important factors in the occurrence of the disease. The Vincent's organisms are sensitive to both penicillin and metronidazole, but treatment with antibiotics alone is insufficient to eradicate the gingival or pericoronal infection. Various local factors also need to be treated by the patient's dental surgeon. Unless the disease is controlled, severe damage to the attachment of the teeth can result.

The organisms may also infect deep human bites where anaerobic conditions exist in the depths of the wound.

Cancrum oris is a severe form of the disease affecting young, poorly nourished children and occurs as a complication of measles and other childhood illnesses. Malnutrition is a predisposing cause and the presence of erupted teeth appears necessary for the onset. Vincent's ulceration start on the gingivae and spreads over and into the bone of the jaw. From the gum it spreads on to the inside of the cheek and then through to the skin surface, producing a large area of full thickness tissue loss.

Unless treatment is prompt the child is likely to die. The treatment is systemic penicillin and metronidazole, local wound irrigation and the introduction of a high-protein, vitamin-rick diet with a nasogastric tube. Healing results but with gross scarring which prevents movement of the mandible and, of course, there is often a large hole through the cheek. All scarred tissue should be excised and the defect repaired with a lined, tubed pedicle flap which is inset with the mouth wide open.

Rhagades occur at the corners of the mouth in congenital syphilis which, when they heal, leave radiating scars and furrows. These usually extend further round the lips than the lesions of perleche for the latter condition rarely causes scarring.

AIDS (Acquired Immune Deficiency Syndrome) may be the cause of oral lesions and a patient with AIDS may first present with these. Kaposi sarcoma is rare outside Africa but may occur in AIDS; when these lesions appear in the mouth they present as reddish or purple pedunculated lumps, sessile or only slightly raised swellings. The palate is a not infrequent site. Leucoplacia or chronic hypertrophic candidiasis should also raise suspicion if they occur in a young adult.

Burns

Burns and scalds from hot food and drink may cause blistering, but rarely severe injury. Children occasionally put the spout of a kettle in the mouth and this causes more severe scalding. Hydrocortisone cream and chlorhexidine mouth washes may help but little else is possible for the acute injury. Small electrical connections from a mains electric plug to portable radios and razors etc may be placed in the mouth by a small child to produce

severe electrical burns. The lips particularly are damaged, resulting in complete loss of skin and mucous membrane. Subsequent surgical correction of microstomia may be required; however a dentist is sometimes able to construct an appliance to keep the raw surface stretched until secondary epithelialisation occurs. Chemical burns are also seen occasionally. The commonest type is the aspirin burn. The patient places a tablet of aspirin in the sulcus against a painful tooth, where it produces a white, soggy, sloughing lesion.

The Single Oral Ulcer

The most frequent cause of a single ulcer in the oral cavity is acute or chronic trauma. Toothbrush abrasions produce linear ulcers in the sulcus. The flange of a denture which has been over extended, or which is too deep because the ridge has atrophied, will produce recurrent acute or chronic linear ulcerations also in the sulcus. Thinly epithelialised scars of healed ulcers may be seen. The tongue is an inquisitive organ and once it has discovered a sharp point or edge on a tooth, a cavity in a tooth, or a projection on a dental appliance it will constantly seek it out and explore it until an ulcer results. Such ulcers may persist for weeks or months, in which case the possibility that a malignant neoplasm is the cause must not be overlooked.

Any obvious source of irritation should be dealt with, but a biopsy is necessary if rapid healing does not follow. In some benign ulcers biopsy provokes healing, perhaps because the surgical lesion is painful and deters the patient from rubbing his tongue over it.

Tuberculosis, syphilis and various fungi can produce chronic ulcers which require biopsy, or special microbiological examinations to find the cause. Wegner's granuloma (Friedrich Rudolf Georg Wegner, 1843- . German Pathologist.), eosinophil granuloma and reticulum cell sarcoma may all produce chronic, slowly progressive ulcers. Because of the effects of secondary infection the diagnosis may not be readily apparent, even in a biopsy.

Salivary Mucus Extravasation and Retention Cysts

Mucus cysts of minor salivary glands produce pinkish, bluish, or yellowish, globular, soft swellings up to 1.5 cm in diameter on the inner aspect of lips or cheeks. A few are retention cysts behind minute calculi. The majority are extravasation cysts. Mucus escapes into the tissues following rupture of the duct. The cyst and the associated minor gland from which the saliva is leaking should be excised together.

Ranula.- Ranulas (*ranula*, lat a diminutive of *rana* = a frog) are extravasation cysts arising from a damaged sublingual gland. If the saliva distends the floor of the mouth a translucent bluish swelling is formed with prominent blood vessels running over its surface. If mucus is escaping from a posterior sublingual gland it may flow over the posterior margin of the mylohyoid and down into the neck to form a *plunging ranula.*

The wall of a ranula is composed of a delicate capsule of fibrous tissue and is lined by a layer of macrophages. Many ranulas rupture and discharge. Some fail to refill, but many will recur. Gentle dissection to identify the sublingual gland from which the swelling is arising permits the offending gland mass to be removed. It is unnecessary to explore the neck

for a plunging ranula. All that is required is to remove the entire mass of sublingual glands on the same side and to pass a drain from the floor of the mouth down into the neck cavity.

Lingual and Sublingual Dermoids

Dermoid cysts produce an opaque swelling. They may be lined by stratified squamous epithelium, with, or without, dermal appendages. Such cysts are filled with a doughy mass of keratin. Others are lined by ciliated, mucus secreting epithelium and filled with mucus. These are fluctuant. Dermoid cysts may be found in the midline of the tongue, or in the floor of the mouth, either in the midline or laterally in the sublingual region.

Median sublingual dermoids are seen more often than the other types. They are probably derived from epithelial rests left as the two contributions to the tongue from the back of the mandibular arch merge together, they enlarge backwards between the genial muscles, into the tongue and down towards the hyoid. They lie above the mylohyoid muscle, but bulge downwards towards the submental region as the mouth is closed and the oral swelling is compressed by the tongue. Their removal is best effected via an incision which extends vertically in the midline from the tip of the tongue to the attachment of the lingual frenum to the mandible.

Lateral sublingual dermoid cysts develop below the submandibular duct and lingual nerve, and anterior to the stylohyoid ligament; that is, from the region of the first branchial pouch. They displace the submandibular salivary gland backwards as they enlarge. Small ones are removed through the floor of the mouth and large ones through a submandibular incision.

Tumours of the Cheek and the Floor of the Mouth

A miscellaneous variety of abnormal lumps may be found within the substance of the cheek or floor of mouth or arising from their surface. Some are benign neoplasms, some are hamartomas and others are inflammatory hyperplasia. A few of the more common examples are:

Fibro-epithelial Polyp.- If the cheek is repeatedly traumatised at one place a thickened, submucous scar is formed which may be pulled out on to a stalk by the suction of deglutition. A lump of cheek may be sucked by a similar mechanism into a gap where a tooth is missing. The result is a soft, rounded, pedunculated, fibrous swelling.

Fibro-epithelial polyps are excised through the base of the pedicle.

Denture-induced Granuloma.- Fusiform, fibrous masses arising from a linear pedicle are formed in edentulous individuals in the buccal or lingual sulci; sometimes singly and sometimes several together parallel to one another. These granulomatous masses arise as a result of chronic irritation and ulceration from the flange of an ill-fitting denture.

Papilloma.- Papillomas may occur on the cheek, alveolar mucosa, palate or floor of the mouth. They may be solitary or multiple. Some are a mass of tiny, close-set, finger-like processes, others have a knobbly surface like a cauliflower. They are covered with keratinised epithelium which is white in the moist environment of the mouth. Some patients have

multiple papillomas in their mouth and may have viral warts elsewhere. In children, such other warts may be on the knuckles. In adults the lesions may be genital warts.

Haemangiomas.- May occur in the cheek and floor of the mouth. Small cavernous haemangiomas up to 0.5 cm in diameter may be found on the inside of the cheek opposite the occlusal plane and arise as a result of a bite which damages a submucosal vessel. More massive haemangiomas surround the buccal pad of fat in the buccal space.

Lipomas.- Produce yellowish submucosal swellings in the cheek and floor of mouth.

Neurofibromas.- Most often produce a diffuse thickening of the gums spreading out into cheek or floor of mouth. Occasionally a single, fusiform, soft, fibrous mass is found along the line of a nerve, such as the lingual nerve. Others may produce pedunculated swellings.

Lymphangiomas.- Tend to occur posteriorly in the cheek over the coronoid process and look like brownish frog spawn.

Salivary Tumours.- Pleomorphic adenomas, muco-epidermoid carcinomas, adenoid-cystic carcinomas, and more rarely other salivary neoplasms, may arise in buccal, retromolar, labial and sublingual salivary glands (see Chapter 31).

Facial Lymph Node.- Some individuals have a lymph node which is an outlying member of the submandibular nodes; it is found lying over the body of the mandible along the path of the facial artery as it goes towards the angle of the mouth. When enlarged it may produce a palpable lump in the cheek or lower buccal sulcus (Chapter 33).

Carcinoma of Cheek

While adenocarcinomas may arise in a minor salivary gland, these are uncommon. The majority of carcinomas of the mucosal aspect of the cheek are squamous cell in nature. In members of Western races they are more common among those who smoke heavily and drink spirits. Some arise in candida-infected speckled leucoplacia. Carcinoma of the cheek is specially common where betel nut is chewed and the 'pan' or plug stored in the cheek pouch. It is also a complication of submucous fibrosis. An exuberant papilliferous tumour may be produced by a verrucous carcinoma.

Treatment of Carcinoma of Cheek.- The best results from the point of view of function and appearance are achieved by radiotherapy. Interstitial radiation may be delivered by ^{137}Caesium needles or by ^{192}Iridium wire. The latter can be afterloaded into nylon tubes which may be placed accurately without risk to the operator and the position checked by radiography with non-active wires in position. Alternatively external beam treatment with megavoltage machines may be used.

Surgery is required for residual or recurrent tumour or where radiotherapy is not available. Post-irradiation surgery may also be advisable for large tumours involving the adjacent jaw. A small or superficial tumour can be excised and the resultant wound grafted with split skin applied on a polyurethane foam pad which is sutured in place for 10 days.

Larger tumours require full thickness resection of the cheek and repair. A deltopectoral flap, previously raised and lined with split skin, may be used, or a combination of a forehead flap on the inside and a deltopectoral flap on the outside of the cheek. Adequate size of flaps should be used to recreate buccal sulci so as to permit movement of the mandible.

Neck nodes are treated by pre-operative radiotherapy to 4000 rad and then block dissection of the neck 6-8 weeks later.

The Tongue

Developmental Anomalies

Tongue-tie.- The lingual frenum is short, often thicker than normal and fibrous. It holds the top of the tongue close to incisal edges of the lower central incisors. Attempts to raise or protrude the tongue result in eversion of the lateral margin and a heaping up of the mid-portion of the dorsum. More often than not the deformity causes no disability and no action is required. However, it may result in lisping. The patient can also have difficulty in cleaning the backs of the lower front teeth. Where division is necessary a small quantity of local anesthetic solution is injected with a fine needle. A traction suture is passed through the tip of the tongue and the frenum divided with scissors just below the undersurface of the tongue until adequate mobility of the tip is achieved. The wound takes on a linear shape in a vertical direction if the tip of the tongue is raised and may be closed with fine, plain catgut.

Congenital Fissured Tongue.- Fissures of varying depth run laterally from a median groove, but the surface between is covered with normal papillae. Sometimes the fissures become infected with candida albicans (the syphilitic tongue tends to be bald and with longitudinal fissures).

Lingual Thyroid.- Produces a reddish lobulated mass behind the foramen caecum (Chapter 37).

Median Rhomboid Glossitis

A smooth, lobulated, oval, or triangular patch immediately anterior to the foramen caecum. The mucosa may be of a rather deeper colour than the rest of the tongue and firm. Chronic infection of the fissures between the lobules with candida albicans is not uncommon. The condition may be mistaken for carcinoma which is rare in the midline of the tongue.

Geographic Tongue (Glossitis migrans)

Small red patches develop with a white, furred margin. The patches spread and recede in an irregular fashion and fresh patches appear. Keratinised epithelium and inflammatory cells accumulate on the filiform papillae to form the white margin and are then shed. The condition is quite benign but the real aetiology is unknown. It is more common in patients with congenital heart defects. It is also seen in those with acute gastrointestinal problems.

Macroglossia

A variety of developmental conditions and hamartomas can result in a persistent, painless, enlargement of the tongue. The tongue may be of normal structure, but large in proportion to the oral cavity. The patient can usually retract it into the mouth, but is able to protrude it to a surprising degree. Neurofibromatosis may be a cause of macroglossia, but often one side only is affected. Cavernous haemangiomas also tend to produce unilateral enlargement while lymphangiomas tend to occur bilaterally and produce a thick and cumbersome tongue which prevents closure of lips and jaws. All developmentally enlarged tongues exert continuous pressure on the teeth and alveolar process resulting in spacing of the teeth and proclination of the incisors.

Elongation of the tongue may be corrected by wedge resection from the midline of the anterior third or more. Vertical thickening may be reduced by wedge-shaped slices removed from the lateral margins. Care should be taken not to damage the nerve supply or the major branches of the lingual artery. While the tongue is a very vascular organ, damage to both lingual arteries can lead to problems with wound healing.

Acquired Causes of Enlarged Tongue

Acromegaly.- The tongue enlarges in a similar fashion to the lips, nose, liver, etc.

Amyloidosis.- Deposits of amyloid in the tongue in primary amyloidosis may cause macroglossia.

Cretinism.- In long-standing hypothyroidism the tongue is enlarged as a result of an accumulation of a mucoproteinous material in the tissues.

Laceration of the Tongue

Lacerations most commonly arise as a result of the patient biting his tongue. Epileptics may do this during the clonic convulsive phase of a grand mal attack unless a suitable pad, gag or rubber ring is placed between the posterior teeth. Fractured jaws sustained during road traffic accidents may also be associated with a laceration of the tongue if this organ lies between the teeth during the accident. A blow on the face or a fall while the patient is smoking a pipe can result in the stem breaking and lacerating the tongue. In an unconscious patient the brisk haemorrhage which follows such an injury can endanger the airway.

Severe haemorrhage as a result of damage to the lingual artery can be controlled by hooking the tongue forward with a finger and compressing the tongue against the mandible and between the fingers and a thumb in the submental region. Fortunately the tongue has such a good blood supply that quite extensive lacerations may be sutured with every hope of sound healing.

A *closed injury* of the tongue or a fracture of the mandible in an elderly patient can result in a haematoma which spreads in the tongue and floor of mouth, eventually producing respiratory embarrassment so that a tracheostomy may be required.

Inflammation of the Tongue

Pyogenic infections of the tongue are extremely rare. There may be some oedema of the tongue associated with a cellulitis of the sublingual space and in *Ludwig's angina* (Wilhelm von Ludwig, 1790-1865. Professor of Surgery and Midwifery, Tübingen, Germany.) the infection spreads backwards between the hyoglossus and genial muscles, but not often into the tongue proper. The tongue of course is raised and protruded from the mouth in these conditions because of the swelling of the floor of the mouth.

Great swelling of the tongue can follow a *wasp sting.* Classically this occurs when a holiday-maker drinks from a beer bottle which has been left open.

Angioneurotic oedema, or hereditary angio-oedema may affect the tongue to produce life-threatening swelling. The insertion of a nasopharyngeal airway is the quickest way to establish an airway behind a swollen tongue, but in some circumstances a tracheostomy may be required.

Tuberculosis can affect the tongue in two ways: to produce shallow, oval, indolent, painful ulcers with overhung margins, or to produce a circumscribed, interstitial tuberculoma. Both types of lesion only occur in advanced, untreated tuberculosis.

Syphilis may produce lesions of the tongue at each stage of the disease.

Primary syphilis of the tongue. An extragenital chancre can occur on the tongue. The submaxillary and submental lymph nodes become greatly enlarged, as in the case of a similar lesion on the lip. There is a comparative lack of pain compared with other ulcers of the tongue.

Secondary Syphilis

1. Multiple shallow ('snail track') ulcers may be present on the sides and undersurface of the tongue.

2. Mucous patches occur on the tongue and on the fauces.

3. Hutchinson's wart (Sir Johnathan Hutchinson, 1828-1913. Surgeon, The London Hospital.), really a condyloma, is always found in the midline.

Tertiary Syphilis

1. The, now rare, gumma starts as a slowly enlarging, non-tender, midline swelling. Following necrosis of the overlying tissues the wash-leather slough separates to leave a deep, crater-like ulcer. Two features distinguish the gummatous ulcer from a carcinoma: firstly, carcinomas very rarely arise in the midline of the tongue and secondly there is no tethering of the tongue with a large gumma.

2. Multiple small gummas heal with scarring to produce fissures.

3. Chronic superficial glossitis causes a loss of papillae on a tongue which is also fissured due to the underlying interstitial glossitis. The result is a bald, lobulated tongue. The epithelium becomes hyperkeratotic and squamous cell carcinoma tends to arise in the resultant leucoplacia. There is a reduced blood supply to the syphilitic tongue due to endarteritis, and radiotherapy may be contraindicated as a treatment of carcinoma arising in a tongue affected by chronic superficial glossitis. The increased ischemia which follows the radiotherapy may lead to necrosis.

Hyperkeratosis and Leucoplacia

In the mouth, a hyperkeratotic epithelium is kept moist by the saliva and appears white. The term 'keratosis' is usually used where the aetiological factor is readily recognised and where withdrawal of the irritant tends, in time, to regression of the white patch; for example, a friction keratosis, or a smoker's keratosis. Leucoplacia is also used as a descriptive term for a white, hyperkeratotic patch in the mouth, or on certain other mucous membranes. It is used in different ways by different clinicians, but in general is used where the aetiological agent is not recognised or known or where the lesion has become established to a point where it no longer regresses when the causative irritant is withdrawn. Some pathologists require that the epithelium should exhibit dyskeratosis on histological examination of a biopsy.

Tradition has it that **S**moking, **S**yphilis, **S**epsis, the **S**harp edge of a tooth (chronic frictitional irritation), **S**pirits and **S**pices are the causes of leucoplacia (the Six **S**s). There is more than a grain of truth in this tradition and to this list should be added *candidiasis.*

The majority of ulcers caused by broken, carious teeth or dentures are innocent, but from time to time hyperkeratosis and a carcinoma is found just where the sharp edge rubs against the tongue. Syphilis, as we described above, causes chronic superficial glossitis in which leucoplacia develops, again with malignant change a likelihood.

Smoking causes hyperkeratosis of the hard and soft palate. The mucous gland ducts, plugged with keratin, stand up as prominent points. Hyperkeratosis also develops on the cheek behind the commissure of the lips, on the floor of the mouth, where chemical-laden saliva flows, and on the lips where a cigarette or cigar is held. A white patch may also develop on the tongue opposite the end of the cigar or pipe stem. Leucoplacia and squamous cell carcinoma are most likely to develop if spirit drinking is added to smoking, but mainly of tongue or floor of mouth. Carcinoma rarely arises in the palatal smokers' keratosis.

The classical 'spice' is betel nut. Areca (betel) nut mixed with tobacco, spices and slaked slime is wrapped in a betel leaf (from the plant *Piper betle*). Leucoplacia develops adjacent to where the 'pan' is normally held in the mouth, usually on the cheek and the side of the tongue. Another leaf, Qat, is chewed in the Yemen.

The relationship of candida albicans to leucoplacia is incompletely understood. Leucoplacia appears to arise following long-standing candida infection *(chronic hypertrophic candidiasis).* In other instances the candida seems to be a secondary invader of the hypertrophic epithelium. The clinical appearance may be speckled white on a pink background rather than a uniform white.

Speckled leucoplacia is particularly prone to develop a carcinoma. Initially in the development of leucoplacia there is a hyperkeratosis with a small increase in the prominence of the rete pegs. Lymphocytes and plasma cells accumulate in the dermal papillae. At this stage there is a milky blush on the mucosal surface. Later there is a marked increase in the thickness of the epithelium, the rete pegs elongate (acanthosis) and the round cell infiltration becomes quite dense. Mitoses increase in number among the basal cells. Clinically there is now a distinct, paint-like, smooth, white patch which is dry and rough to the touch. Finally mitoses appear in the cells above the basal layer and some cells become keratinised before reaching the surface (dyskeratosis). The leucoplacia is then only a step away from carcinoma *in situ* and the development of a frank squamous cell or verrucous carcinoma. At the dyskeratotic stage patchy loss of the keratinised layer tends to occur to give a patch-work appearance of red and white. A warty appearance of the surface heralds the development of a carcinoma.

Opinions vary as to the risk of carcinomatous change and what is advised depends partly on the extent of the lesion and partly on the judgment of an experienced clinician. The patient should be counselled to give up identified irritants, then early lesions may disappear after some months but will return promptly if the old habits are resumed. Small white patches may be excised and large ones biopsied. A prolonged treatment with local nystatin or amphotericin B should be tried if candida are present. Hyperkeratotic lesions without premalignant change should be watched and monitored by exfoliative cytological examinations from time to time.

Dyskeratotic lesions and carcinoma *in situ* should be excised and the area grafted.

Where there is a likelihood of malignant change, this may not be confined to the obvious white patches however. In some months the whole of the mucosal surface is unstable and at risk. Atrophic red areas are as likely to undergo malignant change as the white patches.

Radiotherapy will get rid of the leucoplacia, though the hyperkeratosis tends to reappear after months or years. Unfortunately there is evidence that radiotherapy increases the likelihood of malignant change.

Other Hyperkeratotic Lesions.- Other conditions which produce white patches are: *lichen planus,* which characteristically produces a striated and lace-like appearance, but may form a plaque. White patches are sometimes seen in *discoid lupus erythematosus.* The *white sponge naevus* has a folded appearance and covers both cheeks and the floor of the mouth. It is inherited in some cases.

The symmetrical white patch of the underside of the tongue and sublingual region is also considered to be a developmental lesion, but can give rise to a squamous cell carcinoma.

Leucokeratosis mucosae oris is another congenital abnormality in which all the oral mucous membranes have a filmy white appearance.

Submucous Fibrosis.- Submucous fibrosis produces a mottled or marbled pallor of the mucosa of cheeks, palate, tongue and gingivae, but in this case the pallor is due to

collagen deposited in the submucosa. The lips and cheeks become stiff and lose their elasticity. Bands of fibrous tissues develop beneath the mucosa and limit jaw movement so that opening of the mouth becomes greatly restricted. It is thought that the condition results from a hypersensitivity to chilli. Some of the chemical constituents of the betel nut chew (Pan) also may be causative (see leucoplacia). Squamous cell carcinoma is a not infrequent complication.

Hairy Tongue.- The keratinised layer fails to desquamate normally from the filiform papillae and becomes greatly elongated to resemble a coating of hair. The 'hair' may take on various colours due to the presence of chromogenic organisms to produce a black or brown hairy tongue. Sometimes, following the use of antibiotics, the normal filiform papillae becomes slightly lengthened by keratin and coloured black by antibiotic-resistant organisms such as fungi. This form of black hairy tongue clears up soon after the use of the antibiotic ceases. True hairy tongue is more intractable, but can be kept under control by mechanical scraping. Both conditions must not be confused with the normal furring of the tongue in an ill patient. Dehydration and lack of mechanical cleaning in a patient who is not eating normally combine to let keratinised cells accumulate on the filiform papillae. Bacteria and food debris accumulate among the papillae. Heavy smoking increases the keratinisation of the papillae and stains the fur black and brown to make it more obvious.

Ulcers of the Tongue

It is advisable at this stage to review the more common types of ulcers of the tongue, as their differential diagnosis is so important.

Dental Ulcers.- Mostly occur at the side of the tongue and an obvious cause will be seen. This may be a carious and broken tooth, or a broken denture. Usually the ulcer lies in relation to the sharp object at rest so the relationship is not apparent when the tongue is protruded for inspection. More extensive oval ulcers may occur to one side of the upper surface in relation to a sharp palatal cusp of an upper tooth, or even close to the midline and related to a rough or fractured surface of an upper denture. The dorsum of tongue is reasonably tough but once a rough surface is discovered the patient may explore it and habitually run the tongue against it.

If the ulcer does not heal promptly when the apparent cause is removed, or if there are features suggestive of carcinoma, it *must* be biopsied. Often a biopsy will provoke an indolent, innocent ulcer to heal.

Post-pertussis ulcers occur on the upper part of the lingual frenum and the undersurface of the tip in patients who protrude the tongue over the lower incisors during bouts of coughing; typically with whooping cough.

Aphthous ulcers (see above).

Ulcers in lichen planus, pemphigus, erythema multiforme, etc (see above).

Syphilitic ulcers (see above) occur typically in the midline.

Tuberculous ulcers (see above) occur on the dorsum, towards the midline or near the tip and irregular with undermined margins. Pale, indolent, granulations in the floor, sometimes with a thin slough.

Malignant ulcer (see below).- This is usually a squamous cell carcinoma, but may be a salivary adenocarcinoma or a lympho-epithelioma when the ulcer is on the posterior third.

Neurological Lesions of the Tongue

Common site of surgical damage to the *lingual nerve* are at the upper pole of the submandibular salivary gland, beneath the mucoperiosteum lingual to the third molar and where the nerve crosses beneath Wharton's duct (Chapter 31) in the floor of the mouth. Return of sensation may be imperfect (paraesthesia), or accompanied by causalgic pain. Another cause of lingual neuralgia is as an aftermath of herpes zoster. Pain and anaesthesia, often with referred pain to the ear, result from invasion by a malignant neoplasm.

Hemiatrophy follows damage to the *hypoglossal nerve,* either during the extracapsular removal of the submandibular gland, or as the carotids are approached, high in the neck during a block dissection. In the former location the nerve is accompanied by easily damaged *venae comitantes* and the nerve can be included in hastily applied artery forceps. Paralysis of the musculature on the same side and deviation towards the affected side on protrusion are seen.

Glossodynia occurs typically in middle-aged and elderly women. Often no cause can be found. Sometimes there is an atrophic candidiasis. Sjögren's syndrome (Chapter 31) and diabetes are two possible underlying conditions which should be considered if this is so. Some middle-aged patients complain of an unpleasant change in taste. The abnormal sensation can be confirmed by appropriate taste tests. The cause is unknown.

Benign Neoplasms

Benign neoplasms are uncommon, compared with squamous cell carcinoma.

Papilloma (see above) must be distinguished from the rare Hutchinson's wart (see above) which resembles a pinkish, soft, cauliflower-shaped papilloma. Papillomas are readily excised, or if multiple can be treated with a small cryoprobe.

Fibro-epithelial polyps (see above), usually small and opposite a source of mild, chronic irritation such as a gap between the lower incisor teeth.

Pregnancy tumour (Chapter 33), usually found on the gum, but can occur on the tongue.

Haemangioma and lymphangioma (see above).

Plexiform neuroma and neurofibroma (see above).

Lipoma is another uncommon entity recognised by its softness and yellow colour. Rarely gets large before advice is sought.

Osteoma of the tongue is a clinical curiosity. A hard swelling found beneath the foramen caecum or in the posterior third of tongue.

Sublingual varicosities should not be confused with a cavernous haemangioma. The sublingual veins are greatly dilated and tortuous in this condition. Initially small veins enlarge to produce a caviare appearance, then the larger ones. Not uncommon in the elderly. Reputed to indicate chronic heart failure, but this is not so.

Granular cell myoblastoma produces a firm mass in a mobile tongue. The overlying mucous membrane is smooth and often hyperkeratotic. A biopsy of the epithelium alone reveals pseudo-epitheliomatous hyperplasia which may be mistaken for carcinoma. A deeper biopsy reveals the eosinophilic granular cells. A benign, but not encapsulated lesion which is cured by conservative local excision.

Juvenile fibroma produces a softer, central, slowly enlarging lump. At operation this benign tumour has no readily discerned margin from the adjacent muscle, consequently is not easy to remove, particularly as it tends to occur in the small tongues of young children.

Carcinoma of the Tongue

Since 1910 there has been a decreasing incidence of carcinoma of the tongue in *males.* Possible factors bringing this about are: more efficient treatment of syphilis so that while this disease is still common, the late effects are rare; the passing of the clay pipe, improved standards of oral hygiene and oral health, and possibly a decrease in the consumption of spirits. While the reduction of the incidence in males is gratifying, there has been an increase in females (Russell) (Marion H Russell, 1907-1966. Medical Statistician, Christie Hospital, Manchester, England.), possibly related to an increase in female smokers, so that the sex incidence is now equal.

Clinical Features. As in carcinoma in other situations, early stages of the disease are virtually symptomless. Observant and fastidious patients may seek advice for a comparatively small lesion. Others may mention it in passing to doctor or dentist. Because the mouth is so easily examined, doctors should inspect the oral cavity and tongue whenever the fauces need to be examined. Dentists likewise should be on the look out for suspicious lesions when they examine a patient.

Carcinoma of Tongue may Present as:

1. An oval, raised, papillated plaque with white keratin flecks on the surface.

2. An ulcer with sloughing from an indurated base and with everted margins.

3. A deep, often infected, fissure with surrounding induration.

4. A lobulated, indurated mass, possibly with seemingly normal mucosa over it, sometimes with yellow patches of submucosal necrosis which appear like pointing abscesses.

Many patients seem to disregard the lesion in its early stages or are too frightened to seek help until a large and offensive tumour is present. These more advanced cases present with:

1. *Pain in the tongue,* which is initially due to infection and ulceration but later is due to involvement of the lingual nerve. Once the lingual nerve is involved there may be referred *pain in the ear.* Pain on swallowing or pain in the back of the tongue may mean a carcinoma of the posterior third. In such cases careful inspection of tongue and vallecula in the conscious patient with a laryngeal mirror, or direct examination with a laryngoscope with the patient under anaesthesia, is essential. The posterior third can be palpated, but induration due to neoplasm is not easily appreciated because of the lumpiness of the lingual tonsils.

2. *Salivation.*- Pain promotes salivation and a stiff, lumpy, partially fixed tongue makes swallowing difficult. The elderly man sitting in outpatients and spitting into a handkerchief may well have a carcinoma of tongue.

3. *Ankyloglossia.*- Inability to protrude the tongue or deviation to one side means it is fixed by extensive infiltration of the floor of the mouth.

4. *Dysphagia.*- Difficulty in swallowing may occur with any advanced lingual carcinoma, but is more pronounced when the growth is in the posterior third.

5. *Inability to articulate clearly.*

6. *Foetor*.- Once necrosis occurs the lesion becomes grossly infected and highly offensive.

7. *A lump in the neck* due to secondary deposits in the draining lymph nodes.

8. *Alteration in the voice* may be a first indication of a carcinoma of the posterior third. In this inaccessible situation a tumour may escape notice. Such tumours are more often anaplastic than those of the anterior third and a large tumour with nodes in the neck may be present by the time the diagnosis is made.

Spread of the Disease

Local Spread.- Carcinoma of the anterior two-thirds usually starts on the lateral margin and frequently reaches the floor of the mouth before it extends across the midline. At the junction of anterior two-thirds and posterior third it may invade the mandible. Carcinoma of the posterior third spreads laterally into the tonsil, side of the pharynx and cervical spine and up into the soft palate, posteriorly into the epiglottis and downwards towards the larynx, and across to the other side of the tongue.

Lymphatic Spread.- Metastasis to the submandibular nodes, particularly if the floor of the mouth is involved, means that tumour is already present close to the periosteum of the mandible and resection of the relevant segment of the mandible may be necessary to ensure surgical clearance. Once the anterior part of the floor of the mouth is involved bilateral spread to neck nodes is common.

Blood Stream Spread.- Death tends to occur as a result of uncontrollable primary tumour or lymphatic spread so that distant metastasis rarely manifests itself. Metastasis by the blood stream is more likely from posterior third than anterior two-thirds tumours.

Terminal Events

Death from uncontrolled primary tumour occurs as a result of:

(i) Inhalation bronchopneumonia as a result of inhalation of infected material from necrotic neoplasm.

(ii) Haemorrhage from erosion of the lingual artery, or in the case of a carcinoma of the posterior third, erosion of the internal carotid.

(iii) Combined cancerous cachexia and starvation due to the pain and difficulty of attempting to swallow where a large tumour replaces the tongue, or where there is compression of pharynx or esophagus by metastatic deposits in lymph nodes.

(iv) Asphyxia, which is most often due to pressure on the air passages from enlarged and fixed carcinomatous lymph nodes. Sometimes obstruction is due to oedema of the glottis.

Treatment

Surgery or radiotherapy or a combination of the two. The most appropriate use of chemotherapy in relation to the main methods of treatment is not yet settled.

Establishment of the Diagnosis.- An adequate biopsy must precede treatment. But there is no place for the so-called excision biopsy. Only rarely will such an excision be generous enough to avoid incomplete removal and local recurrence. Indeed should the lesion prove not to be malignant, unnecessary mutilation will have been inflicted by such an excision.

Preliminary Preparation.- Teeth are scaled and polished and oral hygiene established. Where the surface of the lesion is necrotic or infected, frequent irrigation with eusol is required. Any teeth which will be in a field of substantial irradiation should be extracted.

Tests for syphilis should be carried out and treatment initiated if necessary. In some cases a diagnosis of syphilis will mean that radiotherapy is not advised.

Definitive Treatment

1. *Carcinoma in situ* may be excised with a 1 cm margin and to a depth of about 0.5 cm. Excision wounds on the lateral border may be closed by undermining and advancing mucosa from the floor of the mouth. Larger defects are grafted with split skin.

2. *Lesions less than 2 cm* are treated primarily with radiotherapy, because if treatment is successful there is virtually no functional disability. For lesions of the anterior two-thirds of the tongue of this size, and where there are no palpable lymph nodes, interstitial irradiation with ^{137}Caesium needles or ^{192}Iridium wire is used. A close watch is kept upon the patient, with follow-up appointments at monthly intervals. If regression is incomplete, or recurrence occurs, or neck nodes become suspiciously palpable, surgery is added.

3. *A small carcinoma confined to the lateral border* may be removed by local excision with a generous 1.5 cm margin beyond visible and palpable disease. The configuration of the resection depends upon the site of the tumour, but approximates to a hemiglossectomy. The success of the operation depends upon the care with which the excision is carried laterally below the neoplasm for it is here that the operator may fail to maintain an adequate margin.

If there are palpable nodes a block dissection of neck is required. Advantage should be taken to tie the lingual artery before the primary lesion is resected as bleeding will be reduced and surgical precision increased. An in-continuity resection facilitates an adequate margin on the deep aspect of the primary tumour.

If a block dissection becomes necessary resection of the primary site should be considered even if it is apparently controlled by previous radiotherapy. Pre-operative irradiation of the neck to between 4000 and 4500 rad may be advisable.

4. *Primary lesions larger than 2 cm* are usually irradiated by external beam irradiation. The submandibular nodes may be included in the field of irradiation even if no nodes are palpable. Great care is required with follow up to avoid extensive spread occurring should the treatment fail to achieve healing. Large tumours require a submandibular dissection to enable adequate excision. Because this makes a subsequent block dissection difficult a prophylactic neck dissection should be considered if local control is not achieved, even if no nodes are palpable.

5. *If there is a large tumour with palpable nodes* both primary and the neck are irradiated to 4500 rad then, 6-8 weeks later, an in-continuity resection is carried out. (Surgery should be undertaken within three months of the irradiation but after the reaction has subsided.) Where the tumour extends on to the floor of the mouth, or up to the anterior pillar of the fauces, the mandible should be included in the specimen.

6. *Tumours which involve the tip of the tongue* may metastasize to the submental nodes. Tumours which involve the anterior part of the floor of the mouth are likely to metastasize to both sides of the neck. Prophylactic irradiation of the second side should be considered, even if nodes are not palpable.

7. *Bilateral neck nodes* may be dealt with by various strategies without sacrifice of both internal jugulars. Resection of both internal jugulars is to be avoided because of the high mortality due to post-operative cerebral venous engorgement and oedema of the head and face should the patient survive.

(i) Bilateral block dissections may be carried out, but the internal jugular left on the least involved side. The operation should be completed first on the side in which the vein is to be retained.

(ii) Alternatively low-dose pre-operative irradiation should be given to the worst side followed by a block dissection and a therapeutic dose given to the other side. Contrary to previous opinion, squamous cell carcinoma metastases in lymph nodes will respond to megavoltage irradiation. The problem is that recurrence of tumour after irradiation of nodes is often not amenable to salvage surgery.

(iii) A full block on one side and a suprahyoid block on the other is another possible form of treatment.

Pre-operative irradiation of the neck reduces the incidence of diffuse, local recurrence of tumour which is difficult to differentiate from post-operative induration.

8. *Posterior extension of the carcinoma* requires division of the lower lip and section of the mandible for access. The incision should be vertical through the lip, curved around the chin pad, and then broken into a **Z** until the hyoid crease is reached. Posterior third primary tumours are treated almost entirely by external beam irradiation as they are rarely amenable to surgery.

Where the anterior part of the mandible is sacrificed with a carcinoma involving the floor of the mouth, a Bowerman-Conroy implant (John Ernest Bowerman, Contemporary. Oral Surgeon, Westminster Hospital and Queen Mary's Hospital, Roehampton, London.) (B Conroy, Contemporary, Senior Chief Maxillo-Facial Technician, Queen Mary's Hospital, Roehampton London.) is inserted and the hyoid and tongue suspended from it (Wilson) (John Samuel Pattison Wilson, Contemporary. Plastic Surgeon, Westminster and St George's Hospital, London.).

Repair.- The tip of the tongue may be repaired by suture. Small lateral defects can be closed by advancing floor of mouth mucosa.

There are two ways in which mobile raw surfaces in the mouth, like those resulting from section of the tongue, can be covered with a Thiersch graft (Chapter 10); by quilting on the graft (McGregor) (Ian Alexander McGregor, Contemporary. Consultant Plastic Surgeon, Canniesburn Hospital, Glasgow.) and by use of a foam plastic pad (Seward) (Gordon Robert Seward, Contemporary. Professor of Oral Surgery, The London Hospital, London.). In McGregor's method small, plain, interrupted, catgut sutures are placed at the corners of squares across the graft after suture of the periphery. Tiny stab incisions are made in the centres of the square to let out any exudate or haematoma. Alternatively (Seward), a pad of 1 cm thick polyurethane foam is cut slightly larger than the defect, and the Thiersch graft is draped over it, wrapped round, and sutured to the outer surface. A suture transfixes the centre

of the pad, picks up the centre of the defect and passed back through the pad and is tied. The margins of the pad are then sewn to the margins of the defect.

Where there is a sizeable loss of tongue a forehead flap makes an excellent repair. The forehead flap is brought into the mouth deep to the zygomatic arch. Adequate space to bring it through may be created by resecting the coronoid process, by sectioning the zygomatic bone or by resection of the anterior part of the temporalis muscle.

For very large defects of tongue and floor of mouth a delto-pectoral flap must be brought in via the submandibular wound. It can be de-epithelialised over a strip to enable the oral wound to be sealed by a suture line. Where a block dissection of neck has been performed a pectoral myocutaneous flap may be brought up to repair the tongue. The pedicle, composed of a strip of pectoralis major muscle and vessels can be used to cover the carotids under the neck skin flaps.

Palliation.- Where there is a large, unresectable primary tumour or where there are already fixed nodes in the neck a full dose of therapeutic irradiation should be delivered to the tongue. Where irradiation has been used already and either primary or the nodes are unresectable, resection of the primary alone may make the patient more comfortable. Necrotic lingual carcinoma is difficult to tolerate for the patients, the relatives or the attendants. Irrigation of the mouth with eusol and hydrogen peroxide can reduce the local infection and the foul smell.

In some patients chemotherapy with the Price-Hill regimen (Leonard Anthony Price, Contemporary. The Institute of Cancer Research and the Royal Marsden Hospital, and Bridget T Hill, Contemporary. Imperial Cancer Research Fund.) may be helpful and indeed, in some patients, of substantial benefit. This treatment involves the administration of a carefully selected list of cytotoxic drugs to a precise schedule. In 1975 Price and others reported a trial involving the use of the following drugs in the treatment of oral squamous cell carcinoma, including cases of carcinoma of the tongue: Vincristine, Adriamycin, Bleomycin, Methotrexate, Fluorouracil, Hydroxyurea and Mercaptopurine.

Rapid advances are being made in this field, and clinicians intending to use this form of treatment would be advised to make themselves familiar with the most recent papers and with the pharmacology and toxic effects of these highly potent drugs. Strict adherence to the chosen advocated regimen is necessary if unexpected problems are to be avoided. Great care must be taken also in the preparation and administration of these substances to avoid dangers to the staff involved.

Difficulty with swallowing may require the patient to be fed via a nasogastric tube and respiratory obstruction may require a tracheostomy. Adequate analgesia and sedation with morphine, or the 'Brompton cocktail', should be used to control pain and apprehension (Chapter 63).

Prognosis.- Despite advances in the treatment of cancer and significant improvement in the functional results achieved for patients with carcinoma of tongue, the overall mortality at 5 years has remained surprisingly constant. The prognosis for women is better than men

and approaches 50% survival at 5 years while no more than 25% of men are alive at the end of 5 years.

Sarcoma of the tongue is rare and usually fatal. **Malignant melanoma** of the tongue is also rare and has a bad prognosis, although vigorous local treatment may eradicate the primary tumour and permit a less distressing death from metastases.