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Occupational Diseases of Teeth

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Summary
Occupational diseases of the teeth have, in general, received scant attention. The chief cause of this is lack of awareness among occupational physicians. Exposure to various chemical substances is one of the causes of occupation-related dental disorders. Physical and biological factors also contribute. The combination of these factors plus poor dental hygiene aggravates the condition. The present article aims to focus the attention of occupational physicians towards this important problem.

Introduction

Teeth are affected in a number of occupations. In fact, dental manifestations may be the very first signs of an occupational disease and their early detection may help us in preventing such diseases. It thus becomes essential for dental surgeons, general practitioners and specially industrial physicians to acquire a good knowledge and experience of the dental manifestations of occupational diseases. This will help them to eliminate the chances of erroneous diagnosis and in screening cases of occupational origin to prevent further hazards.

Workers may develop disorders of teeth because of physical factors or exposure to chemical substances, organic or inorganic, specific to their occupation. Chemical substances are the principal causes of occupational diseases. With the considerable expansion of chemical industries producing many synthetic formulations there should be a vigilant check on the workers for any systemic or dental ill effects. Apart from the chemical agents, physical factors like radium, X-ray and biological factors produce ill effects on workers.

Dust

Dust of abrasive quality such as cement or sand, may collect on the occlusal surfaces of the teeth and produce generalized abrasion. Such a condition is usually found among cement and sand workers, grinders, stone cutters and miners etc. Peterson and Henmar[1] reported 100 per cent prevalence of dental abrasion among workers in the Danish granite industry. Similarly Enbom *et al.*[2] found a statistically significant higher degree of occlusive wear among miners than among white collar workers in Sweden.

Organic dusts

Bone, celluloid, sawdust, flour, tobacco etc. may produce staining of teeth, pigmentation of gingivae, generalized abrasion, calculus, gingivo-stomatitis and haemorrhage in workers exposed to these dusts.

A high prevalence of tooth decay has been reported among workers like sugar refiners, bakers and candy makers who are exposed to sugar dust. Sugar tends to deposit itself along the labial gingival surfaces of the crown where it stagnates and induces abnormal fermentation and, with the aid of bacteria, acid production. The highest prevalence of caries is reported among sugar bakers. The historical paper of Gelbier[3] has summarized the provision of some types of dental care facilities in chocolate factories in the early twentieth century.

Inorganic dusts

METALS

Metallic poisoning used to be confined to mercury, lead and arsenic but with the expansion of industrial processes more and more metals are being used and the worker is now exposed to the hazards from them. Though some pure metals may be harmless, their compounds and particularly their acid salts are toxic.

Copper produces greenish stains on the teeth due to inhalation of the dust. It is also reported to occur among musicians who use brass instruments which impinge on the front teeth, which are affected by the copper in the alloy. The colour is due to the formation of the carbonate or subacetate of copper.

Nickel reproduces green stains on the teeth of the workers because of inhalation of the dust and as a result of the action of the salivary contents on the metal.

Workers in iron mines develop a fine black line on the teeth approximately 1 mm or so above the gingival line and in crevices. Sometimes the black pigmentation is present over a rather large surface. The stain usually recurs after its removal. It is interesting to note that the teeth of individuals with such stains tend to show a reduced incidence of caries. Iron may be inhaled in the form of dust or fumes in the processing of steel rods with hydrochloric acid.

Bismuth handlers and dusting powder makers, who are constantly inhaling dust, develop a black or purplish pigmentation round the gingival margin due to the precipitation of sulphide. A similar patch of discoloration is often produced on the mucosa of the cheek in the part that is resting against the gingivae.

Air contaminated with chromic acid mist or with the dust from chromates or bichromates of potassium and sodium is the principal source of exposure in industry. Exposure to these substances occurs among chromium platers, colour workers, calico printers, photographers, litho-etchers, chrome tanners and steel workers. Exposure to chromic acid and chromates and bichromates may produce blue pigmentation of gingivae and oral mucosa, gingivostomatitis, necrosis of bone and ulceration of gums and oral tissue. Gomes[4] reported that more than 50 per cent of the workers engaged in electroplating in Brazil had dental disorders caused by high chromium content in the work environment.

Lead produces the well known blue line on gums, sometimes called the 'Burtonian line' and described by Grisolle[5] and Burton[6]. It consists of fine granules of pigment arranged in the form of a dark blue stippled line within the tissue of the gum and about a millimetre from the margin. It is more marked round teeth having infected gingival troughs and may occasionally be found on the mucosa of the cheek opposite such teeth. The line is more frequently seen on the mandibular gum than on the

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maxillary and in the incisor region than in the molar. Although this is not a disease of the teeth it has been mentioned here because the stain does not occur if there are no teeth. The stain is a precipitate of lead sulphide caused by the action of hydrogen sulphide upon the lead salts in the circulation.

Despite its lying within the tissues, careful cleaning of the mouth and teeth removes the pigment. The stain is indicative of absorption and not of intoxication. Its intensity and size provide a rough guide to the duration and severity of exposure to lead. It occurs in plumbers, and compositors, through inhalation of dust and from the fumes from lead battery casings. Workers involved in the manufacture of hair dyes and cosmetics containing lead may also become affected.

Localized argyria may result from exposure to metallic silver during industrial processing of the metal. It gives an appearance similar to that of tattoo work.

Manganese is used in the manufacture of iron and steel, organic chemicals and dry cells, in photography, in the fertilizer industry, paints and ceramics, glass industry, dyeing, printing and bleaching processes, in the preservation of wood and in disinfecting and oxidizing processes apart from being used in various mining operations. Sometimes a black deposit of hydrated manganese dioxide may be formed on the teeth when the condition in the mouth predisposes its formation.

Workers are exposed to cadmium during various operations in zinc smelting plants, rolling mills, nickel–cadmium battery factories, electrical industry, automative engines, aircraft engines, marine engines, electroplating welding and soldering processes, manufacture of glass, dentistry and photography. The workers in these industries may show some changes in their teeth because of exposure to cadmium. The workers may get yellow or gold-brown stains particularly on the labial surfaces of the front teeth, and these stains are most intense on the neck. Calculus is also stained. A staining of calculus (i.e. the hard deposit on teeth) rather than the teeth themselves can be removed by scaling as the stain is simply chipped away and lost with the calculus when the latter comes off. However, staining of the underlying tooth tissue might still remain. This pigmentation is a 'danger' sign of toxic absorption.

Yellow staining of teeth is sometimes seen among workers exposed to tin. It is due to a deposit of tin sulphide. In printing presses the lino type metal is an alloy of 85 per cent lead, 12 per cent antimony and 3 per cent tin. It is kept in a molten state in a container on the machine. The metal fumes come out and may be inhaled by the workers. Workers in tin mines are also exposed to stannic oxide dust. The staining presumably occurs when there is a pre-carious or early carious lesion present and non-carious enamel shows no colour changes.

Striations are produced in the dentine of the incisors of rats which have been fed with diets containing strontium. No authentic report of the effect of strontium on human teeth is available.

Workers in mercury mining, the manufacture of thermometers, barometers etc., electrical industry, pharmaceutical industry, photoengraving, manufacture of felt hats, identification of fingerprints etc. are exposed to the hazards of mercury poisoning. Since mercury evaporates even at an ordinary temperature it contaminates the air during various industrial processes. Although it can be absorbed through skin, poisoning occurs mainly through the respiratory tract. Apart from producing symptoms like salivation, stomatitis, tremors, nervousness, irritability, depression, insomnia, cachexia etc., it also affects the teeth and gums. The gums become tender, swollen, red, ulcerated and bleed readily and the teeth become loose. Kussmaul[7] reporting the pitiable condition of mirror makers found almost every male adult to be without a single tooth in Furth and Nuremberg. This may have been because of poor dental services in the nineteenth century. A mercurial line on the gums is now hardly ever seen. It usually resembles the blue line due to absorption of lead but is sometimes dark brown. Vigliani *et al.*[8] reported gingivitis and loss of teeth among workers in the felt hat industry exposed to mercury.

Dentists are also subject to danger from mercury[9].

NON-METALS

Flourine, hydroflouric acid and silicon fluoride are used in the superphosphate industry, during the manufacture of phosphorus and in the production of hydrogen peroxide. Fluorine or the vapours of hydroflouric acid, if inhaled, cause a burning pain in the chest, cough and even hemoptysis. The ultimate result is slow ulceration of the gums, nasal mucosa, larynx, bronchi and conjunctivae.

Fluorspar or fluorite is calcium fluoride and is used in the manufacture of steel, ceramics and hydrofluoric acid. Fluorapatite, a fluorine compound, is evolved as a by-product in the manufacture of super phosphate. Cryolite (sodium aluminium fluoride) is used as a flux in metallurgy and contains as much as 54 per cent fluorine. Crushing, grinding, grading, drying and all handling of it produce dust and all workers in the factory are exposed to its hazards. Apart from leading to fluorosis of bones and ligaments, anaemia and other toxic signs and symptoms the workers are likely to develop fluorosis of teeth in their formative stage. The condition consists of greyish and chalky-white blotches and streaks scattered over the entire tooth surface involving all the teeth. The surfaces of some teeth are dotted with minute, irregular and shallow pits in the enamel. Sometimes there is incomplete calcification of the cusp tips. In about 40 per cent of cases this general condition is aggravated by discoloration of the enamel from light brown to almost black. The essential malformation is in the cementing substance between the enamel rods on the outermost part of the surface of the enamel. Evidently fluorine compounds produce a direct local action on enamel-forming cells. Lezovic and Arnost[10] reported four cases of occupational fluorosis in individuals who had been working in an aluminium plant for periods of up to 12 years. Their teeth contained unusually high fluorine levels. It may be because of ionic interchange at the tooth surface in these workers. Such ionic activity occurs on the enamel surface, for example, during periods of dental decay and subsequent re-mineralization of the teeth. During the latter phase, the decay process is reversed.

DENTAL FLUOROSIS IN THE OFFSPRING OF THE WORKERS

It is to be noted that mottling of the dental enamel can occur only when the teeth are subjected to fluorine compounds during their development. In case of permanent teeth such exposure must take place before the age of nine. It follows that adults absorbing fluorine compounds during their work in a factory cannot have alterations in their dental enamel. However, it was noted that the children of female cryolite workers in Copenhagen showed mottling of the teeth. These cases show that fluorine

compounds are excreted in the milk of the women after their exposure to cryolite dust. This seems so far to be the only authentic example of transmission of an occupational disease to the offspring of a factory worker. The exact mechanism of such a phenomenon needs elucidation.

Workers are exposed to phosphorus in the manufacture of matches, rat-poison, fireworks, smoke screens, marker shells, tracer bullets, bombs, hand grenades, phosphor-bronze, cellulose, dyes, soaps, fertilizers, plasticizers and insecticides, and in petroleum refineries, paper industry, printing and rust-proofing of steel are exposed to its hazards.

The first symptom of phosphorus poisoning is toothache which usually begins in a tooth already affected with caries. A dull red spot on the buccal mucosa is seen at this stage and there is usually a sinus surrounded by dull red mucosa leading to a cavity underneath. Sequestra up to one centimetre in diameter may be found. They are both osteoporotic and carious. A major report on the problems of phosphorus workers was produced in 1899 by a dentist George Cunnigham and two of his colleagues. Therein they described a condition called 'phossy jaw'. Among other recommendations they stressed the need to appoint a dentist in each factory. As a result, Bryant and May started a two-chair surgery, probably the first of its kind by way of an industrial dental service at their London factory[11]. Ward[12] reported 18 cases of phosphorus necrosis among workers engaged in the manufacture of fireworks. Hughes *et al.*[13] reported 10 typical cases of phosphorus necrosis of the jaw.

Teeth are known to remain unaffected by arsenic. However, oral mucous membrane may become intensely inflamed and severe gingivitis with pain may occur. Local contact with arsenic trioxide often produces ulceration of the gums. Frost[14] has described the harmful effects among industrial workers of exposure to arsenic. Hairdressers in the thirties and forties suffered from arsenic toxicity because of their practice of holding hair grips with their teeth.

Citric acid, tartaric acid, hydrochloric acid, nitric acid and sulphuric acid etc. affect the teeth of the workers exposed to these acid fumes. Decalcification of enamel and dentine occurs following exposure to acid among workers in factories manufacturing explosives or acid dippers. The acid fumes deposited on the exposed portions of the teeth react with the enamel and decalcification results. The earliest reaction consists of a superficial decalcification of the enamel of the labial surface of the tooth which is exposed the most. Mastication and tooth brushing wear off the partially decalcified areas and produce flat smooth surfaces. The degree of erosion increases with the length of period of employment. The eroded surface is smooth and polished and never pitted. When the enamel has been destroyed, the dentine is attacked and there is brown or black discoloration of the affected teeth but they retain their polish. While the erosion is taking place the pulp chamber shrinks and the condition is painless except in rare cases where the erosion is so rapid that bacterial invasion of the pulp cavity occurs causing abscess formation. Barsotti *et al.*[15] reported that 19.2 per cent of workers exposed to tartaric acid showed erosion of the incisive and canine teeth. Malcolm and Paul[16] in a study of workers in the storage battery industry reported that workers exposed to acid mist were found to have erosion of the incisor teeth. Gamble[17] in a study of acid battery workers found that the ratio of observed to expected prevalence of teeth etching and erosion was about four times greater in the high acid-exposure group. The earliest case of etching occurred after exposure for 4 months to an estimated average exposure of 0.23 mg sulphuric acid/m^3.

Physical Factors

Occupational injuries may give rise to concussion, loosening or fracture of teeth.

Modifications of the teeth by abrasion occur among shoe makers, upholsterers, glass blowers, dress designers, dress makers and seamstresses. They result from holding nails, tacks, needles, glass tubes and thread reinforced by metals between their teeth. The cobbler holds a ready supply of nails – 30 or so – in his mouth and serves them out from his teeth, and the upholsterer and sofa maker does the same with tacks. Nurses using hairgrips in the same manner sometimes develop changes in their teeth.

Musicians, especially the wind instrument players, develop dental problems. These are mainly the problems of 'embouchure'. The way in which the lips and mouth are applied in the blowing of a wind instrument is known as 'embouchure'. The presence of dental defects can affect the playing of the instruments. The dental problems among wind instrument players have been studied[18–22].

Teeth of people who have received harmful doses of X-ray radiations like X-ray technicians, radiographers, research workers and watch dial painters (who lick their brushes) are sometimes affected. Way back in 1925 Hoffman[23] reported four cases in which necrosis of the teeth and jawbones had occurred after the practice of pointing the paint brush with radium on it. The damage by radiation may not appear for several years. The gingivae become inflamed, ulcerated and painful and a foul breath may be present. This may be followed by gingival recession, periodontitis and damage to alveolar bone. The teeth become loose and show resorption of the root. In the later stages, osteomyelitis, osteonecrosis and osteosclerosis of the jaw accompanied by loss of teeth and the formation of sequestra occur. The most common manifestation of radiation injury is a typical destruction of tooth substance resembling dental caries at the cemento-enamel junction and sometimes called 'radiation caries'. Teeth often seem brittle, and pieces of the enamel may fracture away from the tooth. Recent experiences with radiotherapy of malignant diseases[24–26] have shown that excessive dosage of radiation has severe adverse effects on the teeth.

Increased atmospheric pressure may produce bleeding from gingivae among people working in compressed air chambers. The same complaint may be noticed among aviators due to decreased atmospheric pressure. During the Second World War dental pain was observed in some of the personnel of aircrews flying at high altitude or entering the low pressure chamber. The cause of the pain has been attributed to air embolism being present in the dental pulp. Pain is particularly liable to occur if there is already an inflammatory condition of the pulp.

Effect of Adverse Environmental Factors on Rat Incisors

Comparison between the histology and chemical composition of the incisors of rats acclimatized for 18–24 weeks to cold, neutral or hot atmospheres, at various

barometric pressures showed interesting results. Cold by itself induced negligible histological changes, but altitude (750 or 380 mmHg) produced changes in the mesenchyma of the teeth. These were made severe by superimposed cold (3°C), but superimposed heat (36°C) counteracted some of these effects. The latter group, however, had ectodermal changes. Chemical studies revealed significantly reduced concentrations of calcium, phosphate and magnesium in the teeth of the attitude- and heat-exposed rats. The teeth of rats are continuously growing and this process is similar to the unerupted human tooth formation, and therefore the effects observed in the rats in this experiment may presumably be seen among children if they are exposed to the same conditions.

Because of physiological strains in certain occupations people may develop bruxism or bruxomania, i.e. the habit of constantly grinding their teeth. Athletes engaged in physical activities often develop this habit. The exact reason for this is unknown. Occupations in which the work has to be near precise such as that of the watch maker are likely to cause bruxism. When the habit is firmly established severe searing or attrition of the teeth may occur.

Importance of oral hygiene

Neglect of oral hygiene predisposes the teeth to the development of occupational dental diseases and aggravation of the disease once it occurs. The presence of the aetiological agent alone is not enough to case dental diseases. The general and oral health of the patient are equally important.

The scope of this paper does not include a consideration of the treatment of various occupational dental disorders. However, there is no denying that prevention of their occurrence is better than a treatment once they occur. Prevention can be effected by proper working conditions and observation of strict oral hygiene. Since oral hygiene is a matter of habit it should be inculcated in early childhood. So that a person is well guarded against any occupational hazard in the course of his employment. Moreover, the training for a particular occupation should include attention to the general health of the worker and particularly to those parts of the body which are exposed to dental occupational hazards. Adequate provision for industrial health should aim at prevention, if possible, or an early recognition and treatment of dental occupational diseases. L'Epee[27] has stressed the importance of oral and dental examination as part of the occupational health service.

More studies of occupational diseases of teeth should be conducted in order to check or confirm previous reports and to discover possible manifestations arising among workers in new industries. It is essential for all concerned to have an epidemiological knowledge of the state of oral health of the workers, especially if there are conditions in the industry likely to cause dental disorders[28].

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