LEUKOPLAKIA

Gk.: Leucos - white, plakos - patch or tablet.

Oral leukoplakia, hyperkeratosis and dyskeratosis of the oral mucous membranes. It was first named by Schwimmer in 1887, prior to this it was called ichtyosis, tylosis or psoriasis oris.

Butlin (1885) includes the terms leucoma, leukoplakia, psoriasis, ichtyosis, tylosis, keratosis and plaques opalines. Although by far the most occur in the mouth, the other tracts are affected, the upper airways and the gastro-intestinal tract; the involvement of the oesophagus is usually concomitant with chronic oesophagitis and varices. The urogenital tract, the vulva and glans penis being affected, and although it is rarer here it is considered to be a prodrome of Carcinoma. Hobaek (1946).

The rectum and the anal canal may be involved by an atypical Leukoplakia. Zegarelli and Kutscher (1957).

It is extremely important to accept at this stage that the term leukoplakia is not acceptable as a pathological diagnosis. Kollar et al (1954). There are a number of whitish mucosal lesions, all similar in appearance but of varying aetiology, which are but a clinical expression of disorders in the maturation of oral epithelium. Some white lesions accompany certain diseases; Diphtheria, Fusospirichaetosis, Thrush, Syphilis and Yaws. Surface coagulation by chemical and physical agents produces a white coagulum, which is readily wiped away and should provide no problems of differential diagnosis. Common agents in this field are: Zinc chloride, Silver nitrate and Acetylsalicylic Acid. Kollar et al (1954).

Leukoplakia is seen in about 1 of every 500 adults examined, mainly in the fourth, fifth and sixth decade, although it is not uncommon in the third. Burket (1957).

Hobaek (1946) considers the greatest incidence is between the fifth and seventh decade, although it is not rare during the

third. It is very rarely seen in coloured races and is chiefly confined to males, although a 5% incidence in women has been reported. The exact aetiology of leukoplakia is not known, in most cases both systemic and local factors are thought to be involved. Chronic irritation of a predisposed mucous membrane by a mechanical, less often by a thermal, chemical or electrogalvanic irritant (Leukoplakia Electrogalvanica is frequently accompanied by a metallic taste) are thought to be aetiologically important. Hobaek (1946). Hot and heavily spiced foodstuffs; alcohol; Betel Nut and Catechu are considered to be factors in the irritation of the delicate oral mucosa.

Simple trauma of a chronic nature arising from the sharp edges of teeth, roots and prosthetic appliances, and unconscious habits of biting oral tissues may give rise to hyperkeratosis. Burket (1957).

Leukoplakia appears to develop under dentures only when they are ill fitting. Leukoplakia and malignant degeneration may follow the repeated application of caustic agents to the oral mucosa.

Tobacco in any form is an important local cause although Hobaek (1946) considers Smokers Leukoplakia is essentially different from True Leukoplakia since it is characterised by spontaneous regression when the irritant is removed. Leukoplakia and its relation to the use of tobacco has been well dealt with by Cummer (1946).

Van Reenen (1954) has dealt quite fully with the effects of tobacco smoking on the oral cavity and considers that both chemical irritation by the tars, nicotine and phenolic bodies in addition to thermal irritation by both smoke and the hot stem (of a pipe) have a part in the insult to the mucosa.

Although the French writers considered Syphilis to be the principal cause of Leukoplakia, it is thought by others to occupy a lesser place Hobaek (1946), Thoma (1954).

Thirty percent of patients with Leukoplakia of the tongue have a syphilitic background, Schaffer (1951-52), and hypovitaminosis A and B complex are considered by Burket (1957).

Arnott (1958) is impressed by the results he has obtained by Vitamin C administration.

A hyperkeratosis has been produced by injected Estrogenic Hormones, by a hypercholesteremia and also by allergy. (Leukoplakia allergica.) In a few cases it has been associated with the Plummer-Vinson Syndrome, Hobaek (1946).

Pindborg (1947 (I), 1949 (II), 1949 (III)) correlates the use of tobacco with the development of gingivitis, (ulcero-membranous and chronic marginal) and the deposition of calculus.

The oral tissues undergo desiccation whilst one is smoking, from the heat, smoke and partly open mouth. Initially Stomatitis Nicotina begins as a simple mucosal rubor which is often localised, according to the individual idiosyncrasies of the smoker, "Inhalation through a pipe stem affects the palate, chewing the butt of a cigar bathes the floor of the mouth with tobacco juice, chewing tobacco does the same, while the heat of the pipe stem may cause lesions of the lips. Exhalation of the smoke through the corner of the mouth affects the buccal mucosa near the corner of the mouth", Thoma (1954).

The lesions progress from a simple rubor (for example Smoker's palate) to a hornified mucosa studded with grey flat-topped papules, or red hemispheric papules. These lesions are painful to touch and there may be an accompanying stomatopyrosis and stomatodynia. Abstaining from tobacco in any form frequently allows this to clear up, if not, a protective prosthesis can be constructed. A relatively small number of cases suffer malignant change. Cummer (1946).

Clinically there are two types described by Hobaek (1946). Leukoplakia plana is a symptomless, greyish-white discolouration of the mucosa which represents the initial stage of every Leukoplakia. The lesion is sometimes finely wrinkled and may

remain stationary for up to fifteen years. Its transition to Leukoplakia verrucosa is fluid and the period may be of short or long duration. The lesion becomes thickened and infiltrated, rhagades or small ulcerations form and may be responsible for some pain with spicy foods and hot drinks. If the tongue is affected the lesion frequently becomes papillomatous.

The distribution within the oral cavity is not fixed and it may be localised or generalised (to the mouth). Hobaek (1946) gives a pattern of distribution in his very excellent paper, suffice to say anywhere within the mouth but less frequently on the uvula and soft palate. Commonly the colour is grey-white to milk white but a certain opalescence is not rare and less commonly it is pure grey with a brown or yellow tinge, due perhaps to minor haemorrhage. Hobaek (1946).

There is a great deal of misunderstanding as to what constitutes a diagnosis of Leukoplakia, Kollar et al.(1954) has suggested a classification of the disorders of epithelial maturation. Hyperkeratosis simplex is the term he applies to epthelial hyperplasia; hyperkeratosis, and hyperkeratosis with inflammation. Usually removal of the irritant results in its remission and the patient should be reassured of its benign nature. Periodic biopsy, however, is necessary if it persists.

Hyperkeratosis complex is the term applied when dyskeratosis occurs in addition to hyperkeratosis and inflammation; it is only a short step from here to the intraepithelial cancers and the Epidermoid Carcinomas.

Kollar et al. (1954) recommends that the term Leukoplakia be discontinued as a diagnosis for it is misleading. They further consider that biopsy is essential in the diagnosis and the management of every persistent white lesion of the oral mucous membrane.

Simple forms of Leukoplakia will often resolve when local irritants are removed and general disorders corrected. Surgical excision, Radiation therapy, Electrodissection and endothermy may be necessary in some cases. Malignant changes occur and must be dealt with accordingly Burket (1957) Thoma (1954). In a tentative diagnostic test to differentiate lichen planus and cheek trauma from Leukoplakia, the tissues are dried and Lugols Iodine solution is applied; areas of hyperkeratosis associated with Leukoplakia will not take on the deep mahagony stains because of the decreased glycogen content of the cells comprising the lesions. Burket (1957).

The differential diagnosis of Leukoplakia from other white plaques may in some cases be difficult.

Pachyonychia Congenita was first described by Judassohn in 1906, Everett and Noyes (1953) and is a congenital and sometimes familial malady characterised by dystrophic changes in the fingers and toe nails and occasionally in the hair and the cornea. There is hyperkeratosis of the palms and soles and hyperhydrosis of the hands and feet. Oral lesions are nearly always present and may occur on the buccal mucous membrane and tongue or throughout the entire mouth. Angular cheilosis is commonly present. Gorlin and Chaudhry (1958). Everett and Noyes (1953) (quoting Judassohn) describes the appearance of white lines on the under surface of the tongue.

Histologically the epithelium is acanthotic; there is uniform intracellular vacuolisation and the intracellular bridges in the prickle cell layer are missing; Parakeratosis is marked and the stratum granulosum is absent.

Pachyderma Oralis, or as it may also be called White folded gingivo stomatitis Everett and Noyes (1953) familial white folded hypertrophy of the mucous membrane Zegarelli and Kutscher (1957), white spongy naevus of the mucosa, congenital leukokeratosis of the oral mucosa, Kinney and Derifield (1956) and leukokeratosis oris.

Pachyderma oralis is an atypical Leukoplakia according to Kinney and Derifield (1956) who report on a case. The names it has received suggest that it occurs only in the mouth, this is not correct, it may involve any mucous surface, of the Respiratory, Urinary and Digestive tracts (vagina, rectum and anal canal).

Zegarelli and Kutscher's (1957) cases occurred in one family. The mother had a peculiar silvery lesion of the vagina which did not respond to treatment, and affecting the oral mucosa was a dense, deeply folded, scrotal lesion with a greyish-white hyperkeratotic surface. The tongue was a deeper white and heavily coated. Both mucosal surfaces revealed essentially similar histopathologic findings. Irritation deep within the folds produced some pain from time to time. The children had similar extensive, but less obvious oral lesions. No biopsy had been taken at that stage.

Everett and Noyes (1953) classify these patients as belonging in the group of congenital ectodermal dysplasias.

VINCENT'S INFECTION

Trench Mouth, Fusospirochaetosis, Ulceromembranous stomatitis, Necrotising ulcerative Gingivitis or Stomatitis, "Plaut's Angina" or Plaut-Vincent's Infection.

Since the disease varies in both severity and extent, it is necessary to provide a classification. The following classification is commonly in use: Acute necrotising ulcerative stomatitis; acute necrotising ulcerative gingivitis; subacute necrotising ulcerative gingivitis; Vincent's Angina.

The disease has a complete and not yet fully understood aetiology. A viral theory has been suggested. Stones (1954). The evidence is, however, not of a positive nature and the theory is not very attractive.

We know that many local and general factors can be involved, and any oral abnormality may contribute, and it appears that the production of anaerobic areas favours the disease (operculum, periodontal pockets, chronic gingivitis, infected tonsillar crypts, poor oral hygiene, faulty restorative work).

Hartman (1945) reports that chemical trauma may result from the excess use of tobacco, alcohol or spicy condiments.

The incubation areas eventually become cyanotic with the resulting tissue oedema creating a larger anaerobic area. Schluger (1943).

In edentulous mouths, providing the mucous membrane is intact, and there are no anaerobic areas, Vincent's Infection does not develop. However, in debilitated persons, ulcers may develop from a damaged mucosa, in which the Vincent's organism predominates. Jacobs (1943).

Schluger (1943) states that the Fusiform bacillus and Treponema vincenti, with or without their toxins, are the responsible organisms. The normal mouth has these organisms almost universally present, and Rosebury (1942) considers it becomes increasingly more evident

that tissue vitality, and the factors which raise or lower it, are of the utmost importance aetiologically.

Vincent's Infection resembles those diseases in which potential pathogens become pathogenic, for example, peritonitis due to intestinal perforation, (which is not communicable, and neither is Vincent's Infection.) Rosebury (1942).

Epidemic Vincent's is no exception, for in the epidemic form the patients lived with similar conditions of housing, hygiene and nutrition. Early evidence of communicability was given by observers who demonstrated the Vincent's organisms on articles contacting the mouths of individuals with the disease, for example, toothbrushes, pipes, cigarette holders, and mess tins.

Schluger (1949) considers that the factors of proximity, intimacy and possible contamination were less important than general health, tissue resistance, fatigue and oral hygiene. In Schluger's group of cases, those with Vincent's Infection were not isolated, but continued their daily routine. There were no new cases. The evidence of man to man transmission is circumstantial at best, and on the whole, unconvincing. Rosebury (1942).

General factors influencing tissue resistance are of prime importance in the development of Vincent's Infection, and include Avitaminosis B (especially Nicotinic Acid) and Avitaminosis C. Kirkpatrick (1939) considers diet to be a predisposing factor in the aetiology of Vincent's Infection.

A loss of gingival keratin occurs with low oestrogen levels, and is also significant.

Vincent's Infection may accompany tuberculosis, blood dyscrasias, general debility and fatigue.

Jacobs (1943) was unable to produce oral symptoms in normal animals following the injection of Vincent's organisms, but in animals with an avitaminosis (and trauma) he was.

All attempts to produce Vincent's Infection in human subjects by direct transmission have been unsuccessful. Report (1945).

From this and other evidence we must conclude that Vincent's Infection is not a communicable disease.

Miller and Greenhut (1944) report a seasonal (Fall season) and age (15 to 30 years) relationship in New York civilians.

Smears taken from cases of Vincent's Infection show the presence of the Fusiform bacillus and Treponema Vincenti in 86% of cases. This should not eliminate the streptococci and the oral moulds, which may act as symbiotic agents by contribution to the anaerobic state.

There are three types of oral spirochaetes, namely, Treponema microdentium, Treponema macrodentium and Treponema Vincenti, (which is the most motile.) Two types of the fusiform bacillus are to be found: Fusobacterium dentum and Fusobacterium polymorphuem. These organisms vary from a large, straight, tapered rod to a small, hooked, almost banana-shaped organism. Cobe (1948).

Seymour (1957) states that Vincent's organisms have been found in the pharynx, bronchus, lungs, eyes, nose and penis. Also in cases of scarlet fever, otitis and relapsing diarrhoea (rectum and sigmoid).

Mathis (1956) emphatically states that Vincent's Infection is not due to fusospirochaetal symbiosis (although it may be involved), but rather to the vasoconstriction of the terminal blood vessels in the oral tissues following irritation of their nerves.

Inhibition of the circulation results in necrosis.

Vincent's Infection may affect the gingiva only (acute necrotising ulcerative gingivitis, Vincent's gingivitis), the entire oral mucosa (acute necrotising ulcerative stomatitis, Vincent's stomatitis), or the throat (Vincent's angina). Thoma (1954). There are three forms of the condition, acute, subacute and chronic, although Thoma (1954) recognises two only.

Acute Vincent's gingivitis has a sudden onset, the marginal gingiva is highly inflamed and the interdental papillae are engorged. Small greyish ulcers form on the papillae, flatten them out and progress in each direction about the teeth, until a continuous slough is formed. Separation of the gingival margin

results in the formation of crevicular ulcers, deep destruction of the periodontal tissues and loosening of the teeth. A greyish-brown slough covers the ulcers, but is readily detached, leaving a raw, bleeding surface.

Local symptoms accompanying the gingivitis vary with its severity. There is considerable pain usually, and the patient has difficulty in masticating; tooth brushing is impossible. The teeth may feel numb, elongated or wedged apart. The saliva is thick, ropy and very plentiful, and drooling may occur (especially in the angina, when dysphagia is present). The tongue is frequently swollen and coated, the patient complains of a metallic taste, and there is a very marked and characteristic fetor oris. Local complications include sialoadenitis, tonsillar ulceration, angina, bone necrosis and noma.

General symptoms are not always present with the gingivitis, but are usually found in the stomatitis and the angina. They include general malaise, anorexia, insomnia and constipation, increased pulse and respiration rate, pyrexia and leucocytosis. Cervical lymphadenopathy is seen in severe cases.

Sub-acute Vincent's gingivitis is more common, but frequently missed. It may follow the acute form or be due to a very mild form of the infection. The gingiva, which have a rose or bluish discolouration, are sensitive to touch, but the margins, although hypertrophied generally, show no evidence of ulceration and sloughing. Thoma (1954).

Acute exacerbations occur occasionally about unerupted third molars, but more frequently involving the interdental papillae (which may be split). There are no general symptoms.

Chronic Vincent's Infection is readily recognised by the pale, cyanotic appearance of the mucosa and the lack of inflammatory reaction. There is tissue loss interproximally, and the labial and lingual gingiva are merely curtains. Sometimes a mild osteomyelitis affects the uncovered bone.

Involvement of the tonsil, pharynx or larynx by Vincent's Angina, with extensions to the trachea and lungs, may result in such

complications as lung abscess, pulmonary gangrene or bronchiectasis. Ballenger (1954), Thoma (1954).

In acute Vincent's stomatitis, not only are the gingiva involved as described, but also the remainder of the oral mucosa. The tongue is swollen and shows evidence of ulceration and desquamation. According to Schaffer (1951-52), primary Vincent's infection of the tongue is rare. The lips may be swollen and cracked and the sloughs are more massive and greenish-yellow in colour. The submandibular and carotid lymph nodes are tender and enlarged. Fish (1952).

Histopathological examination shows extensive destruction of the epithelium and surface of the corium without correspondingly great reaction in the underlying tissue. The epithelial attachment may be involved by the necrosis, and if the condition is of long standing, the inflammatory changes extend down and destroy the transeptal fibres. The organism may be demonstrated (frequently in a pallisade fashion) just below the necrotic tissue by special staining techniques. Thoma (1954).

Provided that it is taken by an experienced bacteriologist, a smear readily demonstrates the organisms. Fish (1952). Cobe (1948) reports positive smears in 86% of cases, but Hirshfield (1942) considers them to be of questionable value.

The management of Vincent's infection is one which has raised great controversy in the past, and there is still a great deal of argument as to what constitutes the best treatment.

Fish favours parenteral administration of penicillin, together with careful local application of 20% chromic acid solution in water, followed by 20 volume hydrogen peroxide. A semi-fluid diet is very important at this stage, and no tooth-brushing should be attempted. When the acute symptoms have subsided, complete scaling and prophylaxis should be carried out; a gingivectomy may be required. Strict home care is essential; the patient should not smoke and must have plentiful rest and sleep.

Some authors advise early scaling but others consider this may jeopardise the patient's life by causing a fuso-spirochaetal infection of the blood. Schluger (1943).

Lyons (1948) considers that although chromic acid shortens the disease period, it damages the gingival papillae.

Whatever treatment is instigated, early debridement is essential and medication takes a secondary place. It is important that the predisposing factors, whether within the mouth or those influencing the general health of the patient, be corrected. Report (1945).

ORAL SIGNS AND SYMPTOMS

Angular cheilosis, Black hairy tongue, Cacogeusia, Ageusia, Hypergeusia, Parageusia, Furred tongue, Glossodynia and Glossopyrosis, Halitosis, Pigmentation, Xerostomia.

ANGULAR CHEILOSIS, Perleche.

Since people with the disorder frequently moisten the effected areas with their tongue it is understandable that the lesion has been named perleche (from the French pourlecher = to lick).

Although it may be initially symptomless, the lesion usually has a mild dry feeling, with slight pyrosis. Deep infected fissures are frequently very painful. Either or both labial commisures may be involved by a series of wrinkles of varying depths. The wrinkles may progress to fissures with red bases and later they become macerated, the wrinkling increases in depth and actual epithelial detachment occurs with the production of single or multiple linear ulceration(s). There is little haemorrhagic tendency.

Extension outward from the angles of the mouth to the skin may occur, usually in the form of a localised erythematous scaling dermatitis with fissuring. The condition frequently undergoes spontaneous remissions and exacerbations.

A great number of conditions may produce an angular cheilosis, but quite frequently a mechanical factor is present, namely, as a result of dentures with a decreased vertical dimension. Unsupported folds form at the angles of the mouth and provide a receptacle and incubation zone for saliva and organisms. Finnerud (1944).

The lesions have been attributed to infection by monilia albicans blastomyces, Histoplasmosis, Levy (1945), cryptococcus, staphylococcus and streptococcus. These organisms may be the cause of the lesion or a secondary invader. Thoma (1954). An interesting case in which a Diptheritic infection became superimposed on the angular cheilosis of ariboflavinosis is described by Riddell (1950).

The angular cheilosis of ariboflavinosis is well known, that accompanying Kwashiorkor, Sprue and Avitaminosis A are less known but are considered in detail in the section dealing with the nutritional deficiencies. The lesions associated with congenital syphilis are of a similar type. Finnerud (1944).

Angular cheilosis of allergic origin is proposed by Vickers (1952), although Burket (1957) considers that it must be extremely uncommon.

Angular cheilosis is commonly present with pachyonychia congenita. Gorlin (1958).

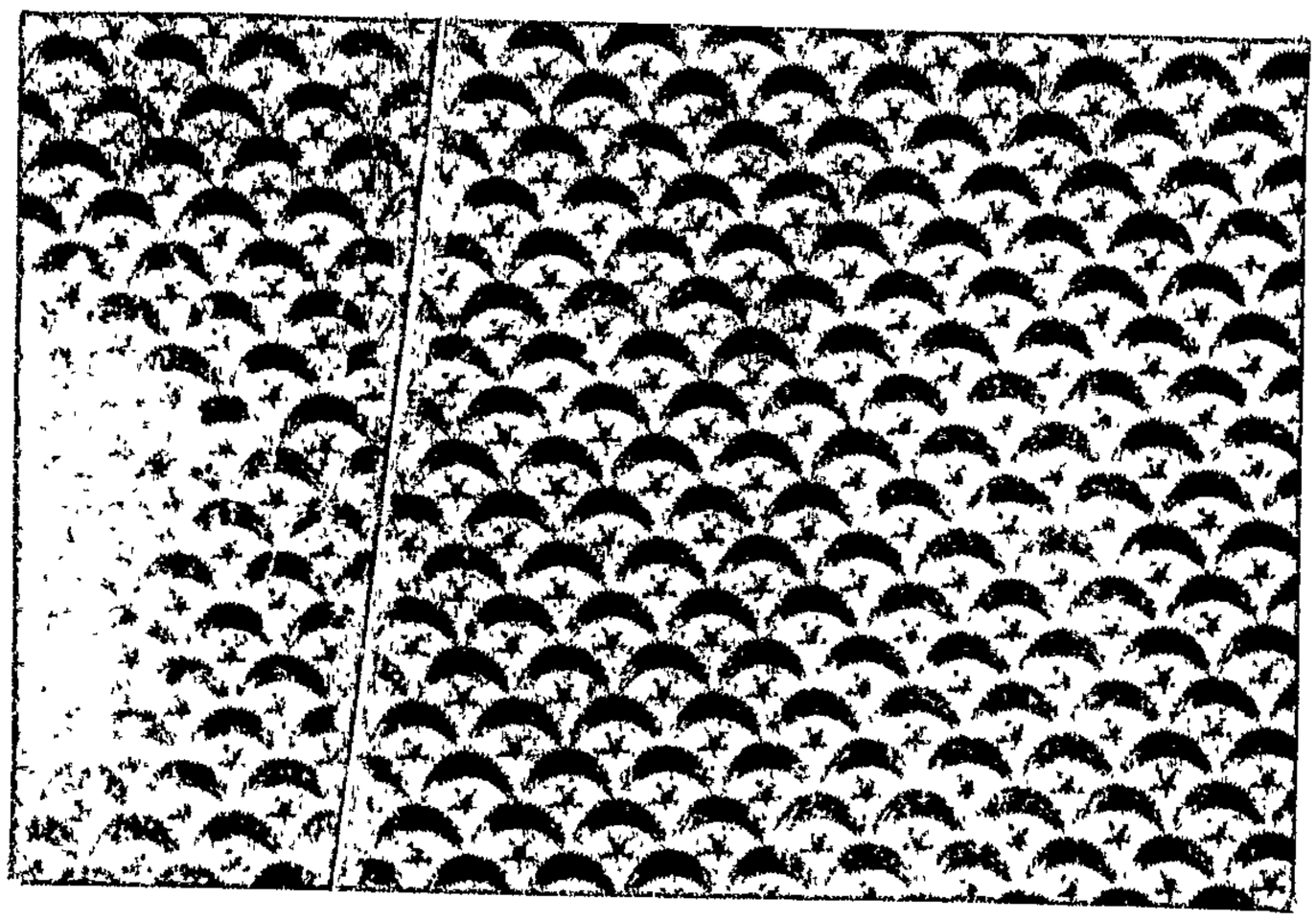
BLACK HAIRY TONGUE

Synonyms: Black tongue. Lingua villosa nigra. Lingua nigra. Shira (1957).

Is an uncommon disease of doubtful aetiology, frequently associated with mal-hygiene and antibiotic therapy. Wolfson (1949) states that discolouration of the tongue accounts for 30% of all oral reactions to penicillin. An immediate thought is that penicillin depresses the normal oral flora with a proliferation of the fungal or chromogenic organisms. Schaffer (1951) attributes the condition to chromatogenic bacteria. Cross (1949) was unable to detect chromogenic bacteria or fungi although Monilia Albicans was present in several cases.

Other aetiologic considerations include a nicotinamide deficiency; irritation from the lozenge base or even individual sensitivity to penicillin or its impurities. Cross (1949) believes that the ingested penicillin inhibits those intestinal organisms responsible for the synthesis of nicotinamide; he eliminated irritation by the lozenge base from his aetiologic test with a control blank lacking the penicillin. He was unable to produce any toxic effects in patients who had previously developed glossitis and other symptoms with the lozenge, or even individual sensitivity to penicillin or its impurities. He considers that discolouration follows two to four days after commencing treatment. Wolfson (1949) says two to nine days, and wears off in five to fourteen days. Clinically the tongue shows pigmentation of the filiform papillae, most pronounced in the central furrow and towards the dorsum. The colour which is absent at the sides and tip may be yellowish-brown, brownish-green, greenish-black or black. It is unfortunate that he was unable to obtain sufficient pigment to make a spectrographic analysis.

Bartels et al, (1955) investigated the nature of the "hairs" by clipping them from the tongue and culturing them. He concluded that they were actually hypertrophied prolongations of the filiform papillae and consist of non-nucleated epethelial cells, cornified and adherent to one another. The serrated edges form a protected



Black Hairy Tongue

After Colby (1956)

area for a microbial invasion which he believes is of a saprophytic nature rather than causal.

Phillips (1950) claims to have seen two cases following the use of Aureomycin troches.

Shira (1957) considers that the cause is either a superficial infection by the pigmentary producing bacteria, molds or fungi; or by the impaction of food between hypertrophied and elongated papillae.

CACOGEUSIA

is defined as the presence, in the mouth, of a foul, disagreeable taste.

Chemical, Neurologic, Otolaryngologic. Hart (1938). Pulmonary and oral pathology may give rise, French (1945), to this bad taste which has been described by Hart's patients as "bad", "sour", "bitter", "salty", "brackish","metallic", "funny", "nasty", "flat", "disagreeable", "gassy", "mattery" and "like decayed food".

The galvanic activity produced in the mouth by dissimilar metals gives rise to a "metallic" taste, similar to that found in patients suffering from intoxications by mercury, gold and thallium. Poisoning by lead and trichloreythylene give a "sweetish" taste; by vanadium a "salty" taste; by tetrachloroethane a "peculiar" taste; by trinitrotoluene an "unpleasant" taste, and by methylbromide a "bitter taste of burnt rubber". Walters et al (1952).

Cacogeusia may accompany Xerostomia and a coated tongue in severe fever. French (1945).

In a series of cases he treated, Hart (1938) noted that Cacogeusia and sexual maladjustment frequently occurred together. The rejection of the sexual role was a common factor in all his cases, and he concluded that many women, particularly when suffering from dental, nasal or pelvic disorders, may complain of Cacogeusia when their sex life is frustrated and void of any pleasure.

Parageusia is defined as a perversion of the taste sensations and may accompany pregnancy, hysteria, insanity, epileptic aura and streptococcal glossitis. French (1945). Hallucinations of taste may occur in association with those of smell as a result of an irritative lesion involving the neighbourhood of the Uncinate gyrus. Lesions of this neighbourhood may also cause Parageusia. In this condition many substances will excite the same unpleasant flavour. Brain (1955).

Parageusia may be associated with the climacteric. Massler (1951).

AGEUSIA

is defined as the loss or impairment of the sense of taste. It may follow a cortical disorder (Central Ageusia), a disorder of the nerve endings (peripheral ageusia), or as the result of a lesion in the nerve between its origin and distribution (conduction ageusia). Hoerr and Osol. (1952).

From a diagnostic viewpoint, Ageusia is only important in rare cases. When the impairment is unilateral, it is almost certainly due to a lesion involving either some portion of the third branch of the trigeminal nerve, the chorda tympani or the glossopharyngeal nerve. If taste is impaired in the posterior third of the tongue upon one side, the lesion probably affects the glossopharyngeal nerve, and there may be paresis of the same side of the palate or partial paresis of the pharynx at the same time.

When the lingual branch of the trigeminal nerve is involved, the impairment of sensation is in the anterior two thirds of the tongue on the same side, and furring of the tongue may be localised to the same part. The lesion may be a tumour or an injury affecting the lingual nerve in the mouth, or a more general affection of the trigeminal nerve. When sensation of both sides of the tongue is affected, it is possible that the lesions described may be bilateral; but it is more likely that the defect is then not of primary nervous origin unless it is due to bulbar paralysis. French (1945).

Unilateral ageusia may result from lesions of the tractus solitarus and its nucleus. Lesions near the midline of the pons may cause bilateral ageusia from the destruction of both gustatory fillets. Brain (1955).

Affections of either the oral or the nasal cavity may give rise to impairment of taste. Allergic coryza, rhinitis, nasal polypus, adenoids and other varieties of nasal obstruction are aetiologically important.

The destruction of the nerve endings in the tongue by corrosives

taken accidentally or with suicidal intent, stomatitis resulting from Bromism, Iodism and the Mercurials, and superficial streptococcal glossitis may cause Ageusia. French (1945).

Ageusia may accompany intoxication by Chromic Acid. Walters et al (1952) Abstract (1). It may be associated with the Ramsay-Hunt syndrome. Findlay (1949).

When testing a person's sense of taste, the tongue should be well protruded, and the substances placed on the tongue, (which is dried between each application) in turn. The anterior two-thirds and the posterior third should be tested separately. Since there are only four sensations of taste,(sweet, salt, bitter and acid,) they can be readily tested with solutions of sugar, common salt, quinine and acetic acid. Brain (1955).

Hypergeusia is an abnormal acuteness of the sense of taste and will not be considered further here.

THE FUR OF THE TONGUE.

The abnormally coated tongue has always been associated with Gastro-Intestinal dysfunction. Burket (1957), (quoting Crohn and Drosd) however, refutes this statement but does admit that an "upset Stomach" may be followed in six to twelve hours by a furred tongue. Loudon (1956) is of the opinion that furring is not connected with disorder or disease of the Gastro-Intestinal Tract as such. However, vomiting associated with Pyloric or Intestinal Obstruction is usually accompanied by a furred tongue, the vomiting of pregnancy and sea-sickness is not.

There is no evidence that any particular condition of the large or small bowel will result in an abnormal coating of the tongue. Deeny (1958) states that furring is not seen in Gastro-intestinal upsets unless they are associated with fever, and considers that the increased body temperature favours the increased growth rate of the Yeasts. Thoma (1954) reminds us that in respiratory diseases gastro-intestinal disturbances and any other disease associated with fever, the tongue is heavily coated or furred.

The experimental constipation of healthy individuals for from four to six days failed to produce any change in the tongue coating. Burket (1957).

The mechanism of furring is one of both mechanics and physiology. Normally a coating is continuously forming on the tongue's rough surface. However, speech, mastication and other functions involving tongue movements cause this accumulated material to be removed. A detergent diet and a normal flow of saliva also help this clearance. Thus any condition which results in a partial or complete Xerostomia (whether of a permanent or temporary nature) may contribute to a coating. (I consider myself that there is a greater concentration of salivary salts in this decreased volume of saliva, and they are more readily precipitated.) The decreased volume also interferes with the normal flushing action, and in this lies a possible second mechanism. Butlin (1885) describes the fur as being not composed chiefly of epithelial and food debris, but of

Schistomycetes mingled with the spores and threads of baccillus subtilis and other organisms. Epithelium is always present on fur scraped from the tongue and that is possibly why it is present; the harder and deeper the scraping the greater the quantity of epithelium.

Deeny (1958) considers that yeasts (Schistomycetes) constitute the bulk of the deposit, but food debris, mucous bubbles, epithelium and saliva contribute to the remainder.

The oral cavity provides an ideal incubator for the growth and development of the Schistomycetes, which enter the mouth with food, liquids and inspired air. These yeasts catch on the tongue and develop in a moist, warm and dark environment. The longer the filiform papillae the more fur present, the shorter the papillae the less fur (as is seen in infants).

The friction of the anterior two thirds of the tongue against the palate, teeth and gums aids in its cleansing, but the dorsum which is not thus abraded frequently becomes the site of a heavy coating.

An accumulation in other places, or of a unilateral nature should make one suspicious of local pathology. Butlin (1885). (For example, irritation from a carious tooth or denture.)

Local factors influencing airflow and thus contributing to the furring are mouth breathing, nasal infection and heavy smoking.

Schaffer (1951) considers the fur is only hypertrophied filiform papillae. He attaches little significance to a moist coated tongue and attributes it to the "day after". The crusted tongue, however, is dry and coated and may be white to brown in colour. It is seen in infectious diseases, chronic debilitating disease, rheumatic fever, gastro-enteritis and following surgery.

Thoma (1954) describes a brown crusted tongue seen in intestinal obstruction, peritonitis, terminal cardiac failure and malignancy.

The colour of the fur is greatly altered by foodstuffs, condiments and drugs the patient may ingest. The odour may be quite intense (fetor ex lingua) but can usually be eliminated by removing the coating.

The elimination of the fur is readily accomplished, and although it forms again (while conditions favour its formation) its removal may give the patient comfort. Blackburn (1957) recommends sodium bicarbonate brushed or swabbed over the tongue, but Burket (1957) favours sodium perborate, and Butlin (1885) prefers a weak carbolic solution (less than 2%), a weak Condy's solution, or a solution of 3% Boracic acid.

GLOSSODYNIA AND GLOSSOPYROSIS.

Glossodynia is defined as pain in the tongue. It is sometimes incorrectly described as a burning sensation of the tongue. (glossopyrosis).

Frequently the symptom is associated with demonstrable lingual pathology and known systemic disease. However, in some cases the condition affects an apparently "normal" tongue. It is important that local irritative factors are eliminated, malposed mandibular teeth, prosthetic appliances and poorly executed restorations. A pernicious habit of rubbing the tongue against the lower teeth during times of stress may be demonstrated. Burket (1957). Other local causes include electrogalvanism, smoking, irritating food and drink and mouth breathing.

One of the symptoms of the so-called "Costen's syndrome" may be glossodynia. Schaffer (1951-52).

Fixed drug eruptions are commonly found on the tongue, and an allergic basis should be considered in the differential diagnosis. Burket (1957).

Waldman and Pelner (1948) have considered the symptom in some detail. Ariboflavinosis, aniacinosis and avitaminosis B complex may give rise to glossodynia, or more accurately, glossopyrosis. An excessive vitamin loss may be associated with ulcerative colitis and other diarrhoreal diseases. Glossopyrosis accompanies Xerostomia in the diabetic state, and is the result of mucosal desiccation, and in thyrotoxicosis as a result of the increased utilisation of the vitamins.

Poor vitamin absorption occurs in Sprue with a resultant avitaminosis and glossodynia. It may also be associated with liver damage. Haematological causes are quite important and glossopyrosis or glossodynia may accompany Pernicious Anaemia and the Plummer-Vinson syndrome.

Glossodynia and glossopyrosis of apparent idiopathic origin is sometimes associated with the menopause. Massler (1951).

Neurogenic and psychoneurotic causes can be found in certain cases. Glossopyrosis may be a symptom of thrombosis of a small intracranial vessel. Waldman and Pelner (1948). "Mental strain" and cancerophobia may be significant, as is also disturbed gastric acidity, (hyperacidity, hypoacidity and anacidity). Schaffer (1951-52).

The glossodynia and glossopyrosis of the geriatric patient can not always be explained. Burket (1957).

With the elimination of probable local causes, the treatment where possible is aimed at the correction of the systemic disorder. In some cases this is not possible, and symptomatic and supportive therapy may be necessary.

Waldman and Pelner (1948) have had success in alleviating the symptom by providing the patient with a sialogogue and increasing his fluid intake. Some patients will require psychiatric treatment. Injection of the lingual nerve or cautery of the surface of the tongue is usually only a last resort. Schaffer (1951-52).

HALITOSIS.

HALITOSIS

"Man's Breath becomes infected by the bad qualities of food, by the bad state of the teeth and still more by his age". (Natural History-Pliny the Younger 23-79 A.D.)

HALITOSIS (Latin halitos breath
osis abnormal or diseased)

Synonyms: Foul breath, bad breath, foetor ex ore (local or systemic).

I prefer the use of the term halitosis to foetor ex ore since the latter conveys the misleading impression that the mouth is the origin of these odours, which in fact it may not always be.

The breath may be a very useful diagnostic aid to the clinician in the detection of systemic disorders Massler etal (1951).

Socially the presence of halitosis is a handicap, it severely hampers social intercourse and if the patient is aware of this real or fancied Halitosis, the fear of offending reduces his confidence of approaching others, it can interfere with his effectiveness in any activity and may precipitate a true neurosis Hine (1957). Frequently the patient is unaware of the existing condition and continues to offend. This attitude of "Even your best friend won't tell you" is to be deplored and it is the clinician's duty to inform the patient of the condition and endeavour to eradicate it.

The Symptomatic Treatment of Halitosis by the use of strong antiseptics, dentifrices and deodouring tablets in an endeavour to mask the odour is undesirable. Odour signifies pathology - eliminate the pathology and the odour is eliminated. The patient may not be aware of the odour or he becomes Prinz (1930) accustomed to it for the olfactory organs are rapidly fatigued by continued exposure to odourous material. This phenomenon is known as odour adaption Hine (1957).

Sulser etal (1939). The average breath odours are just on the border line between offensive and non-offensive. Odour intensity increases with age and halitosis is quite prevalent in older people.

This may be the result of retrogression of the body's processes or gingival recession producing greater areas for food impaction and putrefaction (or a combination of both), salivary stagnation and periodontal disease. Massler etal (1951). Young children and infants, however, have a sweet and pleasing breath but during adolescence and young adulthood it becomes heavier and more pungent but not unpleasant. In middle age the quality is less pleasant even in those with fastidious habits. In old age the breath is heavy, pungent and often sour. Odours vary greatly in quality (which is determined by its origin) and intensity (or concentration) which determines its social acceptance. Massler (1951).

Quality can be described e.g. neutral, foul, sour, Freeman etal (1936) but intensity can be measured by use of the osmoscope and the cryoscope Brening etal (1939). Clinically it may not be possible to use the osmoscope Massler etal (1951) and the breath may be sampled in much the same way as other noxious fumes (by wafting a hand or card towards your nose through the path of the fumes). The intensity may be measured 0; 1+; 2+; 3+; 4+ to indicate absent, mild, moderate, pungent or very objectionable. It is important to test the breath at the same time of the day whilst noting relationship to meals, menstruation, and sleeping habits.

Massler etal (1951) states that not every unpleasant breath is indicative of pathosis, and the breath varies with the time of day and state of hunger. On awakening a pungent and disagreeable morning breath is present which has been attributed to putrefaction of food debris and saliva in a mouth which has not been constantly cleansed by the detergent action of the tongue, cheeks, speech, passage of food and mastication. Over-indulgence, chronic ailments, dehydration state and mouth breathing increase the halitosis.

Hunger Breath often occurs in adults prior to meals and increases with the increased time between meals. It may be due to oral stagnation and putrefaction or to the volatile products of the intermediary metabolism of protein and fat. It is interesting to note that any food taken rapidly dispels this odour.

Halitosis may arise from the Oral Regions, either from the dental structures, their substitutes or the soft tissues. Other

causes are Nasopharyngeal disease, Bronchopulmonary disease, digestive tract, metabolic disturbances, infections, febrile and genitourinary disease. Absorbed drugs or poisons. Prinz (1930). Food Condiments and Stimulants. Dummett (1944).

Odours arising from within the mouth may be due to:

Food retention and putrefaction account for more than90% of all cases of halitosis. Interdental and cervical areas, prosthetic appliances and tongue and gum flaps associated with erupting teeth provide ideal areas for retention and incubation. In a deeply fissured tongue food retention and putrefaction will occur. Massler etal (1951).

Salivary putrefaction produces a most disagreeable odour and may be especially noted on awakening. Hartzell (1931) noted that the putrefactive accumulations of the tongue produced similar mouth odours. Morris etal (1949) state that mechanical cleansing of the tongue will reduce the odour level in many instances.

The tongue coating frequently has the same odour as the breath. Massler etal (1951). It is thought that non-detergent diets have a lot to do with the formation of the coating but smears show desquamated epithelial cells, gas-forming micrococci and coliform bacilli. Deeny (1958) discusses the composition of the fur and the mechanism of its formation.

Washing the tongue with cotton rolls or gauze napkins wet in a suitable disinfectant or hydrogen peroxide effectively copeswith the deposit. Blackburn (1957) recommends that the tongue be cleaned with sodium bicarbonate.

If there is necrotic tissue present Periodontal disease will contribute to mal odour, as will salivary putrefaction, which occurs more readily in these individuals.

Vincent's infection sometimes produces a "Metallic", sometimes a "rotten hay" odour, but Noma produces a putrid stench.

Chronic gingivitis may be accompanied by a foul odour which is in part due to the presence of decaying blood.

Blood, (Following Oral Surgery, exodontia and Gingivitis). Clotting in the mouth is rapidly covered by proteolytic organisms with the production of a characteristically heavy, pungent and

repulsive odour. In the case of patients with chronic intra-oral haemorrhage (haemophilia, purpura, leukaemia and scurvy) the odour is intense. Massler etal (1951) Prinz (1930). A cavity may serve as a retention area for putrefying food debris. The intensity of the odour will depend on the number and size of these cavities. A necrotic pulp will produce a foul odour. Massler etal (1951).

Mouth Breathing may be accompanied by halitosis, but whether this results from precipitation of salivary material and debris on the tongue, or from chronic marginal gingivitis (and its subsequent haemorrhage) involving the gingival tissues in the anterior region, or both, has not been determined. An increase in the alpha streptococcus is thought by some to be aetiologically significant. Massler etal (1951).

Dehydration states with resulting partial Xerostomia may produce an offensive odour.

Denture wearers are not immune since the porosity of vulcanite provides areas for food retention, and mucoids and saliva adhere and stagnate to the denture base material, and give the typical odour ("denture breath", "false teeth smell").

There is some dispute as to whether smokers' breath is of local or pulmonary origin. Possibly it is both. The odour is pungent, dry, disagreeable and goes completely unnoticed by the heavy smoker. Massler etal (1951). Morris etal (1949) showed that cigars produce the higher odour level and cigarettes a lower odour level.

Tissue Ulceration of any type that may occur in the mouth, will from time to time be responsible for mal odour. Syphilitic and malignant ulcers provide an excellent example.

In the Nasopharyngeal region, chronic and atrophic rhinitis will cause ozena, a condition considered fairly rare by some, although it is frequently found in many conditions including cretinism.

Chronic Sinusitis may be accompanied by a post-nasal drip, and produces a foul odour, Massler etal (1951) as does Tonsillar infection (with an odour of Limburger cheese) Hine (1957), and the presence of plugs and concretions within the tonsillar crypts. Adenoids, chronic laryngitis, pharyngitis, deviated septum, diphtheria, rhinoscleroma, foreign bodies, swabs left after surgery and

otitis media can also be convicted as aetiologically responsible.

Bronchopulmonary disease may be responsible for some particularly foul odours, namely, chronic foetid bronchitis, putrid bronchitis, gangrene of the lung, pulmonary abscess, pulmonary tuberculoses, bronchiectasis, and empyema.

Disorders of the digestive tract are a very popular source of odours, but it must be remembered that air cannot pass up the collapsed oesophagus unless the patient belches.

Disease of the Oesophagus, viz., strictures, cul-de-sac, and its dilatation, neoplasms, ulceration and gastric ulceration will, however, produce foetor. Prinz (1930).

Stercoraceous vomiting gives a typical feacal odour, and Constipation may very occasionally do the same.

Aromatic substances are excreted from the blood via the lungs, in some metabolic disturbances, and thus aromatic metabolites originating from ingested foods and digestive or excretory products of callular metabolism find their way ultimately into the breath.

Kidney dysfunction, chronic kidney disease and cystitis. (Ureamic breath). Ureamia (Ammoniacal odour). Liver (Mousy breath). Cirrhosis of Liver (Cadavorous breath). Diabetes Mellitis (Acetone odour). Chlorosis (Skatol odour). Intestinal haemorrhage (Blood odour).

A temporary halitosis may result from prolonged speaking and psychic excitement.

Yellow Fever patients produce a peculiar putrid odour.

Fats may be responsible for halitosis; especially is this to be noticed in gastric ulcer patients on a milk diet. The theory is that uncompletely broken down fat produces volatile fatty acid radicals which cause the odour. By restriction of the fat intake it is sometimes possible to eliminate the foetor. Massler etal (1951)

Absorbed Drugs and Poisons may produce foetor.

Organic arsenicals, bismuth preparations, cacotylates, salvarsens, asphenamine (garlic-like odour), hydrocyanic acid (bitter almonds odour), tellerium salts (garlic-like odour), oil of turpentine (sordid violet-like odour), lead, halitus saturius, phenol, lysol, laudanum.

Food, Condiments and Stimulants may do likewise. Cheese (depends on type and ripeness), cooked eggs, onions, garlic, leek, spices, mace, cloves, ginger, tumeric, cinnamon and allspice, yeast (beer odour), alcohol, tobacco, cabbage, celery, orange, bananas, Stallard (1927).

Stallard (1927) attributes menstrual odours to a retro-nasal secretion which contaminates the expired air. However, Hine (1957) suggests that a menotoxin may be responsible. Massler etal (1951) describe it as unpleasant and mousy, and it may resemble the odour of decayed and clotted blood (especially in dysmenorrhea). Pregnancy may be accompanied by a disagreeable odour.

A patient with Epilepsy, dementia praecox and hemicrania may have foul breath. Stallard (1927).

It is interesting to note that volatile substances arising in the Gastro-Intestinal Tract are carried from intestine to liver, liver to bile and to blood, blood to lungs and lungs to breath. Experimental work shows that garlic held in the mouth and chewed but not swallowed is cleared from the breath in a short time, but garlic inserted through a gastrostomy, enterostomy, ileostomy or colostomy appears on the breath a few hours later and remains for some hours. Crohn (1942).

Morris etal (1949) notes that Oral odours can be reduced for a period of about two hours by the use of a dentifrice. This does not appear to be purely a masking effect since the odour of the dentifrice flavour disappears in less than 30 minutes.

A plain water rinse had no effect on mouth and breath odours.

An antiseptic rinse causes a reduction in mouth and breath odours from which no significant recovery takes place within 3 hours. This is not a masking effect as the odour of the rinse disappears in 20 minutes.

Brushing with a flavourless dentifrice will reduce all odours of early morning breath by 66%. Brushing the teeth with a dentifrice is more effective than brushing without. Sulser etal (1940).

Treatment of halitosis must first be directed at location and elimination of the cause. A thorough oral examination should be carried out and all suspected dental abnormalities treated. A

complete physical examination of all the systems may be required to eliminate foetor due to extra oral causes.

PIGMENTATION AND COLOUR CHANGES OF THE ORAL CAVITY.

The pigmentation of the mucosa (and skin) is determined initially by racial peculiarity. However, it may be readily altered by pathologic disturbances or intoxication by chemicals and drugs administered accidentally or therapeutically. (The latter will be dealt with in detail in the section on intoxication, but brief references will be made here.)

Sands (1958) states that the colour of skin is determined by several factors; by Melanin pigment in the basal epidermis; by carotene in the dermis and by the relative amounts of oxyhaemoglobin and reduced haemoglobin in the subpapillary networks. Where the mucous membrane is concerned, however, the stratum corneum is thinner than it is in the skin, and the colour will depend largely on blood pigments.

A purplish blue colour of the mucosa is seen in cyanosis; the lips particularly being affected. Thoma (1954). Cyanosis becomes apparent when the reduced haemoglobin concentration exceeds 5 grammes per cent and also when the methaemoglobin exceeds 1.5 mg. per cent. Cyanosis is associated with a great many conditions but I will not discuss these in any detail.

It is found when approximately 20% of the venous blood is shunted into the arterial tree through collapsed or consolidated lungs or septal defects in the heart. It is seen when mechanical faults such as strangulation or asthma prevent aeration of the pulmonary vasculature, and finally when stagnation of bloodflow, as in cold or peripheral failure permits over reduction of haemoglobin. Patients with high haemoglobin concentration may more easily have 5 grammes per cent (in reduced form) in their peripheral vessels, and the patient with polycythemia is commonly cyanosed. Sands (1958).

Cyanosis may be seen in adrenal haemorrhage, anthrax, aniline, methyl alcohol or arsenic poisoning, in Hodgkins' disease, and in many other conditions. Cecil & Loeb (1956).

Elphinstone (1954) reports a case of a baby born spontaneously but grossly cyanosed after birth, the head, neck, lips and tongue were a dark purple and multiple almost confluent petechiae covered

the skin of the face. Pallor of the mucosa results from inability to see the blood pigments in the dermal vessels because of oedema or constriction of the blood vessels from shock or fear (e.g. patient in dental chair). Sands (1958).

Chlorosis, various forms of anaemia, asthma, hypotension and hypothyroidism may produce a pale mucous membrane. Thoma (1954).

Defective Melanin production may be seen in Addison's Disease or in menopausal women with the same result. Sands (1958).

A yellow colouration may result from bilirubin, urochrome, carotene, cholesterol, Sands (1958), or haemochromatosis. Colby (1956).

The antimalarial drug atabrin may produce a yellowish-brown gingival pigmentation. Walters et al (1952) Abstract (85), however, state that when used clinically, symptoms of intoxication include the appearance of a bluish pigmentary deposit on the hard palate, finger and nail beds. Excoriation was noted at the angle of the mouth after prolonged exposure.

The jaundiced patient usually has a coated tongue and a yellowish green mucosa, particularly at the junction of the hard and soft palate. Burket (1957), Prinz (1932).

Colby (1956) reports a case of Xanthosis cutis in a vegetarian who consumed the juice of 100 pounds of carrots a month. The carotenoid pigmentation was golden yellow and was limited mainly to the soft palate.

Untreated Diabetics may be affected thus, even if their carotene intake is not high.

Sands (1958) notes that an excess of carotene tends to be associated with diseases disturbing lipid metabolism.

Prinz (1932) records that in Xantholasma the tongue may be the site of temporary yellow spots.

Butlin (1885) describes a case of Xantholasma which had yellowish-white, oblong patches, quite soft, but slightly raised on the surface of the tongue.

Haemochromatosis (Bronzed Diabetes) may sometimes show an oral pigmentation, Prinz (1932), accompanying the slate-coloured skin. Sands (1958).

Although there is a chalky white pallor in cases of Pernicious Anaemia, a green yellow tint may or may not be present. Prinz (1932).

Pulmonary and abdominal tuberculosis may be accompanied by a light brownish grey or smoky discolouration of the surface of the tongue and the cheeks. The palate may be a pale grey with a slight bluish tint. Prinz (1932).

Melanosis is defined as abnormal deposition of melanin. This increased pigmentation of the mucosal and cutaneous surfaces or internal organs may not in itself have any deleterious connotations, but it is frequently associated with a variety of significant disorders and may provide an important diagnostic clue. Cecil & Loeb (1956).

Melanin is produced by the dendritic neural crest cells which have migrated to the basal cell layer, eyes and meninges. Melanin is formed from tyrosine through the Dopa reaction with a copper protein catalyst tyrosinase, moderated by sulph-hydryl binding of the copper of the catalyst. Its production is subject to the endocrine control of the melanocyte stimulating hormone (MSH) of the intermediate lobe of the pituitary. Sands (1958).

Melanogenous pigmentation may be due to a racial peculiarity, and in the negro melanin pigment is seen frequently about the gingiva and in isolated streaks and spots about the gum, lips and cheeks.

The Chinese, Japanese and Armenians quite commonly have brownish or chocolate coloured spots on the lips and gums. Prinz (1932).

Derbes (1955) in a recent paper discusses some of the types of intra cranial disease which cause general (and oral) pigmentation. He considers that there must be a common pathway over which they act, and the melanocyte stimulating Hormone probably represents this common factor. It is significant that psychoses, generalised cortical and sub-cortical deteriorations and brain tumours lying near the pituitary have produced pigmentation in the presence of normal pituitary and adrenal glands. These diseases may act through the hypothalmic connection to the posterior pituitary.

An intense brown pigmentation may involve the entire body in

Schilder's disease (enchephalitis periaxialis diffusa). It is a slowly progressive disease, seen chiefly in children and in young people, and is characterised by advancing cerebral blindness and mental deterioration, terminating eventually in complete dementia. Addisonian-like blotches of pigment are seen along the gums and buccal mucosa.

Melanoderma has been reported in Catatonic Schizophrenia. The oral pigmentation may involve the corners of the lips and the buccal mucosa is soiled with irregular black non-confluent spots. In some cases bluish patches may be seen on the sides of the tongue and on the gums.

Blue spots on the lips and vestibule of the mouth have been reported with Congenital Cranial abnormality. Derbes (1955).

Although it is not common (or else it is overlooked) Oral Melanotic pigmentation does occur in Albright's Syndrome (polyostotic fibrous dysplasia). The melanotic spots are seen on the mucocutaneous areas of the lips, on the hard and soft palate and buccal mucosa and may be unilateral or bilateral. Gorlin (1957). Thoma (1954) describes a case (Tannhauser) with dark brown to black spots on the lips, the hard palate and the gingiva.

Cafe au lait patches are seen in neurofibromatosis.

A case of familial pigmentation with dystrophy of the nails is described by Moon-Adams (1955). The labial and buccal mucous membrane were covered by multiple discrete areas of blackish grey pigment 3-10 mm. in diameter, the dorsum of the tongue was also pigmented. In another two cases he described, the mouth was full of pigment.

Small greyish patches as described by Berlin (1955) are seen in urticaria pigmentosa. Dewar (1955) describes a bullous form in which the patches on the oral mucosa were brownish yellow. It is thought that Heparin is aetiologically significant with this disease.

Acanthosis Nigricans is a benign cutaneous disease associated with glandular carcinoma. The oral mucosa may be pigmented, but Curth (1948) provided no details as to its distribution, texture

or intensity.

Sands (1958) notes that melanosis may be seen in thyrotoxicosis, pregnancy, Addison's Disease and Acromegaly. The pigmentation of thyrotoxicosis may resemble that of Addison's Disease. Prinz (1932). I can find no reports of oral pigmentation accompanying the melanosis of pregnancy or acromegaly.

The intoxications frequently produce quite striking colour changes in the oral mucosa. Aniline produces a blue grey colour of the lips and gingiva. Stones (1954). The blue line of Lead poisoning and the bluish black line of Bismuth poisoning are both well known. Localised or generalised Argyria produces a blue grey discolouration.

A faintly purple and erythematous gingiva is seen in cases of Auric stomatitis, while Copper produces a blue green hue on the gingiva and teeth, and Zinc a bluish grey line. Iron causes a blackish discolouration of the dorsum of the tongue, but Tellurium produces (in high concentration) a blackish grey discolouration of the lips, teeth, tongue and the angles of the mouth.

A yellow ring forms on the teeth and at the labio gingival margin in some cases of Cadmium Intoxication.

It is well to be aware and suspect wilful pigmentation of the Oral mucosa by Hysteriacs and mental defectives in order to make their "complaint" look interesting. Prinz (1932).

Superficial staining of the oral mucosa by Tinctorial and other agents is considered briefly in the interests of differential diagnosis.

Black: Inks; Red wine; Mulberries; Cherries; Steel wine; Iron preparations.

Brown: Tobacco; Liquorice; Fresh Nuts; Prunes.

Brown-Red: Chocolate.

Yellow: Saffron; Laudanum; Rhubarb; Nitric Acid (if effect is superficial); Chromic Acid.

Red: Red Quinquinia; Rhatany; Raspberries; Cherries; Acid nitrate of Mercury.

Pink: Phenolphthalein; Cochineal.

Grey-White: Sulphuric Acid; Oxalic Acid; Carbolic Acid.
Grey and gelatiniform: Caustic Potash.
White or Pearl Grey: Silver Nitrate; Corrosive Sublimate.

Butlin (1885), Prinz (1932).

Peutz-Jegher Syndrome.

It is significant that such a simple clinical sign should provide the solution to the diagnostic problem of abdominal pain and intestinal obstruction. This syndrome of oral and cutaneous melanin pigmentation and intestinal polyposis was thought by Jegher to be inherited as a simple Mendelian dominant, Staley & Schwarz (1957).

The majority of cases are recognised during the second or third decades of life, and the patients had, in general, been symptomatic for less than five years. Small bowel intussusception may occur with production of vomiting and pain which will progress to emergency proportions unless the polyp happens to be spontaneously liberated, in which case the symptoms subside.

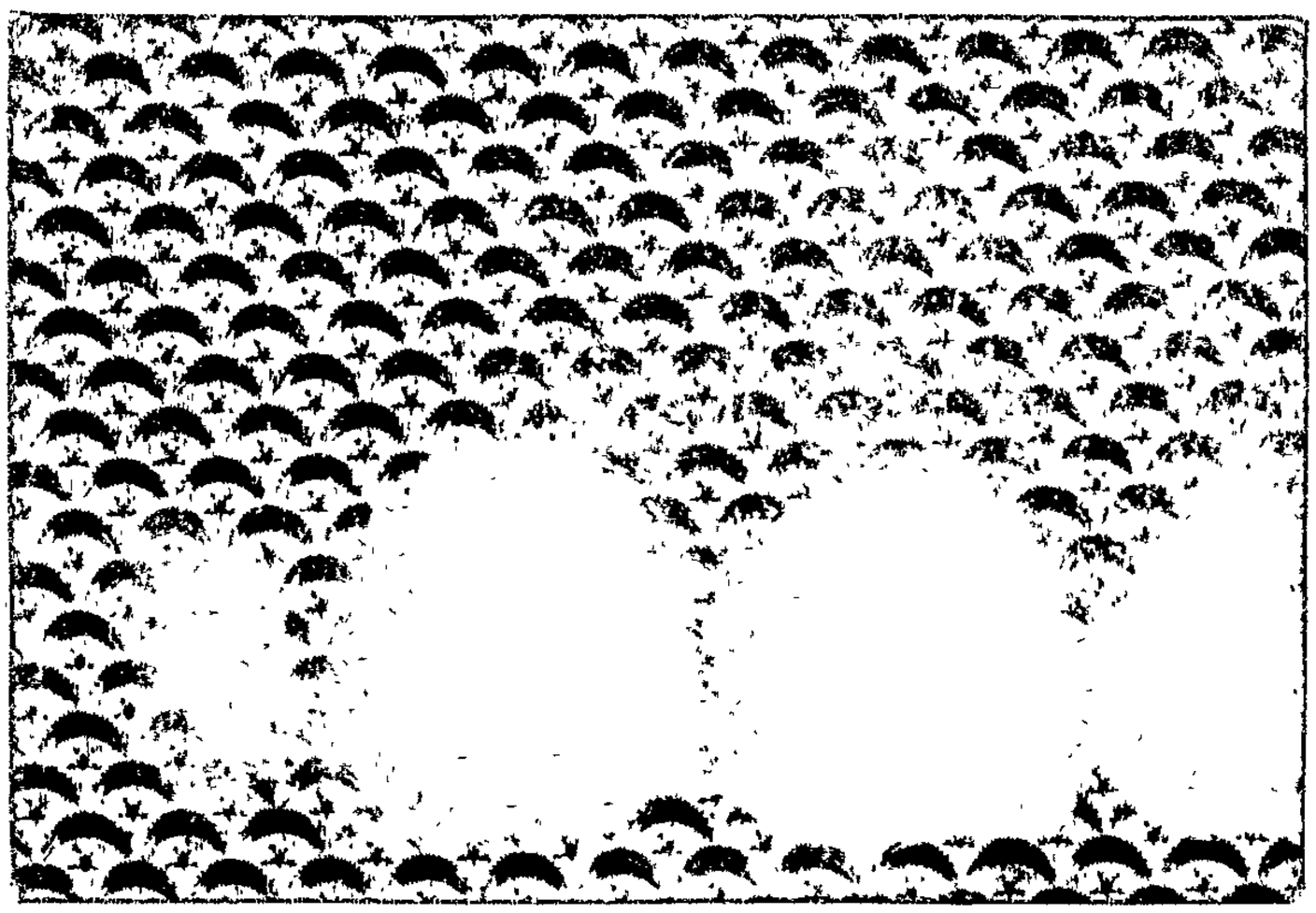
Other symptoms may include diarrhoea, haematemasis, pallor, weakness, anaemia and possibly the appearance of a prolapsed polyp through the rectum.

Macular Melanin spots, with well delineated margins and with little tendency to coalesce, are seen on the mouth, face, fingers and toes. They are up to 5mm. in diameter, although usually somewhat less. Their colour varies, generally those seen on cutaneous surfaces are brown or inky brown, Andrews (1954), but they may be grey or black with a bluish cast.

Oral lesions are seen to involve the buccal mucosa, gingiva, palate, less frequently the tongue, but rarely the floor of the mouth. Staley & Schwarz (1957).

Fung (1957) claims that peri-oral pigmentation is marked and that the lower lips and labial mucosa are well pigmented. Staley & Schwarz (1957) agree that the lips are quite commonly involved, the lower more so than the upper.

Melanin Pigmentation associated with Intestinal Polyposis
(Peutz-Jegher Syndrome) After Colby (1956)



Melanosis Gingivae (Physiologic Pigmentation) in half-caste
Australian Aboriginal woman. Author's patient.

Andrews (1954) claims that reliable witnesses are convinced that the pigmentation increased during a bout of symptoms. He goes on to state that the buccal lesions appear essential to the syndrome to exclude the diagnosis of elphides (freckles).

Rohrs (1957) and Bluefarb et al (1956) have also reported cases.

Kesten (1955) reports that in her case the melanin spots began as small red dots. Schaffer & Sacks (1952) describe the syndrome.

Addison's Disease.

True Addison's Disease is a relatively rare condition which may be caused in the main by either fibrocaseous tuberculosis of the adrenal gland or by an idiopathic process of bilateral adrenal cortical atrophy.

Less common causes encountered are bilateral tumour metastases, leukaemic infiltration, amyloidosis, haemochromatosis and histoplasmosis. Rawson et al (1951).

Usually the disease has an insidious onset although occasional a crisis may be precipitated by surgery or an acute infection. The patient loses weight, is asthenic and easily fatigued; he may become irritable, nervous and depressed. There is an increased degree of mucosal and cutaneous pigmentation and the patient may have gastro-intestinal complaints.

Harrison (1957) . Laboratory investigations are essential in less clear-cut cases, and a simple preliminary test involves the diuretic response to ingestion of a litre of water in the fasting state. It is unlikely that the patient is suffering from the disease if more than 700 ml of urine are excreted in the following four hours. Failure of diuresis is only consistent with Addison's disease; it is not diagnostic. The most definite diagnostic procedure is the demonstration of a lack of adrenal cortical response to the administration of ATCH. Cecil & Loeb (1956).

Although Prinz (1932) states that oral pigmentation is almost the rule in Addison's disease Davies (1955) reports a

case that was unaccompanied by pigmentation.

Burket (1957) states that oral pigmentation may be an early symptom of the disease, but in some severe cases it is absent. Oral pigmentation is, however, diagnostically more significant than cutaneous pigmentation. Any part of the mouth may be involved, the lips, tongue, buccal mucous membrane Harrison (1958) or palate. Burket (1957). The pigmentation may be bluish black or brownish grey and take the form of streaks, blotches or irregular spots. Prinz (1932) describes it as pale brown, deep brown, chocolate, or dusty pearly grey to dirty, sooty black.

The vermillion border of the lips may be involved or intra-orally extending with fan shaped spread from the angle of the lips along the buccal mucous membrane. The tongue may also be discoloured and Butlin (1885) described its involvement in some detail. The areas appeared to be only stains, very dark or black marks, sharply defined but neither raised nor depressed. It is interesting to read that he considers this pigmentation to be of no great clinical importance.

Involvement of the gingival festoons occurs as a continuous dark brown line, never uniformly pigmented but not diffuse.

Oral pigmentation is described by Nordin (1955) and Hopp (1958).

Hypersecretion of the melanocyte stimulating hormone follows decreased secretion of circulating hydrocortisone and results in the increased deposition of melanin.

Prolonged treatment with cortisone may depress this hyper-secretion and allow some lightening of the pigmentation. Harrison (1957).

XEROSTOMIA (Greek: Xēros = Dry; Stoma = Mouth)

The condition is frequently due to local pathology involving the salivary glands, but it may be the result of factors interfering with the nervous mechanisms controlling the secretion of saliva, of general disease, debility, intoxication or medication. The mechanisms of salivary secretion are complex and not well understood. However, Best and Taylor (1950) give a revealing account.

The salivary glands are controlled by both a nervous and a chemical mechanism. The nervous mechanism is primarily important in man and produces a rapid response to stimulus. The Chemical (Hormonal) control produces a slow but sustained response. Under natural conditions secretion is governed by both conditioned and unconditioned reflexes. Training and experience are the basis of the development of the conditioned reflex, and in the case of salivary secretion are not due to the stimulus of the nerves of the mouth but those of special sense. Substances placed in the mouth call forward the unconditioned reflex; the volume and quality of saliva varying with the substance introduced.

The Saliva has at the very least an eight fold function,namely: Digestive, Cleansing, Moistening and Lubricating, preparation of food for Deglutition, Solvent action, Excretory capacity and partial maintenance of Water Balance.

In the almost dry mouth the consistency of the saliva is often readily recognised. It is mucoid, sticky, webb-like, bubbly and frothy, it is adherent to the mucosa, balls up readily and is hard to expectorate. It does not moisten the mouth satisfactorily and the mucosa becomes sticky, dry, red and painful. Allington (1950)

Saphir (1941) describes the replacement of a normal moist, smooth surface by a rough, cracked and fissured surface which bleeds easily, interferes with eating and chewing and leads to early inanition and cachexia. Suher et al (1953) describe wrinkled and boggy mucosa with hyperaemic and hypertrophic gingival tissues. The tongue becomes heavily coated often and may be superficially

crusted. Allington (1950). The remainder of the mucosa may be dry, smooth, pale and semi-transluscent. The caries rate increases and denture problems frequently ensue. The lips are dry, inflamed, scaled and fissured. There may be an angular cheilosis. Patients thus affected find it necessary to moisten their lips with a wet rag whilst speaking, or to sip water during the night to prevent their lips adhering or their tongue sticking to their palate.
Causes of Xerostomia.

The lack of salivary gland(s) either as the result of aplasia (defective development or congenital absence) or following surgical extirpation will produce a partial or complete xerostomia dependant on the involvement. A case of congenital aplasia of both parotid glands is described by Faber (1943). Suher etal (1953) describe a congenital xerostomia resulting from the absence of the sub-maxillary glands. A natural atrophic change will produce the xerostomia of senility and constitute a major cause.

Allington (1950) (quoting Hadden) describes cases of hereditary ectodermal dysplasia which resulted in notable reduction in salivary secretions.

Local disease of the salivary glands and their duct system may be responsible for partial xerostomia. These include Sialoliths; neoplasms of the glands and surrounding structures; fibrous obliteration of the duct(s) following surgery; infiltration by specific diseases, for example, tuberculosis, syphilis and actinomycosis; acute suppurative infection of the glands, X-Ray damage; following Radium treatment; and surgical extirpation of the glands(s).
Dehydration is a very common cause of temporary (partial or complete) xerostomia and may be the result of profuse sweating, diarrhoea, haemorrhage, acute febrile conditions, diabetes insipidus (and possibly to a lesser extent Diabetes Mellitis). Vitamin A deficiency stands convicted for the appearance of xerostomia in patients with thyroid disturbance, genital atrophy, starvation and alcoholism. To a lesser extent an ariboflavinosis or nicotinic acid deficiency may produce it. Saphir (1941).

Although a lot of attention has been drawn to factors influencing

dysfunction of the salivary glands there has been little focus on factors involving the nervous mechanisms. Allington (1950).

Normally emotional reactions produce change in secretory rates and amounts; minor emotional reactions cause a slight increase while intense reactions will produce a temporary rise in rate and then inhibition. Disgust, Pain, embarrassment, erotic emotions, mental work and concentration have a depressing effect, while pleasurable sensations, satiation and distention of the stomach increase the rate.

In early years (three years) the child has a high rate of secretion and drooling occurs. Between five and six years the rate decreases rapidly, and from eleven to twelve years he approaches the normal adult level. There is a lowering of the activity in the geriatric period which tends to parallel decreases in other glandular activities.

Fear, fright, anger and excitement cause temporary xerostomia. Allington (1950). Xerostomia of nervous origin may be associated with nervous strain, depression, hysteria, emotional disturbances or following emotional shock. There are cases of lecturers and singers who could perform well before small audiences of their friends, but not to large audiences as a result of their (psychogenic) dry mouth. These patients have been called Xerophobes.

Physiological disturbances in the end organs of the nerves innervating the salivary glands may result from occupational hazards involving organic dusts, Zinc poisoning and Botulinus poisoning. Furstenberg etal (1945).

Organic lesions of the Central Nervous System may interrupt the central pathways of the secretory nerves, but in these cases more significant and conspicuous cranial nerve palsies are to be seen. Allington (1950).

Although a decreased rate is seen in early schizophrenia and multiple sclerosis, in the later psychotic stages of schizophrenia there is sialorrhea as there is in cases of microcephalic children, dementia paralytica and post-encephalitic patients.

There is thought to be a balance between cerebral function,

subcortical activities and the behaviour pattern of the child. A dry mouth will not be produced by the dysfunction of a single salivary gland, by the section of peripheral fibres of cranial nerves seven or nine, nor by the destruction centrally of the superior or the inferior salivatory nuclei. Certain types of lenticular involvement, however, cause sialorrhea. Furstenberg (1945).

Peripheral section of Cranial nerve seven or nine will not immediately produce Xerostomia of even a partial nature, although ultimately it does so. For a short time following nerve section the gland continues to secrete "paralytic saliva" which eventually disappears with the degeneration of the gland alveoli. There is some compensation on the part of the other glands provided their nerves are intact.

Suher etal (1953) describes a case of Xerostomia associated with the very rare Moebius syndrome, a congenital facial diplegia with involvement of the other cranial nuclei.

The syndrome first described by Sjogren in 1933, and now bearing his name may be responsible for a complete (or nearly so) Xerostomia and keratoconjunctivitis sicca, the mucosa is always dry, the patient is continually licking her lips, the mouth and tongue are painful and there is dysphagia. The caries rate is increased and there are recurrent Parotid swellings. This disease is most frequently seen in menopausal women and the endocrine theory of its aetiology is quite popular. Morgan and Raven (1952), Allington (1950). Although the nutritional theory has been abandoned through lack of evidence, Neurotropic factors and Chronic Infection should still be considered.

The effect of anxiety on salivation varies from one person to another and cannot be predicted. Xerostomia may occur typically in states of depression and can be a severe neurotic symptom. Moulton (1955).

Glassner (1948) was able to cure a case of Xerostomia in an elderly patient by constructing dentures with a new vertical dimension.

Faber (1943) gives consideration to the estimation of salivary

flow in the normal and diseased state. His first test involves the placement of a piece of sugar beneath the tongue; in the young child the lump dissolves in about 15 minutes; in the older person 20-25 minutes; in the very old 30 minutes; and in cases of Xerostomia 60 minutes or longer. It is important that the patient does not manipulate or suck at the sugar.

The quantitative collection of saliva in measuring cylinders in the resting patient (sitting quietly and avoiding swallowing) prior to and following the injection of Pilocarpine may provide valuable information.

The management of Xerostomia varies with the aetiology; if disease is present it must be treated. Frequently, however, the patient requires symptomatic treatment and in these cases it is often possible to assist by the therapeutic stimulation of the salivary glands. The drugs used include Pilocarpine nitrate or Hydrochloride, and Neostigmine bromide. Denture wearing patients should coat their prosthesis with Petrolatum or other lubricating jelly. The lips can be similarly treated. Burket (1957).

Thoma (1954) considers an alkaline drinking water (Vichy Water) improves the salivary flow in some patients.

The relation of the saliva to the wearing of dentures is considered by Mehringer (1954).

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