

SPECIFIC INFECTIOUS GRANULOMATA

Syphilis.

Tuberculosis.

SYPHILIS (Syn = with. philos = loving)

is an infectious disease, generalised at first, subsequently localised and dispersed, which may involve any organ of the body. A vasculitis of the smaller vessels is the characteristic histologic lesion. Cecil & Loeb (1956).

The causal organism is the spirochaete, *Treponema pallidum*, a slender spiral organism measuring 5-20 μ in length and 0-2 μ in thickness. It has 6-14 regular but fairly angular spirals, is actively motile and rotates on its long axis at a fairly constant speed. Dubos (1948).

The disease may be acquired innocently following the handling of an infected lesion, Leifer (1942), through medical and dental instruments, eating utensils, pipes, etc.

However, the more usual mode of transmission is by sexual intercourse and sexual perversions of an oro-sexual nature (fellatio and cunnilingus).

It is a significant fact that the organism can not tolerate any dehydration and requires a liquid vehicle for its transfer from host to host.

Following its passage through the epithelium at the site of deposition, the organism multiplies and extends via the lymphatics to the regional lymph nodes from which it is disseminated throughout the body by way of the lymphatics and venules to the venous circulation. It enters the pulmonary circulation and is finally transported by the arterial circulation. Thus within twenty-four hours we have a focus of infection near the site of implantation, innumerable metastatic foci throughout the body and a spirochaetaemia which is augmented continuously or intermittently by the primary or metastatic focus.

If it is to appear, and in some patients it does not, the primary chancre is found near the site of initial penetration of the organism. Usually after exposure an incubation period of

between ten and nineteen days is followed by the appearance of the primary, hard, true or Hunterian chancre. The chancre may be located on or about the genitalia or perianal region (genital chancre) or as an extra genital chancre where it may involve the oral cavity, fingers or the female breast.

Cecil & Loeb (1956).

According to Epstein & Zeisler (1933) extragenital chancres comprise 5-7 percent of lesions, and about 75 percent of these are found in the head and neck region; of these 80 percent occur within the mouth.

Muir (1951) describes the appearance of the chancre in its earliest stage as a papular thickening of dull reddish tint. Later it becomes hard, raised and indurated; it is seldom more than a half inch in diameter. It ulcerates in several days and becomes crusted over. The base is very firm and when palpated feels like a rubber button beneath the tissue. Colby (1956).

Straith (1937) states that regional lymphadenopathy appears in a few days and although initially limited to one gland, later involves others. Chancres of the oral region may produce satellite bubo of the submaxillary, submental and pre-auricular regions. These nodes are hard, immobile and painless. Resolution and healing of the chancre occurs after some weeks.

Oral chancre are found on the lips, the tongue, the gingivae and the tonsil and have been described by Straith (1937) as typical and atypical. His typical oral chancre resembles basically that found elsewhere and is described supra. It is an erosive, ulcerated lesion, well limited but with borders not clearly defined. The surface is smooth and glossy and has a raw beef or carmine colour. It may not be indurated. The shape varies and crescentic or half moon shapes are seen with a concavity directed towards the necks of the adjacent teeth. Either one or several teeth may be involved.

The base becomes raised and papular as the ulceration progresses and it may become covered by a grey pseudomembrane.

The atypical lesion has no particular characteristics, the surface may be granular and of a different colour, the exudate may be purulent or bloody and there is slight pain. Straith includes a good bibliography from the European literature in his communication.

Epstein et al (1933) think that any difference seen in the gingival chancre is due to the lack of tissue thickness and the presence of the underlying bone. Faier (1952) considers the gingival chancre a rarity.

The lesions of secondary syphilis appear in 3-4 weeks, Thoma (1956), 2-12 months Colby, (1956) within 2-3 months Stones (1954) after the appearance of the chancre. They are ushered in by the so called fever of invasion which is associated with a sore throat and syphilitic angina. The manifestations at this stage involve cutaneous and mucous surfaces. The pink and macular Roseola is the earliest lesion and may appear anywhere on the body surface. As a result of inflammatory infiltration it becomes the papular syphilide which may progress to the vesicular and later the pustular syphilide. Upon desquamation and with the production of silver scales the papular lesion becomes the squamous syphilide.

The specific oral lesions are: syphilitic cheilitis; snail track ulcers; mucous patches, mucous tubercles, condylomata; and syphilitic gingivitis.

The presence of erosive papules on the lip gives rise to syphilitic cheilitis. The papules are discrete, oval, rounded, greyish-white or reddish-pink patches with an inflammatory halo, which may involve any portion of the mouth. Thoma (1954).

As with the cutaneous lesion the papular syphilide may progress to the vesicular or pustular stage. The erosive papule of the mouth does this with the production of the mucous patch. Burket (1957) considers them to be the most infectious lesion of acute syphilis.

The mucous patch is grey-white, slightly raised and surrounded

by an erythematous base. Pain may be present as a result of tissue movement and oral trauma (which produces a raw bleeding surface). Areas involved include the inner surface and vermillion border of the lips, the tongue, the pharyngeal mucosa and the palate. Stones (1954). In the healing phase the surface of the lesion becomes a translucent grey. Mucous tubercles are mucous patches which have grown out as projections. Butlin (1885) reminds us that the appearance of the mucous patch varies with its site but notes that the patch is initially a very small grey-white spot which enlarges quickly without any sign of inflammation. Larger and irregular patches are formed by coalescence.

Condylomata may form beneath the tongue in areas away from excessive trauma. They are warty (like a cauliflower) and dead white in colour, their base is slightly constricted, but shows no sign of inflammation, the surface is rarely elevated. In more exposed areas the condylomata appear notched, sinuous, sometimes ulcerated and surrounded by a bright red areola.

Snail track ulcers are most frequently seen on the anterior pillars of the fauces of the buccal and the pharyngeal mucous membranes. They develop from catarrhal patches. Muir (1951).

Tertiary lesions may appear within a year of the secondary lesion or after many years. They are characterised by chronic interstitial inflammation with the formation of masses of granulation tissue which may undergo central necrosis (gummatous change), cicatrization and healing. Muir (1951).

Solitary gumma are most frequently found on the tongue, the palatal mucosa, the uvula, Thoma (1954), the tonsils, Stones (1954), the salivary glands and the jaw bone, Burket (1957). The lesion initially appears to be smooth, shiny and tense with the production of a sub-epithelial swelling. Central necrosis follows and the typical ulcer is produced.

A necrotic area with margins sharply defined, overhanging and deeply excavated; a fundus of dirty colour with an uneven

base and indolent character. Thoma (1954). Crusting follows exposure to the air. There is apparently no pain. Stones (1954). Butlin (1885) states that superficial gummata involving the tongue break down with the formation of long lines or fissures lying in a longitudinal direction. These gumma may be so small that they are unrecognisable as such, but feel like hard and knotted cords in or immediately beneath the mucosa. The fissures thus formed eventually widen and deepen with the production of a sharply cut ulcer with perpendicular walls and a surface covered partly with slough and discharge. The surrounding tissues are usually swollen and indurated but not very hard. Function is interfered with as a result of their sensitivity. The deep gummata break down and discharge through a small opening which gradually enlarges to produce a formidable, angular, cleft-like, round or irregular ulcer. The hard and soft palate are involved frequently and perforation may occur. Stones (1954). Huebsch (1955) reports a case of palatal perforation. Butlin (1885) describes the formation of tertiary syphilitic plaques (tubercular syphilide; possibly); multiple irregularly rounded, smooth, raised and erythematous. Later they become cracked, glazed and shiny.

Sclerosing glossitis (glass tongue) according to Thoma (1954) is associated with an obliterating endarteritis which causes atrophy of the lingual papillae. The epithelium is very thin and there may be a hyperkeratosis. Rhagades sometimes form and divide the tongue into lobes. Schaffer (1951-52) describes these lobulations associated with a wine-coloured tongue devoid of papillae. He hypothesises that the fibrosis is the result of the healing of a gumma or of initial fibrosis.

PRENATAL SYPHILIS.

A foetus is infected during the latter half of gestation and may die; be aborted; be born with the stigma of syphilis or be born without it only to develop it later. Stones (1954). The chance of intra-uterine transmission is greatest when infection has been recently acquired and diminishes throughout successive pregnancies. Cecil & Loeb (1956).

The child's face is that of a senile old man, Thoma (1954), Burket (1957). A maculo-roseolar eruption may appear about the nose and mouth. Ulcerocrustaceous lesions and Rhagades, Thoma (1954), form about the mouth and form permanent scars in later life. Involvement of the tongue by small nodular papillomatous and warty growths on its under surface is described by Carr (1948). (Condylomata lata of the tongue).

After the first year gummatous destruction of the hard and soft palate may occur.

A triad of interstitial keratitis, eighth nerve deafness and Hutchinsonian teeth has been described and named by Hutchinson. The differential diagnoses between the facies of congenital syphilis and hereditary ectodermal dysplasia has been considered by Horne (1954).

De Wilde (1943) describes involvement of the deciduous dentition, which Burket (1957) claims to be rare. The eruption may be tardy or delayed and the incisors may be notched. A general account of "syphilitic teeth" is given by Fournier (1884) and also by Cavallaro (1909).

The Mulberry Molar and the Bud Molar of Pfluger are more frequently seen than the Hutchinsonian incisor. Thoma (1954).

The diagnosis of syphilis depends on the serologic (Wassermann) and flocculation (Kahn) tests. It is important to hold in mind, however, that positive serologic tests are not always diagnostic because biologic false positive reactions may be obtained during convalescence from an acute infection, pregnancy and in some tropical diseases. Cecil & Loeb (1956), Lucas & Kramer (1954).

Direct examination of exudate from the lesion is carried out by dark field microscopy. Low & Dodds (1947).

Although penicillin appears to be the drug of choice, Willcox (1951) the tetracyclines have considerable effectiveness, Cecil & Loeb (1956). Arsenic, Mercury, Bismuth and Iodine were used extensively in pre-penicillin days.

TUBERCULOSIS.

Tuberculosis is an infectious disease caused by *Mycobacterium tuberculosis*; it is widespread among men and animals and may be acute and generalised or chronic and localised. The disease is characterised pathologically by inflammatory infiltration, tubercles, caseous necrosis, abscesses, fibrosis and calcification. Cecil & Loeb (1956).

Originally known as consumption or phthisis it was first recognised by Laënnec, whose conceptions were proved by Koch in 1882, who isolated the specific organism. The avian type was isolated by Maffucci in 1890 and the bovine type by Smith in 1898.

The causal organism is the acid fast non-sporing, non-capsulated, non-motile aerobe, *Mycobacterium tuberculosis* which exists in three types: *Hominis*, *bovis* and *avium*. Man may be affected by any of the three types but most frequently it is by *hominis* and *bovis*.

Direct contact with an infected person is found to be the most frequent cause, either by inhalation of contaminated air, contaminated faeces and urine, mouth to mouth contact, superficial tuberculous sinuses, wound infection (especially by Butchers, Post Mortem Personnel and Laboratory Workers) and by ingestion of cow's milk or meat. The foetus may be infected through the placenta (haematogenous transfer) or the amniotic fluid (by inhalation). Dissemination may be by Lymphatic, Lymphoheamic, Heamic and Intracanalicular routes, also through tissue continuity.

Oral tuberculosis is a comparatively uncommon occurrence according to Oppenheim et al. (1951), and Shengold et al. (1951). Thilander et al. (1956) contributes this to the properties of the oral tissues, the presence of saprophytes, the thickness of epithelial covering, the antagonism of striated musculature to bacterial invasion and the cleansing action of saliva. The anti-bacterial effect of saliva on the tubercle bacilli has been demonstrated by Kanter and Appleton (1940). The predilection of

the organism for lymph tissue is significant according to Shengold et al (1951) who point out that the relative paucity (of Lymph tissue) in the oral cavity may be a responsible factor for the decreased frequency of infection. Thus the anterior of the mouth is less susceptible to tuberculous infection than the posterior, which is richer in Lymph tissue.

Oral tuberculosis is usually a manifestation of far advanced pulmonary tuberculosis, Shengold et al (1951), but much controversy has arisen over the probable pathways of dissemination. The view that the positive sputa of pulmonary tuberculosis infects the abraded oral mucosa is extremely attractive, but in fact this rarely occurs, Oppenheim et al (1951), since oral lesions are rarely seen in connection with a positive sputa and pulmonary tuberculosis. With a negative sputa and pulmonary tuberculosis we more often see oral lesions, thus favouring dissemination by an endogenous route. Shengold et al (1951) is less adamant and states that an endogenous or exogenous pathway may be involved. The exogenous route is favoured by many; there is constant trauma with resultant abrasion of a mucosa constantly bathed (by a positive sputa) by tubercle bacilli. The theory of an endogenous route has much to favour it. Pulmonary exacerbations and extra pulmonic invasion may coincide with the appearance of single or multiple oral lesions in regions which are subject to least trauma and in the presence often of a negative sputa. Those favouring this route remind us that exodontia is rarely followed by auto inoculation. Shengold et al (1951) have hypothesised that chronic trauma precedes the endogenous, and external trauma the exogenous routes.

Tuberculosis of periapical granuloma are more common than realised, Shengold et al (1951), and Thilander et al (1956) suggests that non-specific apical granuloma are frequently secondarily infected by the tubercle bacillus. Brodsky et al (1943) gives three possible routes of infection, firstly from a salivary source through a carious lesion, secondarily via deep peridental pockets

and thirdly via the heamic route. Thoma (1954) quotes Gornouec as stating an incidence of 20% in cases of open tuberculosis. Soft tissue lesions may extend and involve the maxillae and mandible with a resultant periostitis, osteitis or osteomyelitis. Periapical granuloma have been similarly convicted, Shengold et al. (1951). Spilka (1955) considers that tubercular osteomyelitis of the mandible is quite rare. A description of the mucosal and bony lesions is given by Hotz (1949).

The mucous membranes are involved comparatively uncommonly according to Oppenheim et al. (1951), who reports, however, that oral tuberculosis may take the form of an ulcer, fissure, fistular, granuloma, tuberculoma, glossitis or lupus.

The ulcer is the most common lesion seen in the mouth and varies greatly in size and distribution. Although the tongue is the most frequently involved, the cheeks, lips and the palate are also affected. Burket (1957).

The shape of the ulcer is inconsistent and may be round, oval, fissured or irregular. The base is oedematous and may be covered by caseous material. Undermined edges are often surrounded by bluish tissues.

Bruce (1952) described an ulceration of the gingiva covered by a pebbly granulation tissue, the centre had sloughed and there was a grey-yellow exudate present. The edge was undermined and had a moth-eaten appearance.

Prostatic tuberculosis with an associated oral lesion of the tuberosity has been described by Osman et al. (1950). This granulating mass was sharply demarcated, with a depressed central region. Osman does not believe that primary tuberculous of the prostate exists. The view that oral tuberculosis may exist before rapidly progressing tuberculosis in other parts of the body has manifested itself is held by Collins et al. (1940).

Although tuberculous periodontitis occurs only extremely rarely as a pure infection, it is more frequently seen in a mixed form, and this has been noted by Thilander (1956).

Lupus vulgaris is a cutaneous manifestation of tuberculosis; the initial lesion which begins as a yellow-red intra-dermal nodule (which pales on pressure giving the appearance of a yellow-red spot) which breaks down and ulcerates. Cicatrization follows. Stones (1954). The oral mucosa may become involved by extension and the mucosal lesions are similar to the cutaneous. Cheyne's (1949) case showed a vesicular scaly and crusting lesion, with very little tendency to the formation of large ulceration. Bright red lesions appeared on the gingiva but were not painful and did not ulcerate. Scarring followed healing.

Air embolism in the vessels supplying the tongue will cause marked pallor of this organ. This symptom of Liebermeisters syndrome may be the first indication of a fatal air embolism and may occur during pneumothorax therapy for Tuberculosis.

Burket (1957).

The diagnosis of tuberculosis may follow the examination of sputum, Tolhurst et al (1955); animal inoculation, surgical biopsy and surgical aspiration, X-Ray examination of lung fields and the tuberculin test.

TREATMENT.

The general principles of treatment are aimed at improving the patient's general health, preventing spread of the lesion and increasing resistance. The use of specific chemotherapeutic agents has been well described by Tolhurst et al (1955).

They stress the use of several drugs concurrently to prevent or delay emergence of resistant bacilli and to obtain a better therapeutic effect.

Drugs used include Streptomycin, Dihydrostreptomycin, Isoniazid, Iproniazid, Para-aminosalicylic Acid, Viomycin, Terramycin, Amithiozone and the Sulphones. Cecil and Loeb (1956).

SARCIODOSIS.

Although a disease of unproven etiology, many observers have cited the acid fast organism *Mycobacterium tuberculosis* as the causal organism. Some writers think that *Mycobacterium Leprae* is responsible. The fundamental lesion is granulomatous and is seen to involve most organs and structures, the skin and the mucous membranes. Cecil and Loeb (1956). The disease may involve the mandible. Poe (1943) or the maxilla Kallman (1954).

Well defined papular nodules or plaques may form on the lips, Thoma (1954), and may be accompanied by a boring sensation. Poe (1943).

Uveoparotid fever, an expression of sarcoidosis, is accompanied by bilateral parotid swelling, Xerostomia and involvement of the Seventh Cranial Nerve. Cecil and Loeb (1956). There is no specific treatment.

THE INTOXICATIONS.

Acids (Chromic, Hydrochloric, Hydrofluoric,
Nitric, Sulphuric, Tartaric), Aniline,
Antimony, Arsenic, Benzene, Bismuth, Cadmium,
Chromium, Copper, Fluoride, Gasoline, Gold,
The Iodides (Bromides, Creosol, Salicylates),
Iron, Lead, Mercurials, Methyl Bromide, Methyl
Dichloride, Methyl Mercury, Paranitraniline,
Silver, Tellurium, Tetrachloroethane,
Trichlorethylene, Trinitrotoluene, Thallium,
Vanadium, Zinc.

ACIDS.Chromic Acid.

In the chromium plating industry, workers are exposed to the acid in the form of a fine, misty, brownish spray containing about 60 per cent chromic acid. Walters et al (1952), Abstract (2)

The tongue is swollen, sore and dry, and the dorsum, which may be coated and cracked, exhibits reddened and enlarged papillae. The buccal mucosa has a milky, sheen and appears thickened. The patient may complain of some degree of ageusia.

Small, multiple and sharply defined ulcers are seen on the floor of the mouth, the tongue and the soft palate. They are covered by a white pseudo membrane which, when removed, does not cause haemorrhage. Walters et al (1952), Abstract (1).

Hydrochloric Acid.

Following ingestion, hydrochloric acid causes severe burns of the lips, the tongue and the throat. The lesions, initially white, become dark brown and are extremely painful. Walters et al (1952), Abstract (3).

Hydrofluoric Acid; Nitric Acid; Sulphuric Acid; Tartaric Acid produce varying degrees of erosion of the teeth, depending on the concentration of the acid and the duration of the exposure. Walters et al (1952), Abstracts (4), (5), (6) & (7).

ANILINE

is used widely industrially in the manufacture of dyestuffs, germicides, paints, varnishes and perfumes, Walters et al (1952) Abstract (80)

Although it is called the "blue lip disease", Walters et al (1952) Abstract (82), there is initially a mucosal pallor which is rapidly followed by the typical cyanotic appearance, flushing of the face, mental confusion, dysphagia, convulsions, coma and death. Walters et al (1952) (Abstract 83)

Mild intoxication is manifest by a mild cyanosis of the cutaneous and mucous surfaces. Walters et al (1952) Abstract (81)

The lips turn blue, Walters et al (1952) Abstract (80), after the initial pallor, then purple, Walters et al (1952) Abstract (84), and in severe cases the patient may turn black. Walters et al (1952) Abstract (81)

ANTIMONY

Unless it is accompanied by lead poisoning, antimony poisoning of occupational origin is rarely dangerous.

The oral manifestations are non-specific, but include mucosal irritation and the appearance of small oral ulcers. Walters et al (1952) Abstract (42)

ARSENIC

poisoning is seen most frequently in workers handling arsenical materials, but may follow its therapeutic usage. Cecil & Loeb (1956) . Homicidal or suicidal administration should also be considered.

A serious desquamative dermatitis, Sweitzer (1941), may be accompanied by arsenic stomatitis. The oral tissues are deep red in colour and very painful. Xerostomia is present. A persistent and painful ulceration follows contact with arsenic trioxide. Burket (1957)

Stones (1954) describes the appearance of pustules on the lips and at the angles of the mouth.

The development of bullous lesions about the mouth after the injection of "Trisodarsen" (an arsenical) is described by Pines (1941).

The breath has a garlic-like odour. Walters et al (1952) Abstract (30)

BENZENE

Oral symptoms exhibited by workers poisoned by Benzene vapours are: Bleeding of the gingivae, ulcers and haemorrhage of the oral mucous membranes, oral pain, sore throat and a dry tongue. Walters et al (1952) Abstract (87). Small ulcers with necrotic bases on the hard palate, tongue and inside of the cheek were discovered at autopsy in one case. Walters et al (1952) Abstract (89)

Purpura haemorrhagica with gingival haemorrhage occurred in another group of cases. Walters et al (1952) Abstract (88)

BISMUTH

Bismuthism may result from the use of Bismuth therapeutically, Burket (1957), or cosmetically. Thoma (1954). There are apparently no important occupational sources of intoxication.

Bismuth pigmentation is a common occurrence, but the stomatitis is rare.

Odontalgia may follow the injection of a Bismuth compound, areas of the mouth become pigmented and in severe cases a yellow pseudo membrane forms on the mucosa, accompanied by regional lymphadenopathy, trismus, salivation and fetor oris. In very grave cases there may be haemorrhage and necrosis of the mucosa and alveolar bone. Thoma (1954)

Bismuth pigmentation is seen with or without the ulceration and a slight bismuth line may be quite asymptomatic, although it is sometimes accompanied by an ulceronecrotic gingivostomatitis. A marked metallic taste, stomatopyrosis and glossitis may occur. The line which appears well demarcated to the naked eye is found, on examination with a hand lens, to be distributed diffusely. Burket (1957). Stones (1954) describes it as a black or purplish line at the gingival margins.

Prinz & Greenbaum (1939) consider that the pigment may take the form of small, black dots, or short lines, while in larger amounts a diffuse black line may encircle the teeth. A greyish-blue line is seen along the occlusal line of the buccal mucous membrane.

The rate of appearance and the intensity of the line vary with the absorption rate and amount, and with local conditions within the mouth. In the clean mouth with sound, hard and soft tissues the blue line takes time to appear, but in the presence of infection it appears rapidly.

Burket (1957) describes involvement of the lips, the tongue and the cheek.

A mechanism of pigmentation has been suggested. Bismuth

salts unite with Haemoglobin or an acid radical of the blood, reach the capillaries of the gingival tissues, and are precipitated. Hydrogen sulphide reacts with this precipitate to form blue-black bismuth sulphide. The hydrogen sulphide is produced by bacteria from decomposing organic material; thus in the dirty mouth there is more hydrogen sulphide produced than in the clean mouth, and hence the difference in pigmentation. Burket (1957)

A simple test to differentiate the lead line from the bismuth line involves placing a small volume of hydrogen peroxide on the discolouration. The lead line will become white with the formation of white lead sulphate, but the bismuth line remains black.

Prinz & Greenbaum (1939)

The treatment of the oral lesions is aimed at the maintenance of good oral hygiene. General treatment includes the use of BAL and Contramine. Stones (1954)

CADMIUM

The occupational exposure of at least two years' duration to Cadmium dust and fumes results in the formation of a yellow ring about the teeth at the labio gingival margins. The ring varies in colour from a light yellow to a golden-brown. Walters et al (1952), Abstract (43). Some patients complain of a sore throat and metallic taste. Walters et al (1952), Abstract (45).

An increased rate of salivation may accompany an acute gastrointestinal episode. Walters et al (1952), Abstract (46).

CHROMIUM

Perforation of the nasal septum and multiple ulceration of the oral cavity with etching of the teeth and the development of a deep orange stain upon them, may occur in chromeplaters exposed to the irritating and corrosive spray. Burket (1957), Prinz & Greenbaum (1939).

COPPER

Although fatal cases of poisoning have been reported, Walters et al (1952), Abstract (47), occupational exposure to copper results in the development of a green, Prinz & Greenbaum (1939), or blue-green line on the teeth and gingiva. Burket (1957).

FLUORIDE.

An extensive consideration of many of the aspects of Fluorine intoxication has been made by Roholm (1937).

Soft tissue effects have been observed by Walters et al (1952) Abstract (33). The nasopharynx, soft palate and fauces were red in colour and there was a sharp line of demarcation between the soft and hard palate. The posterior pharyngeal wall was irregularly raised. A thick bluish-grey mucoid discharge, impregnated with dust was observed in most cases.

The consumption of water having an excessive amount of fluorine causes a typical enamel hypocalcification, Colby (1956), which has been considered in detail by Thoma (1954).

GASOLINE AND BENZINE

are used industrially and in the home. Acute intoxication results from ingestion or inhalation, and is sometimes fatal. It may produce necrosis of the oral mucosa and throat, Walters et al (1952) Abstract (92), and deviation of the tongue. Walters et al (1952) Abstract (93)

GOLD

Although occupational sources are present, it is the intense therapeutic use of gold in the treatment of cutaneous tuberculosis, arthritis and leprosy etc. which commonly causes the condition. Stones (1954)

Auric stomatitis is characterised by erythematous, congested and sometimes ulcerated oral mucosa. The gingiva appear faintly purple, the patient may complain of a metallic taste and often the lips are cracked and fissured. Burket (1957)

Local treatment includes the topical application of the aniline dyes and the use of a mildly alkaline mouth-wash. Burket (1957)

B.A.L. is of value in the general treatment. Stones (1954)

IRON

causes a blackish discolouration of the dorsum of the tongue, and scrapings from these areas contain a considerable amount of iron. Walters et al (1952) Abstract (48)

IODIDES

The Iodides rarely cause an inflammation of the oral or pharyngeal mucous membrane. Sialorrhoea is a symptom of iodism, however, and may accompany the inflammatory condition if it occurs.

The Bromides on rare occasions may also give rise to inflammatory lesions of the oral mucosa.

Creosol may give rise to stomatitis.

The Salicylates may, on rare occasions, give rise to erosive lesions of the oral mucosa and cutaneous eruptions. Stones (1954)

LEAD - Plumbism

Lead poisoning is a toxic state which results when the level of lead absorption exceeds that of excretion. Considerable amounts of toxic lead compounds may be absorbed, stored and excreted without producing intoxication. Lead is absorbed through the lungs in the form of a dust or vapour, through the unbroken skin or following the ingestion of its compounds.

After absorption, lead is deposited in viscera, but particularly in the liver, and then later it is transferred to the skeleton for storage, mainly as trilead phosphate. Cecil & Loeb (1956)

Possibly one of the earliest symptoms to appear is the lead line or seam (halo saturninus, Burtonian line), considered to be pathognomonic of lead absorption, but not necessarily indicative of lead intoxication. This line is seen to be more marked in areas of malhygiene and may take the form of a fine bluish line, more pronounced on the papillae or as a bluish black line 5-10 mm. in width, involving the whole gingival margin and blending into gum. It is rarely seen in the edentulous mouth. Prinz & Greenbaum (1939)

Other portions of the oral mucosa, especially the lips and the cheeks, may show this pigmentation, and there appears to be degrees of involvement. The gingiva of a single tooth may show a faint mottled patch at its extreme edge, or a fine blue-black line may follow the entire gingival margin. A third type is similar to that described by Prinz & Greenbaum (1939), Thoma (1954)

Walters et al (1952) Abstracts (52), (54) describe cases of plumbism with the appearance of the lead line.

The lead line may be very valuable in diagnosis, but should not be confused with the gross discreteness of the bismuth line. Burket (1957)

The line is due to the deposition of lead sulphide about the blood vessels in the tips of the papillae. When examined with

an optical lens, it has a punctate appearance, and seems to lie a little below the margin of the gum. A piece of paper inserted between the gum and tooth distinguishes typical stippling from staining of the tooth root. Levy (1943)

A sweetish taste is present in the mouth on rising. Halitus saturninus (lead breath) may be present. Prinz & Greenbaum (1939)

Whitby & Britton (1957) discuss the haematology of lead intoxication, and consider that a hypochromic or normochromic anaemia in association with a high stippled cell count should always suggest lead poisoning.

Since lead salts are irritating to the oral mucosa, Burket (1957) suggests that by lessening the flow of saliva, a decreased irritant effect is produced. Scrupulous oral hygiene should be at all times maintained, Walters et al (1952) Abstract (49)

MERCURIALISM.

The mercurials may be administered accidentally, therapeutically or with suicidal intent, and since they are rapidly absorbed after ingestion, vomiting, diarrhoea, collapse and delirium follow in a short interval. The triad of diarrhoea, sialorrhoea and gingivitis is seen in cases of sub-acute poisoning, usually following the excessive therapeutic use of one of the mercurials, and subside upon withdrawal of the drug. Cecil & Loeb (1956)

The gingiva, which have a haemorrhagic tendency are initially red and swollen, but later necrosis may occur and a slough forms. A glossitis may accompany this mercurial stomatitis, and is characterised by a slight swelling of the organ and the formation of a white coat which later becomes a greyish white gangrenous membrane. The teeth are covered with sordes and the mucosa often with ulcers. Walters et al (1952) Abstract (68). The tonsillar region and soft palate present with a diffuse redness. The patient complains of a metallic taste, there is marked fetor and profuse salivation.

Periodontal involvement renders the teeth tender to percussion. Stones (1954)

Horizontal alveolar erosion is accompanied by demineralisation of the alveolar bone, Walters et al (1952) Abstract (56), and has been attributed to both the mercury and secondary infection. Although mercurial pigmentation of the alveolar gingiva may be a faint diffuse grey colour, Burket (1957), a blue or dark brown mercury line may be present on the gingiva, Walters et al (1952) Abstract (59), and a coppery colour on the mucosa. Walters et al (1952) Abstract (64)

The mouth is hot and pruritic, and the lips are dry, cracked and swollen. Burket (1957). Speech, mastication and deglutition are very difficult. Walters et al (1952) Abstract (68)

If the sub-maxillary and sub-lingual salivary glands are involved, they are usually quite tender. Stones (1954)

Walters et al (1952) Abstract (59) report that a tremor of the lips and tongue is a frequent manifestation.

The presence of oral manifestations depends very much upon local conditions. In the clean and edentulous mouth they are rarely seen; in the dirty mouth more frequently seen.

The mechanism of appearance of the oral lesions, in which mercuric albuminate is precipitated in the capillary loops is well described by Prinz & Greenbaum (1939).

Local treatment is aimed at the maintenance of oral hygiene. The secretion of mercury is promoted by British Anti Lewisite (B.A.L.). Stones (1954).

Acrodynia - Swift's Disease - Pink Disease

Acrodynia is primarily a disease of infancy and early childhood. Characterised by painful red swollen hands and feet, tachycardia, hypertension, hypomotility, mental apathy, anorexia and photophobia. Cecil & Loeb (1956).

Warkany & Hubbard (1953) have shown, not conclusively, but convincingly, that Acrodynia results from an idiosyncrasy or hypersensitivity to the mercurial drugs. In all Warkany's cases the children had been exposed to mercury. Other workers are not in agreement with this theory, and Clements (1940) considers another three aetiological possibilities.

The gingival tissues are a deeper red colour, and markedly inflamed and swollen. There is an intense sialorrhoea and saliva frequently drools from the mouth in a constant stream. Nelson (1954).

Apparently healthy teeth are loosened and lost, and portion of the alveolar process may be "shed". A case is reported by Bernard (1937) Warkany et al (1953).

METHYL BROMIDE

Patients intoxicated by the inhalation of the fumes of Methyl Bromide may complain that everything tastes bitter and of burnt rubber. Walters et al (1952) Abstracts (94), (96)

Tremor of the tongue and hands is an occasional sequelae. Walters et al (1952) Abstract (95)

METHYL CHLORIDE AND METHYL DICHLORIDE

A definite chloroform-like odour is produced on the breath, Walters et al (1952) Abstract (97), of the person poisoned by Methyl Chloride or Methyl Dichloride. Walters et al (1952) Abstract (98)

METHYL MERCURY

Sialorrhoea, soreness of the gingiva and numbness of the tongue were reported in cases of poisoning by Methyl Mercury. Walters et al (1952) Abstract (99).

PARANITRANILINE

is a yellow powder used as a dyestuff in the textile industry. Walters et al (1952) Abstract (100). An intense violet-coloured cyanosis of the lips and tongue, Walters et al (1952) Abstract (102), develops several hours after exposure. The tongue initially has a blue margin but assumes a leaden hue later. Walters et al (1952) Abstract (101)

This lead-like colour, an important diagnostic sign in cases of acute paranitraniline poisoning, may appear not only on the tongue, but also on the lips, and on the tips of the finger and toenails.

SILVER

Industrial or therapeutic exposure to silver or its compounds may result in Argyria, a disease state associated with pigmentation

of the skin and mucous membranes. Burket (1957).

Although localised argyria is still seen on occasions, generalised argyria has become a rarity. Walters et al (1952) Abstract (69)

A violet line with a metallic lustre may appear at the gingival margin due to reduction of resulting silver albuminates in the tissue by light or by sulphur dioxide from decomposed deposits on the teeth. The prognosis in true Argyria is unfavourable, and, if symptoms occur in the mouth, the treatment should be discontinued. Thoma (1954)

TELLURIUM

A large number of workers in plants where Tellurium was added to molten iron had a garlic-like odour on their breath. Walters et al (1952) Abstracts (70), (71), (72) & (73)

In higher concentrations there may be a blackish grey discolouration of the lips, the teeth, the tongue and the angles of the mouth. Walters et al (1952) Abstract (73)

TETRACHLOROETHANE

Workers using Tetrachloroethane may complain of a peculiar taste in the mouth and an irritating sensation at the back of the throat. Walters et al (1952) Abstract (103)

TRICHLOROETHYLENE

A sweetish taste in the mouth is an early symptom of Trichlorethylene intoxication.

TRINITROTOLUENE

may cause a very unpleasant taste in the mouth. Walters et al (1952) Abstract (105)

THALLIUM

Acute Thallium poisoning causes hypertension and renal disturbances, and can be compared with lead poisoning.

Although intoxication may result occupationally, accidental poisoning does occur; in children during treatment of ringworm of the scalp or following ingestion of rat baits, or in some cases by those with homicidal or suicidal intent.

A generalised ulcerative stomatitis occurs with the appearance of a blue line on the gingiva. The patient may complain of a metallic taste, an xerostomia or in some cases sialorrhoea. Walters et al (1952) Abstracts(74),(75),(76)

The gums are swollen, red, and bleed easily. Winter et al (1954)

VANADIUM

Is used in a great number of industrial processes, and workers exposed to its dusts become intoxicated.

The oral manifestations due to exposure are greenish-black discolouration of the tongue, especially when it is furred or when septic teeth are present and a salty taste in the mouth. Walters et al (1952) Abstract (77)

ZINC

Zinc intoxication occurs chiefly as an occupational hazard in (molten) brass workers, electric welders and galvanisers.

Zinc stomatitis is characterised by a bluish-grey line, Burket (1957), or bluish discolouration of the mucosa. Prinz & Greenbaum (1939). Initially the patient complains of a sweet taste in his mouth, but later that his mouth feels "fuzzy" and he has "lost his taste." (Ageusia)

The gingival tissues shortly become congested and the teeth

mobile following destruction of the alveolar process.

Submaxillary lymphadenopathy and salivary gland involvement may occur. Burket (1957)

DISEASES OF METABOLISM

DIABETES MELLITUS.

DIABETES MELLITUS

is the best known of the pancreatic diseases, and has been recognised for about two thousand years. Comroe et al (1954). It is essentially a disorder of carbohydrate metabolism, associated with the disturbance of the normal insulin mechanism and characterised by hyperglycaemia and glycosuria. Cecil and Loeb (1956):

DIABETES.

There are no specific lesions pathognomonic of Diabetes manifested in the oral cavity. The Diabetic patient is subject to the same oral lesions as the non-diabetic patient, but the lesions vary with the type, severity and duration of the diabetes, the type of patient, his age, race and genic history. The explanation has been advanced that the uncontrolled diabetic is more susceptible to infection, and once incubated, the infection spreads rapidly and extensively. Pollack et al (1947)

Massler (1949) has compared the oral flora of the diabetic and the non-diabetic, and found a predominance of staphylococcus Aureus and beta hemolytic streptococcus in the diabetic.

The term Diabetic Stomatitis has been given to that symptom complex present in the diabetic which, according to Rudy and Cohen (1938) is due to the high sugar content of the tissues and dehydration; consequently tissue resistance is low.

Rutledge (1940) includes hypercholesteraemia as a factor in lowered tissue resistance; the appearance of Lipoids in the paradental tissues is a direct result of a diabetic lipaemia. The metabolic upset allows the lipoids to spill over into the blood only to be ingested by the Reticulo Endothelial cells which form granulomatous masses in bone causing localised osteoporosis. The mucous membranes appear to be deeper in colour. The gums are swollen and highly inflamed with a colour range between salmon, brick, old rose or bright red. The gingiva may be detached from the teeth. A chronic gingivitis is usually present. Comroe et al (1954), Rutledge (1940), Hirschfield (1934).

Hirschfield goes on to state that the clinical appearance in pregnancy may resemble this condition, although the growth, which is often multiple, tends to be more pedunculated than sessile and is less likely to force the gum away from the tooth. The gingival margin may appear frayed and cyanotic.

Periodontal disturbances are always associated with the diabetic. Niles (1932), Rutledge (1940), Pollack et al (1947), Comroe et al (1954), Thoma (1954), Williams (1928), and they

have been classified into two types of conditions, namely, in the Acute diabetic and in controlled diabetes.

Although the periodontitis associated with the Diabetic state is possibly only an extension of the existing chronic marginal gingivitis, Pollack et al (1947) suggest that it may be due to a negative Nitrogen balance associated with the uncontrolled diabetic with a subsequent drain on the body proteins. Since calcium metabolism is dependent on protein metabolism, when deprived of protein the delicate balance between bone resorption and formation is upset. A second point advanced is that a decreased resistance to infection may be due to a decrease in the immune bodies in the gamma globulins, which are a function of protein metabolism, and which in starvation and diabetes may disappear from the blood.

Gingival haemorrhage is common and loosening and eventual loss of teeth occurs. Comroe et al (1954)

Ziskin et al (1944) suggest that: "The underlying factor in producing gingival changes in the diabetic may be the nutritional and metabolic imbalance which the Insulin treatment or diet only partially corrects."

It was Brand, quoted by Rutledge (1940) who first described the diabetic breath as having a fruity odour. It has also been described as resembling "new hay" or "decaying apples" or "acetone."

The teeth of the diabetic are rapidly covered by a soft, yellow calculus, and Rutledge (1940) ascribes this to Acidosis and large calcium output in the saliva.

Niles (1932) recommends that a routine urine examination be carried out in all cases of alveolar resorption. Ziskin et al (1944) contradict earlier reports that endeavoured to correlate incidence of dental caries and diabetes mellitus, and state: there is no data to support the theory that diabetes mellitus through nutritional or other factors alters the caries incidence.

Glossodynia and Xerostomia are present in many cases and are said to be directly due to fluid loss, Thoma (1954). Pruritis is a general symptom of the condition, and, as is expected, it

is sometimes associated with the gingivae. Strean (1938) has described this as Pruritis Gingivae and states that no other cases have been reported in the literature. Three out of five of Streans' cases were Jews, which lends help to the theory that an ethnic distribution exists. Hirschfield (1934) has described single or multiple acute gingival abscesses associated with diabetic stomatitis. Comroe et al (1954) describe several other oral symptoms including cervical sensitivity and frequent occurrence of dry sockets. He describes the Diabetic tongue as enlarged, thick, fissured, raw and ham coloured.

DISEASES OF THE CARDIO VASCULAR SYSTEM.

Congenital defects, cardiac decompensation,
angina pectoris, polyarteritis.

DISEASES OF THE CARDIO-VASCULAR SYSTEM.

CONGENITAL DEFECTS.

The Tetralogy of Fallot consists of pulmonary stenosis, usually infundibular in location, dextraposition of the aorta so that it overrides and receives blood from both ventricles, a high ventricular septal defect and hypertrophy of right ventricle. Cecil and Loeb (1956).

The labial mucosa is very cyanosed and the buccal mucous membrane is a bluish red and somewhat wrinkled. The gingiva are dark bluish red and very inflamed. The floor of the mouth and the fauces are cyanotic and the dorsum of the tongue has a very bright bluish red appearance.

The fungiform and filiform papillae are raised and prominent on an oedematous surface, and the blood vessels on the inferior surface of the organ are prominent. The pulps of the maxillary incisor teeth are enlarged and funnel shaped. Kaner, Losch and Green (1949).

CARDIAC DECOMPENSATION.

Varices of the lingual veins are seen as bluish distensions about 5mm. in length, and are considered by Schaffer (1951-52) to be asymptomatic. Sublingual varices, however, (phlebectasia linguae) are frequently seen accompanying heart disease (as a result of cyanosis and increased venous pressure). Colby (1956) states that in older individuals they are not uncommon and probably have no clinical significance.

Burket (1957) (quoting Birkowitz) describes a clinical test claimed to be an early sign of cardiac decompensation. The patient places the tip of the tongue without pressure on the palatal surface of the upper incisors; a marked distension of the veins on the under surface of the tongue is indicative of cardiac decompensation. The tongue also becomes markedly cyanotic when it is protruded from the mouth and held in a dependent position.

ANGINA PECTORIS.

Angina Pectoris is a syndrome consisting of paroxysmal substernal or precordial pain or discomfort of short duration, frequently radiating to the shoulders and inner aspects of the arms, usually precipitated by exertion, emotion or other states in which the work of the heart is increased. Cecil & Loeb (1956).

The pain of angina pectoris is occasionally referred to the jaws and teeth. It may be a throbbing, aching, localised pain involving the left side of the mandible. There is no apparent dental cause, and this dental type pain may dominate the picture. Stribling and Hurst (1957).

Anginal jaw pain is characterised by its extreme severity, its onset following exertion and its disappearance with rest. Burket (1957).

The mechanism is referred to by Wyke (1958).

POLYARTERITISPeriarteritis Nodosa, Polyarteritis Nodosa or
Necrotising Angitis.

A disease named by Kussmaul and Maier in 1886 (periarteritis nodosa), and characterised by visible nodules along the course of the medium-sized muscular arteries, Cecil and Loeb (1956), by systemic symptoms of infection, and with added regional symptoms depending on the vessels involved. Gottsegen and Gorlin (1949). Although its aetiology is obscure, infectious organisms, bacterial toxins, a specific virus or an allergic mechanism, have been proposed as possible causes.

The arterial lesions consist of necrosis, fibrinoid alteration and hyalinization of the media, with a marked parivascular infiltration. Intimal proliferations lead to thrombosis and arterial obstruction with infarct formation. The characteristic nodules are produced by repair and fibrosis of the adventitial lesions. Cecil and Loeb (1956).

The case of Gottsegen and Gorlin (1949) had involvement of the blood vessels of the tongue. Morgan and O'Neil (1956) describe Wegener's granulomatosis as a form of polyarteritis nodosa. A fatal febrile illness of several weeks' duration, characterised by ulcerative lesions of the nasopharynx. A case they reported had undergone exodontia, and the sockets had failed to heal. Hyperplastic gingival tissue formed, and there had been resorption of the alveolar and inter-septal bone. Oral involvement in this condition is rare, although secondary lesions in the palate (palatal perforation and swelling) and on the gingiva occurs as a sequel to the nasopharyngeal destruction. Massive ulceration, destroying the upper lip, has been recorded. An ulcerative stomatitis may accompany the nasal lesions, and terminal oral haemorrhage occurs.

THE REPRODUCTIVE SYSTEM

Menstruation.

Menopause.

Pregnancy.

Masturbation.

MENSTRUATION AND THE MENOPAUSE

The reproductive life of the human female is controlled by the sex hormones. It commences with the first menstrual period (Menarche) during puberty and usually ends with the last menstrual cycle (Menopause) during the climacteric. The symptoms manifested by the climacteric female have a complex basis of metabolic, structural and emotional changes. Massler (1951).

The oral manifestations associated with menstruation include hyperaemia, pain, swelling and haemorrhage from the gingival margins and papillae, herpes labialis and oral aphthous lesions.

Hyperaemia of the dental pulp may give rise to periodic pain involving sound or filled teeth and is occasionally referred to in the literature as "menstrual toothache". Swelling of the salivary glands, which according to Racine and quoted by Burket (1957) is due to a deficiency of the corpus luteum and is successfully treated with progesterone. Knox (1957). Mucosal ulcerations of periodic nature have been noted.

Thoma (1954) refers to this group of symptoms as stomatitis dysmenorrhoea and describes a case of gingivitis intermenstrualis with gingival changes related to the cycle.

Cyclic neutropenia with oral ulcerative lesions may be associated with the menstrual cycle, Page & Good (1957).

Oral manifestations may appear during the climacteric and have been considered by Massler (1951).

Stomatopyrosis, Glossodynia and Parageusia were noted in some cases. Cacogeusia and sexual maladjustment may occur together and are discussed by Hart (1938). Oral Cancerophobia is present in some cases and is of an intense nature. An atrophic glossitis of the Vitamin B deficiency type was noted by Massler (1951).

Thoma (1954) describes a chronic desquamative gingivitis accompanied by the formation of bullae which quickly burst.

The pathological change is considered to be one due to depolymerising enzymes which affect the ground substance and cementing substance of the epithelium. Treatment is symptomatic.

Sjogrens syndrome may be present, Morgan & Raven (1952), Allington (1950), Cecil & Loeb (1956), Burket (1957).

Chronic canker sores have been seen in patients experiencing the early climacteric.

Massler (1951) suggests that a temporary endocrine imbalance may be responsible for many of the symptoms manifested by the climacteric patient.

Trott (1957) considers the presence and distribution of Glycogen in the gingiva and concludes that its deposition is not influenced by Oestrogen activity.

Treatment is essentially symptomatic and is limited to the most distressing symptoms. Sympathy, genuine interest and understanding should, however, be the keynote. Sedation may be of value, Williamson (1957) in highly strung patients. Exercise graduated to the age and fitness may be more valuable, Burn (1957). Endocrine substitution therapy should only be used in severe cases. The use of hormones is considered by Knox (1957). Vitamin therapy is sometimes indicated, Massler (1951).

PREGNANCY

Gingival changes occur in the pregnant woman most frequently in the second trimester, in those individuals with poor oral hygiene. In a recent paper Gridley (1954) considers Ziskin's classification of oral changes during pregnancy.

It is thought that the first three conditions of Ziskin's classification, namely gingival haemorrhage, mildly puffed gingiva and raspberry-red gingiva cannot be attributed with certainty to the pregnancy. However, the hypertrophic gingivitis of pregnancy and the pregnancy tumour are thought to be the direct result of the pregnancy.

In regard to this, Maier (1948) considers that the gingivitis of pregnancy is an inflammatory condition of local irritative origin modified by the systemic disturbance; the pregnancy can be considered to be a conditioning factor which influences the character of the inflammatory reaction; it is not to be considered to be the primary aetiological factor.

Gorvy's (1954) theory that the embryo activates all the tissues of the gravid woman, which are then influenced by the endocrine glands, is attractive and plausible but rather complex.

Hilming (1952) considers Avitaminosis C to be a primary aetiological factor, but hormonal and local conditions should not be ignored as they occupy a significant place in the development of gingivitis gravidarum.

Pregnancy gingivitis is characterised by a peculiar raspberry colour of the marginal gingiva and ready bleeding. The interdental papillae are especially involved by the hypertrophic changes. Unless there is fusospirillaetal infection present ulceration is uncommon and pain is not a prominent symptom. Burket (1957).

After parturition specific pregnancy gingivitis (47% of cases) is ameliorated. However, non-specific pregnancy gingivitis (53% of cases) is not. Hilming (1952).

Maier & Orban (1949) consider that the histopathological picture seen is not exclusive to the gingivitis of pregnancy.

The relationship of dental health and pregnancy has been dealt with briefly by Rogers and Keen (1954).

A case of a primigravida with no evidence of a blood dyscrasia, who suffered two severe haemorrhagic episodes (oral) just prior to labour is discussed by Johansen (1955) who considers that the spontaneous haemorrhage was due to an alteration in the hormone level in the blood.

The caries rate is not significantly increased, if at all, and experimental and clinical evidence indicates that there is a normal increment of new cavities during the pregnancy. A lack of oral hygiene is the primary cause, the slight increase of salivary acidity and the possible effects of the vomiting of pregnancy are not very significant. Burket (1957).

The pregnancy tumour is another form of hyperplasia which appears in connection with gingivitis gravidarum. These pedunculated tumours arise from the interdental papillae and are thought to grow under hormonal stimulation. Clinically they appear as spherical bodies, generally pedunculated and with a deep red or bluish tinge. They are extremely prone to haemorrhage and may occur singly or multiply anywhere in the oral cavity. They recur following removal during pregnancy but sometimes recede spontaneously after parturition. Thoma (1954).

They have a varied histologic composition but Fabe (1954) classifies them as haemangiomas.

A pregnancy tumour removed surgically and with no recurrence is described by Grich (1950).

Thoma (1952) reports a case of an epileptic woman, with a naevus on the side of the face, treated for multiple pregnancy tumours. This is a case of the Sturge-Kalisher-Weber Syndrome, first described by W.A. Sturge in 1879, by Kalisher in 1901 and F. Parkes Weber in 1922.

It is thought that the vascular dysplasia of the gingival tissues is part of the syndrome. Weber's hypothesis was that the disease is present but not manifest until aroused by some agency, (in this case hormonal).

MASTURBATION.

A case of cheek chewing producing trauma along the interdental line of the buccal mucosa is described by Winter (1949). During the excitement of his experience the patient was unaware that he was chewing his cheeks. Psychiatric consultation revealed the habit which the boy was able to break and his cheeks ceased to be traumatised.

TROPICAL DISEASES.

May have associated oral lesions, either due to the aetiologic agent of the disease itself; to a constitutional disorder or to an accompanying nutritional deficiency.

Mazumder (1953)

LEISHMANIASIS.

The diseases caused by parasites of the genus *Leishmania* include Kala Azar, Oriental Sore and Espundia, and although they are usually described individually, it is thought that they may represent stages in the course of infection with the same parasite. Cecil & Loeb (1956)

Kala Azar (visceral Leishmaniasis) is an infective disease characterised by chronicity, irregular fever, splenomegaly; often hepatomegaly, and the presence of *Leishmania donovani* in these and other organs. Manson-Bahr (1954)

The oral lesions are usually those of gross nutritional deficiency, including avitaminosis B complex and C. A haemorrhagic tendency is associated with a lowered plasma prothrombin concentration. Mazumder (1953)

A post Kala Azar dermal leishmaniasis of the nodular type may develop on the tongue or lips, and is characterised by a chronic granulomatous lesion which does not usually ulcerate. Mazumder (1953)

Soft painless lingual nodules, non-ulcerated and with no pigmentary disturbances are described by Ghosh Dastidar (1939). This is a rare manifestation of post Kala Azar dermal leishmaniasis. Noma is the classic complication of Kala Azar. Mazumder (1953)

Oriental Sore. (Oriental Cutaneous Leishmaniasis)

is a specific granuloma of the skin and occasionally of the mucous membrane of the lower lip and tongue.

Leishmania tropica is the causal agent and the earliest lesion it produces is a red papule, which after scaling and crusting over generally breaks down into a slowly extending and very indolent ulcer. Healing is prolonged and a depressed scar is left. Manson-Bahr (1954)

American Mucocutaneous Leishmaniasis.

Espundia is the chancrous form of the oriental sore. It is a most intractable ulcer which breaks out in the tongue and buccal and nasal cavities, destroying and obstructing them. Balendra (1957). Ulcerating granuloma may form in the mucous membranes of the mouth and pharynx. Mazumder (1953)

MALARIA

is an infectious febrile disease produced by several species of protozoa belonging to the single genus Plasmodium. It is transferred naturally from host to host only by the bite of an infected anopheline mosquito. Cecil & Loeb (1956).

There are no characteristic oral lesions, although febrile herpes labialis is not an uncommon accompaniment of benign tertian malaria. Mazumder (1953).

Haemorrhagic gingivitis has been reported in children 5-15 years of age, in cases, all of which terminated fatally. In adults there may be a chronic gingivitis which is accentuated by the use of the antimalarial drugs. In some cases chronic fibrous gingivitis, with hard leathery gums, has been seen in the absence of calculus.

The mucosa is frequently red and hypersensitive, but pale and whitish with pronounced anaemia. A sore and painful tongue becomes whitish and indurated when the anaemia is severe.

Balendra (1957).

PLAGUE

is a specific, inoculable and otherwise communicable epidemic disease, common to man and many of the lower animals. It is caused by *Pasteurella Pestis* and is communicated by certain fleas. Manson-Bahr (1954)

Although it is customary to consider three types, bubonic, pneumonic and septicaemic, the disease may be considered fundamentally as a single entity. Cecil & Loeb (1956)

In the bubonic type the tongue is swollen and covered by a thick creamy fur which rapidly dries, soon to become brown or almost black. Sordes form on the teeth and about the lips and the nostrils. Manson-Bahr (1954). Bubo may form in the tonsil.

The septicaemic type is characterised by haemorrhage from the gums and the buccal mucous membranes. Mazumder (1953)

YAWS - Framboesia

is a contagious, inoculable disease characterised by a primary sore and an indefinite incubation period followed by fever, rheumatic like pains and papules. Manson-Bahr (1954)

It is caused by a spirochaetal organism *Treponema Pertenuis*. Cecil & Loeb (1956)

The primary sore forms anywhere on the body and is followed by one or more crops of multiple papillomatous lesions. Gerry et al (1952)

The "mother yaw" is not painful unless firmly pressed, and becomes covered with yellow secretion or a scab which is blood stained, granular and covered with encrusted dirt. Deprived of its crust the swelling is smooth and red and oozes yellow serum containing the spirochate. Intense pruritis may be present.

In the tertiary stage the ulceration and destruction may continue with accompanying cicatricial contraction. Manson-Bahr (1954)

A papule may form at the angle of the mouth, Manson-Bahr (1954), Balendra (1957), and can be as large as 3 cm. in diameter, with a desquamating surface resembling the squamous syphilide. Mazumda (1953). Palatal perforation may occur rapidly. Balendra (1957)

Gangosa is a less common but spectacular form of tertiary yaws in which the affected mucosa becomes honeycombed; a destructive ulcer forms and is surrounded by markedly hyperaemic tissues. Mazumder (1953)

Bejel is a non-venereal spirochaetal infection characterised by lesions resembling syphilis. Mazumder (1953)

Drinking vessels often constitute the vehicle of transmission and initial lesions are particularly common about the lips and within the oral cavity. Cecil & Loeb (1956)

DENGUE FEVER

is an acute, specific febrile disease caused by a filterable virus and transmitted by mosquitoes of the genus *Aedes*.

A transient pale pink macular rash is often seen at the onset. Cecil & Loeb (1956)

The mucous membranes of the mouth and pharynx may show an erythema during the first two days of the illness.

Mazumder (1953)

BARTONELLOSIS

is a specific anthropoid-borne infection caused by a minute rickettsia-like micro organism and characterised clinically by an acute, febrile, anaemic stage (oroya fever) followed several weeks later by a nodular cutaneous eruption (verruca peruviana). Cecil & Loeb (1956)

The first stage is accompanied by haemorrhagic spots and bleeding from the gums and the second by the appearance of miliary and nodular eruptions on the oral mucous membranes which may give rise to dangerous bleeding. Mazumder (1953)

Manson-Bahr (1954) describes the development of pink macules on the oral mucosa, which gradually darken and become nodular.

HOOKWORM DISEASE

is a clinical syndrome caused by infection with *Ancylostoma duo denale* or *Necator Americanus*. Cecil & Loeb (1956)

The worm attaches itself to the intestinal (occasionally gastric) mucous membrane by means of powerful buccal armature, and obtains its nourishment from the blood of the host. It frequently shifts its hold and the abandoned sites ooze for a short interval. Balendra (1957)

It gives no oral lesions itself but when associated with severe anaemia it causes characteristic lingual involvement. The tongue becomes large, pale and flabby, and has a matt surface, Mazumder (1953). (a description which is not in agreement with that of another author, considered infra.)

In the early stages the mucosa and tongue are pale, the teeth are yellowish and there is a macroglossia.

As the anaemia becomes more marked in the advanced stages, the oral mucosa becomes white and leathery, the gums pale and parched and the teeth loose and yellowish. The tongue is whitish and enlarged.

The anaemia is severe in the very advanced stages and the mucosa has a thin white parchment-like consistency and touch. There is no haemorrhage on pressure. The enlarged white leathery tongue is characteristic of hookworm infection. Balendra (1957)

BANCROFT'S FILARIASIS

is a filarial infection produced by a delicate threadlike worm *Wuchereria Bancrofti*, which lives in lymph vessels and lymphoid tissues. Cecil & Loeb (1956)

The Elephantoid condition may involve either the lips or the tongue. Mazumder (1953)

TRICHINOSIS

is an infestation of striated muscle by the larvae of the round worm *Trichinella Spiralis*, which is transmitted by inadequately cooked food. Cecil & Loeb (1956)

The tongue may be site of formation of an active cyst, with the production of both swelling and pain. Mazumder (1953)

Oedema of the soft palate, the pharynx and muscles of mastication is common. Burket (1957).

GONGYLONEMA PULCHRUM

Nematode infestation of the submucous tissue of the oral cavity has been described by Burrill et al (1957) and Mazumder (1953). The worms may involve the lips, gums, palate, buccal mucous membrane or tonsil, and feel like rough threadlike spots beneath the mucosa.

Burrill's case had a history of recurrent oral ulcers of 5 years' duration.

A case of oral Myiasis (*Chrysomya Bezziana*) is reported by Grennan. (1946)

CESTODIASIS (Tape Worm Infection)

Of thirty or more distinct species of tape worm producing intestinal infection in man, only 6 are common as human parasites. The beef tapeworm, *Taenia Saginata* is one of the six. Cecil & Loeb (1956)

A case presenting with oral symptoms is described by Mallett (1957). The oral mucosa was red, oedematous and showed small ulcerations. The breath was foetid and sub-maxillary lymphadenopathy was present. The oral symptoms subsided after successful treatment with antihelminthics.

DRACUNCULOSIS

is produced by *Dracunculus Medinensis* (the dragon worm).

The gravid female worm migrates to the surface to discharge her larvae and it is at this stage that the tongue and the angle of the mandible have reportedly been involved.

Mazumder (1953)

DISEASES OF THE SKIN

Erythema Exudativum Multiforme; Leukoplakia; Lichen Planus;
Lupus Erythematosus; Erythema Nodosum; Neurofibromatosis;
Pemphigus; Psoriasis; Scleroderma; Epidermolysis bullosa;
Lichen Sclerosus et Atrophicus; Pityriasis Rosea.

The skin is an organ with special characteristics, and although the diseases which affect it are peculiar to itself, they may reflect underlying conditions. Curtis & Fliegelman (1954)

Since the oral mucosa and skin have a common embryologic origin, it is not surprising that changes in the oral mucosa are frequently observed in diseases of the skin. These mucosal lesions are morphologically similar but clinically dissimilar to the cutaneous lesions, and when the latter are present the diagnosis of the former is not always difficult. However, the oral climate is such that the presence of isolated lesions of the oral mucosa renders diagnosis more difficult and frequently impossible.

Burket (1957)

ERYTHEMA EXUDATIVUM MULTIFORME

This term serves as a collective name for a disease process of obscure aetiology. Although allergy to food and drugs, and hypersensitiveness to bacterial products, appears significant in some cases, Cecil & Loeb (1956), Thoma (1954), a viral cause has been suggested, and viral-type bodies demonstrated by Newman (1956).

Hebra in 1866, Kwapis (1957), first distinguished Erythema Exudativum Multiforme from the heterogeneous erythemas, Moodie (1950), and nowadays a major and a minor form of the disease are recognised. The minor form is accompanied by mild symptoms, but the major is often very severe. According to the distribution of the mucous and the cutaneous lesions and the involvement of the body orifices, the disease has been described and named by several clinicians. Stevens and Johnson in 1922; Behcet in 1937 and Reiter in 1916. There has been comparative consideration made by Robinson & McCrumb (1950).

The onset is sudden and the disease is ushered in by mild to severe respiratory symptoms. Because of the consistency of occurrence of these respiratory symptoms, it has been suggested that the disease be called the "mucosal respiratory syndrome."

The cutaneous lesions are frequently bilateral and take the form of target-like macules or bullae affecting the extremities, the neck and the face. Jacobus (1949)

Jones (1947) reports that the oral mucosa is involved in 6 of 9 cases. Lighterman (1958) reported a case that was limited to the oral cavity.

Burket (1957) quotes other authorities, that oral involvement occurs between 80% and 90% of cases.

Erythema Exudativum Multiforme minor shows a seasonal variance and a predilection for young males. The onset is sudden and accompanied by malaise, sore throat, rheumatic pains, severe pruritis of the hands, profound dehydration and oro-mucosal and cutaneous lesions.

In the mouth vesicles filled with a clear fluid form but are readily broken, leaving a raw, painful ulcer. A bloody crust or a slimy grey white membrane may form and become infected. There is mild to severe fetor and, despite the patient's dehydrated state, intense sialorrhoea.

Stevens-Johnson Syndrome

was first described by Stevens and Johnson in 1922. This syndrome is a particular manifestation of Erythema Exudativum Multiforme major in which the oral and the cutaneous lesions are accompanied by ocular involvement. A catarrhal, membranous or purulent conjunctivitis with subsequent corneal ulceration and possibly panophthalmitis may occur without complication, or as in some cases with permanent loss of vision. Thoma (1954)

In the oral cavity bullae form and appear filled with clear fluid. They rarely remain intact for any length of time, and on bursting form a painful ulcer with crusting and membrane formation. The lips, tongue, palate, cheek and gingiva may be involved and lesions which were initially discrete, coalesce and large areas are involved. There is the picture of a severe membranous stomatitis. Shira (1957), Abstract (1947)

The oral lesions may be so massive that speech is impossible Lozano & Mante (1955).

The lips are commonly involved and have a crusted and bloody appearance up to the vermillion border. The tongue may show well defined indentations along the lateral margins. Burket (1957)

The combination of oral and ocular lesions is often diagnostically significant, and early recognition of the disease aids the ophthalmologist. Theodore (1952)

Behçets Syndrome.

First presented by Adamantides of Athens in 1931. It was not until 1937 that Behçet (a Turk) published his paper on the oral-ocular-genital syndrome which now bears his name. Fraser-Moodie (1953). It is a particular form of erythema exudativum

multiforme seen in males at about the third decade. There is considerable cyclical variation in the instigation and exacerbation of symptoms. The three signs of the "triple symptom complex" will not necessarily be present together, and it may take some years before the three are manifest. The ocular lesions dominate often, and may precede, coincide with or follow the oral and genital lesions. Thomas (1947) (In his case aphthous ulcers were seen at 9 years, ocular at 12 and genital at 18 years.)

Fraser Moodie (1953) suggests that acute vulvular ulcer and periadenitis mucosa necrotica recurrens are possibly the same type. Thomas (1947) considers that the lesions of periadenitis mucosa recurrens and Bechets syndrome look the same. The ocular lesions may be very severe.

From an aetiologic viewpoint it is interesting but not conclusive that the bacillus of Grassus has frequently been isolated from the ulcers. The connection with the Mediterranean may also be significant.

Oral lesions may affect any portion of the mouth, and are acutely painful. A grey exudate frequently forms on the base of the ulcer, which is surrounded by a red halo. The ulcers may be discrete or confluent, in which case large necrotic areas with raised indurated edges form and heal with the formation of thick raised scars. Fraser Moodie (1953)

O'Donnell (1947) reports a case of a Greek patient so affected who also had a geographical tongue.

The lips, tongue, buccal mucosa, hard and soft palate and tonsils may be involved. Theodore (1952)

Reiters Syndrome

is a self-limiting disease, originally reported by Reiter in 1916, thought to be of viral or allergic aetiology, but which has been noted as a complication of bacillary dysentery. Cases are described by Wilcox et al (1947) and Baxter (1946).

The disease is manifest by the triad of Arthritis, Urinary tract involvement (especially Urethritis) and ocular lesions. The oral lesions are rare and generally only seen in a well-developed case. They take the form of an erythematous area with superficial ulceration, Stones (1954), which may be covered by haemorrhagic crusts. Fraser Moodie (1953)

The treatment of the oral lesions include the maintenance of oral hygiene; the application of anaesthetic ointments or troches; soft and bland foods; a mild mouth-wash and possibly an ointment containing cortisone. Burket (1957), Thoma (1954)

The general treatment includes the use of the Antihistamines Chippis (1951), Cortisone and the Antibiotics.

Fluids should be forced and polyvitamins administered. Cecil & Loeb (1956), Burket (1957), Thoma (1954).

LICHEN PLANUS

is an inflammatory dermatosis characterised by a papular reaction on the skin, accompanied by pruritis and manifestations on the oral and other mucous membranes. The cutaneous areas involved include the flexor aspects of the extremities, the neck, trunk and genitals. The mucosal lesions may occur on their own or in combination with the cutaneous.

The aetiology is obscure although it is thought to be related to psychosomatic factors; a history of emotional trauma is not unusual. There is no apparent age or sex disposition to the disease, although the individual seeking treatment is often in a position of responsibility, tends to worry excessively and may be nervous and irritable.

Darling & Crabb (1954) suggest that there may be bacterial or toxæmic factors concerned in its aetiology. In a recent paper Cooke (1954) has described the basic lesion which consists of a pearly white papule on an erythematous mucosa. These papules were discrete or confluent, and tended to form a pattern of which he has described eight types. Any area of the mouth may be involved, the buccal mucosa may be simultaneously involved, often in the buccal sulcus, in the region of the third molar.

The lips may be involved but the dorsum and lateral margins of the tongue are by far the most commonly involved areas, although the tip is usually spared. Although the hard palate is occasionally involved, the soft palate is rarely so.

The floor of the mouth is sometimes involved by extension of the lingual lesions.

A linear pattern of elevated pearly white lines on an erythematous base is often seen. A discrete papular pattern involves the cheeks and tongue, and a confluent papular pattern that has the appearance of tufts of cotton wool resting between the papillae involves the tongue.

The cheek is involved sometimes by the reticular pattern which has a characteristic lace-like pattern, but which may be very coarse and resemble the confluent papular pattern. The

papules may be arranged in circles enclosing an erythematous centre, and this has been described as the annular pattern. It has been seen in combination with discrete papules. Deep brown pigmented papules and plaques on the posterior aspects of both cheeks constitute the pigmented pattern. The centres of these areas may be white and keratinised. A flaccid blister, filled with blood-stained fluid, may form but eventually bursts, leaving a shallow erosion.

The mucosa appears dry, shiny and atrophic, with a white marginal line in the atrophic or erosive form. A shallow ulceration with an adherent grey slough may accompany this atrophy. A tongue affected this way looks smooth and glazed with indolent elongated and ragged ulcers on its sides.

Goldman et al (1951) state that the lesions may be very tender to functional stimuli and exhibit some degree of pyrosis. The erosive phase is rarely seen in the mouth.

Goldberg (1946) reports the high degree of incidence of lichenoid lesions in troops in the South Pacific and Allied areas during World War II. The emotional factors are significant in these areas where isolation and a bad climate were only two of many contributing conditions. Nervous exhaustion or an emotional disturbance frequently antedates or exists with Lichen Planus. Cawley & Kerr (1952).

Darling & Crabb (1955) describe erosive lesions in troops in tropical areas and notes that there is an associated photo sensitivity with Lichen Planus. They state that some authorities include Atabrine (Mepacrine) in the list of aetiologic or contributory agents.

"Wickham's lines" or "striae" is the term given to the lace-like pattern surrounded by inflammatory reaction. Cawley & Kerr (1952), Thoma (1954), and apparently coincides with Cooke's reticular pattern.

Gingival involvement is not common according to Thoma (1954) but the lesions occasionally occur, and when they do it is as a circinate arrangement.

Bernier (1949), however, classes gingival lesions as the nodular type.

Only rarely does a malignant change occur. Cawley & Kerr (1952).

Cooke (1954) considers that pruritis does not accompany the oral lesions, but Cawley & Kerr (1952) say that in some cases it does. Shira (1957) reports a mild pyrosis in some cases.

Ziskin & Silvers (1945) report the simultaneous occurrence of desquamative gingivitis and lichen planus. The latter responded to Vitamin A therapy and the former was aided by an Oestrogen ointment.

Oral hygiene must be maintained and occlusal trauma corrected. A bland agent may be applied to the lesions. Possibly more important is good food, reassurance, sunshine and freedom from worry.

LUPUS ERYTHEMATOSIS

A disease of tuberculous or streptococcal origin, first described by Kaposi in 1872. Svanborg & Solvell (1957). Poor peripheral circulation and endocrine factors may also be significant.

Gold (1951) hypothesised that Lupus Erythematosus, Erythema Nodosum and Erythema Induratum are only variants, and that conditioning factors determine the morphological response.

Lite (1953) considers that the oral cavity is involved in 15 per cent of cases. The lips may become affected occasionally as a result of extension of a cutaneous lesion. Gold (1951)

Burket (1957) considers that oral lesions are most frequently found in the discoid type of the disease, after cutaneous lesions have appeared. The tongue, palate, buccal mucosa, lips and gingiva may be involved. At the site, the epithelium is erythematous, atrophic and quite painful. Well developed lesions are ulcerated and surrounded by numerous whitish pin-head areas. Small dilated and radially arranged blood vessels are seen at the borders. On healing, thin scar tissue replaces the affected parts.

Lite (1953) describes gingival manifestations which appeared prior to the cutaneous lesions. The gingiva were oedematous, ulcerated and eroded. Other oral lesions are irregular, denuded ulcers with an indurated central area and whitish periphery. The epithelium tends to slough. A case with palatal lesions is described by Spies et al (1955).

Although the finding of the L.E. cell in the bone marrow or blood is diagnostically specific, they are not invariably demonstrable at all stages of the disease. Cecil & Loeb (1956)

Kurnick (1957) describes the false positive L.E. cell noted in cases of drug hypersensitivity and Rheumatoid Arthritis.

No form of treatment is available although Cortisone and ACTH may be useful in arresting a usually fatal disease. Burket (1957); Cecil & Loeb (1956).

Erythema Nodosum

is associated in some cases with hypersensitiveness to drugs, to infectious agents and probably to other allergens. Unlike Erythema Exudativum Multiforme, it is limited to a few reddish or purplish, tender and deeply embedded lesions on the arms and legs, and occasionally on the face and oral mucosa. Cecil and Loeb (1956); Thoma (1954).

NEUROFIBROMATOSIS

was described by von Recklinghausen in 1882. It is an hereditary disease of unknown aetiology, characterised by the formation of multiple neurofibromata on cutaneous and mucosal surfaces. The disease has a low frequency of occurrence; it is seen in about 1 in 2000 patients of skin clinics. It affects males and females with the same frequency.

Oral lesions occur with more frequency than other writers think, and it is conceivable that small neurofibromata occur, but are not seen unless specifically searched for. Martin & Graves (1942).

Baden et al (1955) say that isolated or multiple tumours are found in the floor of the mouth, on the marginal gingiva, the buccal mucosa, the hard palate, Jacobs (1946), and the lips, Rappaport (1953). Involvement of the tongue may be unilateral or bilateral and produce a macroglossia, Rappaport (1953).

A case of mandibular involvement is reported by Borkenhagen and Vazirani (1956).

Secondary involvement of the cranial nerves produces a variety of signs and symptoms, including marginal deviation of the tongue. The tumour is considered to spring from the connective tissue sheath of nerves; although all the elements composing the nerve are involved to some degree.

Although the gingival tissues look vascular and spongy, they are firm and fibrous to touch. Minute fibromata-like growths may involve the gingival papillae, Rappaport (1953), and the teeth have been reportedly porous, another possible manifestation of the disease.

Jacobs (1946) reports that cafe au lait spots may precede the formation of the tumours.

PEMPHIGUS

is an acute or chronic disease affecting the skin and orificial mucous membranes. It is characterised by the formation of bullae, which in 54 per cent of cases involve the oral mucosa. Lever (1942). In 25 per cent of cases these oral lesions are the first signs of the disease.

Bernier (1951) cautions that it is not possible to diagnose Pemphigus by its oral lesions, which must be differentiated from Erythema Exudativum Multiforme, Stomatitis Medicamentosa, contact allergy and sometimes Vincent's infection.

It is possible that early oral lesions may be overlooked by the clinician not completely conditioned to a searching oral examination. A higher percentage of oral lesions is reported when the examiner has been trained in clinical oral pathology. Stern (1949).

The disease usually occurs in individuals over 30 years of age, although this is by no means the rule. There is an apparent ethnic difference in its occurrence for the incidence in Jews is higher than in other races. Costello et al (1957). The disease has an insidious onset, there is prodromal dryness and dysphagia with slight pain on taking hot or spicy foods. A few small and irregularly distributed blebs appear on the skin or mucosal surfaces, and rapidly grow. Although filled initially with a clear straw-coloured fluid, this becomes turbid and purulent with infection, or blood-streaked. Sagall (1936).

There is considerable discomfort, pain and pruritis, Spies et al (1955), associated with the lesions.

Mechanical injury causes early rupture of the vesicle and formation of a superficial ulceration with the walls of the bleb curling and clinging to the margins of the ulcer. The "curled-up egg membrane" is a characteristic sign of the bullous lesion. Stern (1949). In the early phases the base is raw, but later as an exudate forms and dries, crusting occurs and frequently secondary infection. The area becomes a deeper red colour and an areola forms.

The odour from the ulceration may be quite obnoxious and the patient suffers considerable pain. As the first blebs heal another crop forms.

There are several clinical varieties, of which pemphigus vulgaris is the most common and exhibits the most severe oral lesions. Intact bullae are rarely seen in the mouth, for the mucosal epithelium is readily broken by oral trauma and slides off the resultant erosions which enlarge by peripheral extension.

A white exudate covers the ulcer, which bleeds readily, The entire oral mucosa is involved in severe cases. The lips, the tongue and the gingiva are swollen, the sputum is tinged with blood and there is sialorrhoea, dysphagia and hoarseness.

Lever (1956) and Quinn (1948).

Clinically, pemphigus vegetans resemble acute pemphigus vulgaris except for the formation of vegetations on the cutaneous erosions. Oral involvement occurs in practically every case, although oral vegetations rarely form. When they do it is as soft, dark red granulations, most frequently along the vermillion border of the lips. Levin (1956) considers the condition in some detail.

The lesions of chronic pemphigus vulgaris rarely start in the mouth, and the bullae are quite large and tense and do not break easily. The erosions do not undergo peripheral extension and healing occurs quite readily. There might be slight sialorrhoea, but the sputum is not blood-stained and the mouth is only moderately tender.

Small, tense, moderately tender and intact blisters involve the oral mucosa in 81 per cent of cases of benign mucous membrane pemphigus (pemphigus conjunctivae). The erosions are clean and not involved by fibrinous or epithelial shreds. Although they heal well, scarring occurs, mainly about the soft palate, tonsillar region, tongue and buccal mucous membranes.

As yet no oral lesions have been reported in cases of pemphigus foliaceus and pemphigus erythematodes (Senear Usher type).

Walters et al (1952), Abstract (18) describe a case of acute

pemphigus associated with the butchering trade.

The pain suffered by the patient depends primarily on the site of the lesion. In the palate, for example, the tissues are tense and lesions painful. In the cheek they are less painful, however, because the structures have a looser texture. Cahn (1947)

A case with lesions involving the palate and gingiva is described by Schreiber (1955). Lesions affecting the tongue are described by Schaffer (1951-52)

Although its aetiology is obscure, Mariani of Italy considers that a viral septicaemia with ectodermal localisation may be causative. His evidence is based on microbiologic and serologic findings: the nuclei of epithelial cells closely resemble cellular changes characteristic of the chickenpox-zoster group of diseases. The histopathologic picture shows a special kind of intradermal and supra-basilar vesicle containing an almost pathognomonic type of degenerated epidermal cell (the Tzanck cell). Fluid accumulation between the basal layers of the epidermis and the lowermost cells of the spinosum marks the earliest event in pemphigus. Next the spinosum cells undergo acantholysis (loss of tonofibrils) and the intra-dermal bullae coalesce. The spinous cells (less their spines) are found in the vesicular fluid. Since the vesicles are also "cleavage vesicles", the superficial layer of skin may be detached by simple thumb pressure. (Nikolsky Sign) Lever (1956)

In presteroid days there was a 90 per cent mortality rate. Costello et al (1957). Nowadays this is 33 per cent, and although many patients are being maintained on steroids, there are many side actions associated with its use in prolonged high dosages. Blackburn (1955). In addition to steroids a high calorie, high protein diet is essential, Arnott (1957); blood transfusions may be necessary for the anaemia; good nursing care is a must. Oral hygiene is difficult to maintain in the patient with severe oral lesions, but useful palliative measures include the use of a warm 10 per cent sodium bicarbonate solution mouth-wash. Burket (1957). Anaesthetic troches or spray may be utilised prior to eating. Ruptured oral lesions can be treated with a 1 per

cent aqueous solution of crystal violet or 2 per cent aqueous solution of methylene blue. A non-irritant denture base (metal) is essential in reducing the incidence of new oral lesions as it avoids the micro trauma often caused by plastic denture bases. Arnott (1957) Dental prophylaxis should be carried out regularly and with care to avoid unnecessary trauma and haemorrhage. Burket (1957).

PSORIASIS

is a chronic disease characterised by persistent red, dry and rounded lesions covered by silver scales. The disease persists for years with remissions and exacerbations. Thoma (1954).

Norins & Yaffee (1957) claim that oral involvement is uncommon if not rare, and Levin (1954) considers that oral psoriasis is nothing more than leukoplakia in one of its stages. When oral lesions do occur, they do so usually only as an extension of the cutaneous ones.

The mucosal lesions are white, Norins & Yaffee (1957), or grey to yellowish white coloured plaques, round to oval and sharply demarcated. Goldman & Bloom (1951). The buccal mucosa is the most commonly affected oral surface, and may be studded with greyish white spots in places. A thick desquamating surface is denuded in places and coloured a deeper red. Becker & Ritchie (1930). Ring-shaped striae sometimes appear opposite the occlusal line, a short distance behind the angle of the mouth. Goldman & Bloom (1951), Usher (1933). The apparent absence of irritative factors would tend to eliminate leukoplakia.

The hard and soft palate may exhibit an erythema. Norins & Yaffee (1957)

Goldman & Bloom (1951) describe a scrotal tongue, beefy red in colour and enlarged. A papular eruption and a greyish white epithelial layer involved the surface. On scraping this layer a red surface was left. Papillary atrophy may accompany the glossitis, Norins & Yaffee (1957) and the pharyngeal and laryngeal mucosa may show a dusky erythema.

A moderately indurated and irregularly shaped grey white plaque covered the lip in the case described by Goldman & Bloom (1951). There was no ulceration, and healing following excision was satisfactory. This patient had suffered from cutaneous psoriasis for a number of years. Another case, one of psoriasis of the scalp, was accompanied by involvement of the floor of the mouth.

The histopathologic picture shows a simple inflammatory process with parakeratosis and microabscess formation in the epidermis. The corium is infiltrated by inflammatory cells and fibroblasts. Levin (1954).

There is no systemic treatment of value, and since the oral lesions are innocuous they require none.

SCLERODERMA - Dermatosclerosis, Progressive Systemic Sclerosis

is a disease involving the collagenous connective tissue, which may cause widespread symmetrical, leathery induration of the skin, followed by atrophy and pigmentation. The cutaneous lesions are merely the external manifestations of a systemic disease, and the muscles, bones, mucous membranes and internal organs may be involved by the same process. Cecil & Loeb (1956).

It is considered by some to be a disease of adaptation, but mental strain, drug intoxication, acute infection and over-exposure to sunlight and cold, Smith (1958), are other significant factors suggested. It appears most frequently in middle life, Cecil & Loeb (1956), but it may occur at any age. Smith (1958). Females are the most frequently affected.

Rosenthal (1948) describes two types; first the localised form, morphea, and secondly the generalised (diffuse) form, more serious and less common.

The initial complaint is one of restricted oral movement. The lips are retracted and thin, the buccal cul de sac is often obliterated and there is circumoral pallor and puckering. Smith (1958). The atrophied dermis readily suffers chronic ulceration. Function is greatly interfered with and oral hygiene is difficult to maintain.

The oral mucosa is pale and thin and tends to ulcerate, particularly in the region of the cheeks where increased pressure causes an irritatingly intimate contact between tooth and mucosa.

Pigmentation is reported as rare by some authors, although Rosenthal (1948) describes it. Pseudoankylosis may follow involvement of the masticatory muscles, and further contributes to the decreased opening. The tongue, soft palate and larynx may be involved and become stiff and difficult to move.

Rosenthal (1948).

The tongue is rigid since fibrous replacement limits its movements, and hard pigmented patches appear on its surface.

Schaffer (1951-52).

Krogh (1950) considers some of the dental manifestations, which include alveolar resorption, tilting, exfoliation and delayed eruption of teeth. Rosenthal (1948) reports loosening of the teeth following alveolar resorption.

Mitchell & Chaudhry (1957) and Smith (1958) report definite thickening of the periodontal membrane. This widening is at the expense of the lamina dura. The posterior teeth are more frequently affected than the anterior teeth. Gores (1957). Following exodontia, haemorrhage might be very severe as a result of the inability of the blood vessels to contract. Smith (1958).

Histopathologically, hypertrophy of the collagenous tissue of the skin and submucous tissues is the most important finding.

An early inflammatory exudate gradually disappears. Delicate collagen fibres become coarse and connective tissue bundles are enlarged and pressed together. The lumen of the blood vessels is reduced by an obliterating sclerosing of the intima. Smith (1958).

The treatment of any form of scleroderma is difficult and unsatisfactory. Cortisone and ACTH may be useful in the treatment of early scleroderma. Burket (1957).

EPIDERMOLYSIS BULLOSA

is an incapacitating hereditary disease characterised by the formation of bullous lesions on cutaneous and mucous surfaces in response to mild trauma. Bullae appear soon after birth, and it has been hypothesised that the absence of elastic tissue in the superficial layers of the dermis may be the cause.

Lesions form frequently on the buccal mucosa in the more severe form of the disease; the membrane of the bullae are thin, semi-transparent and filled with blood; the lesions heal with little scarring but eventually lead to a leukoplakia-like atrophy. McDaniel (1954).

Lichen Sclerosus et Atrophicus

was first described by Hallopean in 1887. There is great controversy as to whether it is related to Lichen Planus or localised Scleroderma, since, according to various authors, its oral lesions may resemble either (although Miller (1957) considers oral Scleroderma to be exceedingly rare).

The lesions affect the mouth, vulva, penis or anus, and are superficially ulcerated white papules which remain discrete or coalesce. The white surface becomes atrophic and wrinkled. Ravits & Welsh (1957).

Histologically there is usually a hyperkeratosis and an atrophic squamous mucous membrane.

Pityriasis Rosea

is an inflammatory dermatosis of unknown aetiology but seasonal incidence, occurring between the first and third decades and characterised by numerous yellowish, pinkish or reddish scaly macules over the body surface. Thoma (1954).

Guequierre & Wright (1941) have described 5 types of oral lesions. The first type affecting the buccal mucosa is a round erythematous macule; the second type is similar to the first but

shows greyish ruffled desquamation. The third type is an erythematous, segmented or incomplete annular lesion with raised borders, a clear centre and ruffled scaling or desquamation. The fourth type is an annular, punctate and haemorrhagic lesion, and the fifth type is a large erythematous plaque covering the entire palate.

A generalised catarrhal stomatitis with sialorrhoea may precede the mucosal lesions. Burket (1957)

Since the disease disappears spontaneously within a few weeks, Thoma (1954), no treatment is necessary.

APHTHOUS ULCERATION; EPILEPSY; LEUKOPLAKIA;
VINCENT'S INFECTION.

APHTHOUS ULCERATION - Acute aphthous stomatitis, vesicular stomatitis, maculofibrinous stomatitis, recurrent aphthous stomatitis, canker sore, dyspeptic ulcers, habitual solitary aphthous ulcer, Mikuliczs aphthae, periadenitis mucosa necrotica recurrens, ulcus neuroticum mucosae oris, ulcus necroticum mucosae oris, fragmentary Behcet symptom complex.

The condition was first described by Hippocrates (460-370 B.C.), but our lack of knowledge of the nature and variations of this disorder is indicated by the number of synonyms it has collected. Sircus, Church and Kelleher (1957).

In Europe the *Bacillus crassus* is regarded as the aetiologic agent, but a viral theory has been popular for some time, although allergy to food and hormones has been suggested.

Twenty per cent of the population suffers from recurrent aphthous ulceration of the mouth. There is a rhythmical periodicity of attacks, but a sharp difference between the sexes in the relative frequency of attacks. One in four of every female suffers from attacks every one to two months, although one in six males suffers an attack about once in four to five months.

Local trauma is also important in its aetiology. On the other hand aphthous ulcers (recurrent) may be associated with idiopathic steatorrhoea, ulcerative colitis, dyspepsia, proctocolitis, peptic ulcer and skin conditions.

There is a very significant relationship between recurrent aphthous ulceration and stress, especially when the disorder is severe. Marital maladjustment, sexual frigidity, the stresses of the social environment and financial worries may be important predisposing factors. An unfavourable early environment was found in 71 per cent of cases.

The ulcers are located on any surface; oral, vulval, urethral, vaginal, scrotal and ocular. The pharynx and fauces may be affected. No portion of the oral mucosa is immune, and the inner surface of the upper and lower lips, sulci, tongue and buccal mucosa may be involved. Sircus et al (1957).

There is a prodromal pruritis or pyrosis of the mucosa, which then becomes erythematous, after which a grey-white plaque is formed and lost. Within 24 hours the centre breaks down and a small ulcer forms which increases in size for three to six days. The ulcer is painful and very sensitive to tissue movement and irritants. The site of the ulcer determines its outline; in the sulci it tends to be more elongated than on a flat surface, where it is usually rounded. If the ulceration persists, it may become deeper with raised and rolled margins. Although cases lasting six weeks have been reported, healing usually commences in six days, and is complete within ten to fourteen days. Farmer (1958).

Histopathologically, we see that intraepithelial oedema produces vesicle formation in the stratum granulosum. The vesicle contains serum and degenerated epithelial cells. When fully developed there is disruption of the epithelium, with the formation of a whitish membrane containing cellular debris. There is round cell infiltration of the papillary layer of the corium. Marked infiltration of the small neurovascular system in the corium and subcorium may account for the hypersensitive condition of the lesion and neuritis. Thoma (1954).

A more severe form of the condition (chronic scarring aphthous ulcers) is described by Weichselbaum & Derbes (1957), and is associated with the formation of deep nodules, painful necrotic ulcers and scar formation, eventually causing microstomia.

Local applications of caustic materials recommended by most authors only tend to perpetuate the ulceration. (Bland solutions, however, like methylene blue, have some palliative value.) Thoma (1954).

Sircus et al (1957) state that topical application of Aureomycin appears to decrease the duration of the ulcers, although it does not change their number or frequency. They consider some of the other modes of therapy employed in the past.

The steroid drugs appear to have a beneficial effect in some cases. Weichselbaum & Derbes (1957).

The use of 9-Alpha Fluorohydrocortisone Acetate is suggested

by Wiesstien (1956).

General treatment consists of the diagnosis and correction of systemic disease. Thoma (1954).

Sircus et al (1957) have noted that suggestion sometimes yields astonishing results.

EPILEPSY

is the most important of the convulsive states, and although there are both genetic and acquired causes, the former is the more important.

Epilepsy is characterised by the epileptic seizure, which is usually preceded by the "aura" and followed by a coma. Excessive clonic and tonic contractions are the outstanding phenomena of Grand Mal. Petit Mal is usually characterised by short periods of loss of consciousness, or inability to proceed with a task. Cecil & Loeb (1956).

Extensive bruising and scarring of the facial areas, lips and tongue may be seen in the epileptic. Epileptics are particularly prone to nocturnal bruxism. Burket (1957).

The most dramatic oral lesions connected with the epileptic state are seen in those patients maintained on the anti-convulsant drug Diphenylhydantoin. This drug was introduced for the symptomatic treatment of epilepsy by Merritt & Putnam in 1938. Goodman & Gilman (1956).

Kimball suggested that the hyperplasia was the result of abnormal ascorbic acid metabolism, but Ziskin has proved this to be not so. Reader (1950).

Staple (1953) considers that the gingival reactions to Dilantin may be a manifestation of a fundamental metabolic upset involving the adrenal glands, and in a recent paper he discusses some of the side reactions associated with Dilantin therapy. He has concluded that there is no correlation between the severity of the oral reaction and the dose or duration of therapy.

Although the edentulous areas show no hyperplasia, Reader (1950), the tissues are much thicker than normal. Staple (1953).

The incidence of oral reactions varies from as low as 6% to as high as 62% in different series of reported cases. Goodman & Gilman (1956).

There is a constitutional tendency and individual sensitivity concerned in the production of the hyperplasia, although obviously local factors are very important and affect the colour, size and form of the basic hyperplasia.

The gingival margins, especially those about the anterior teeth, show painless thickenings with a dense and normal colour. When the entire gingivae are involved, the crowns of the teeth may be slowly covered. The interproximal gingiva may fill the space and begin to coalesce with the lingual, labial and buccal tissues. When the crown becomes completely covered, food impingement during mastication causes pain. The presence of calculus causes venous stasis, and the tissues become light blue, and later dusky blue, in appearance.

If no local treatment is instigated, bone loss occurs with consequent loosening and movement of the teeth.

Histopathologically, there is an extreme connective tissue proliferation with the rapid formation of collagen bundles, and an increase in the number and the size of the blood vessels and lymphatics. The keratin layer is unaltered, but there is a proliferative change in the basal layer, and a slight increase in the mitotic figures in the prickle cell layer. Whorls or pearls may be formed.

Although there is an absence of cellular infiltration in the presence of definite proliferative changes, in advanced lesions there may be a large leucocytic infiltration.

Treatment is aimed at the preservation of good oral hygiene. Surgical intervention should only be made when absolutely necessary.

In the presence of severe oral involvement, the clinician should consider the withdrawal of Dilantin, and its substitution (only if a satisfactory substitute can be found) by another anti-convulsant agent. Reader (1950).