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DENTAL FLUOROSIS

GHOLAMREZA HEIDARI

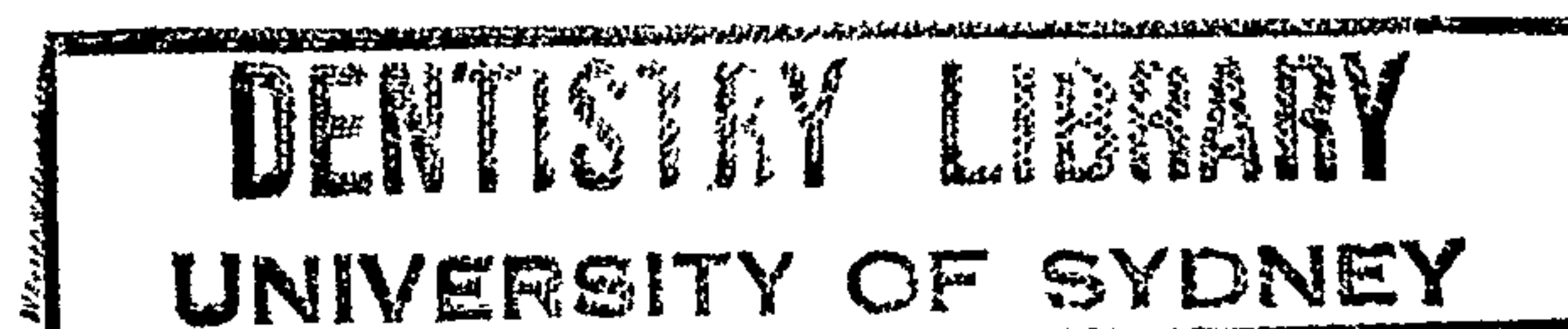
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SUMMARY

Dental fluorosis is a hypoplasia or hypomineralisation of tooth enamel or dentin, produced by the chronic ingestion of excessive amounts of fluoride during tooth formation.

Due to the universal presence of fluorides in drinking water, soil and atmosphere humans are exposed to various levels of fluoride intake through foods, water, industrial and pharmaceutical products. Water-born fluoride, however, represents the largest single component of this element's daily intake. Teeth, bones and tissues are the richest in fluoride and the occurrence can be considered with respect to the distribution and concentration as stored fluoride and transitory fluoride.

The range of fluoride levels varies in different parts of the world in drinking water from 0 to 95 ppm, in soil between 200 and 290 ppm, in sea water 0.8 to 1.4 ppm, in fish 19.3 to 84.5 ppm, in tea leaves 97 ppm, in whole potatoes 6.4 ppm and in milk 0.1 to 2 ppm.

Fluoride is absorbed into the body mainly in the stomach, and this can also occur from the lungs by inhalation of fluoride dusts and gases. The main route of fluoride excretion is via the kidney. In adults 20 to 60 per cent and infant 40 per cent and children 50 per cent of an ingested dose is found in urine.

The extent of fluoride uptake in different parts of the skeleton and dentition depends upon the amounts ingested and absorbed, the duration of fluoride exposure and type, region and metabolic activity of the tissue. The

distribution of fluoride within bone is not uniform, endosteal and periosteal surfaces usually have a higher fluoride content than the central parts of compact bone. Fluoride uptake in dental tissues increases with age and with increasing fluoride concentration in the water supplies, but the fluoride content of dentin and enamel is lower than that found in bone from the same individual and dentin contains four times more fluoride than enamel.

Fluoride was first added to public drinking water in the 1940s and in the 1950s and has been incorporated into toothpaste, other dental products and dietary supplements. Inappropriate use of fluoride can cause toxic effect on human either, acute or chronic. The signs and symptoms of acute fluoride poisoning include, nausea, vomiting, diarrhoea, abdominal pain, shock, pallor, weakness, weak heart tones, cyanosis. Death occurs as early as two to four hours, but favourable response to treatment may be prompt, usually within 24 hours. Chronic fluoride poisoning from exposures up to 100-200 mg daily causes abnormalities in bony tissues such as osteosclerosis, depending on the daily fluoride intake and duration over months or years.

There are several factors which influence the prevalence of dental fluorosis such as drinking water, dietary supplements, temperature and altitude. The first sign of dental fluorosis appears as fine striae of accentuated perikymata. In more affected teeth, fine lines are more broader and pronounced, occasionally merging to produce irregular, scattered, paper white areas. With increasing severity, the irregular opaque areas merge until extensive areas appear chalky white. In severely damaged teeth,

almost the entire enamel surface appears to be "corroded", there is an extensive loss of surface enamel and the pathogenesis of fluorosed enamel is most often is, a direct effect of fluoride ion. To measure these enamel changes there are several classification system or indexes that record all enamel defects such as, Dean's Index, Thystrup and Fejerskov Index (TF) Index and Tooth Surface Index of Fluorosis (TSIF).

Differentiating between fluorotic and non-fluorotic defects of dental enamel is an important diagnostic decision in epidemiology and public health dentistry. The diagnosis of the early stages of dental fluorosis is difficult. Some of these difficulties include enamel opacities of non fluoride origin. Mild forms of mottling enamel require no treatment, but moderate and severe forms of dental fluorosis, most often front teeth need special treatment.

Discovery of the etiology of fluorosis, makes it possible to direct prevention of this disease toward standardisation of the fluorine content in the environment, particularly in drinking water supplies. Therefore, the basis of dental fluorosis prevention consists of regulating the amount of fluorine in drinking water, the replacement of water supplies with high fluoride content, and defluoridation of the water.

The level of fluoride in different parts of Boushehr province in Iran ranges from 1 to 5 ppm, and due to the lack of knowledge of fluoride and dental fluorosis among the professionals, health workers and government decision makers, some recommendations have been made.

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1 INTRODUCTION

Endemic dental fluorosis is a specific disturbance of tooth formation caused by excessive intake of fluoride during the formative period of the dentition (WHO 1984). Clinical dental fluorosis is characterised by lustreless, opaque white patches in the enamel which may become striated mottled and or pitted. The opaque areas may become stained yellow to dark brown. The affected teeth may show a pronounced accentuation of the perikymata and, in more severe cases, multiple pits and larger areas of hypoplasia of the enamel appear to such an extent that the normal morphology of the tooth is lost (Dean 1934, Möller 1965, Murray 1976). The manifestations of chronic fluoride intoxication depend upon the amount ingested, the duration of exposure and the age of the subject (Möller 1982).

The earliest report on a condition which was characterised by opaque, discoloured and disfigured teeth and which appeared to be of endemic origin dated back to 1888 when Kühns reported such cases in certain areas in Mexico. A similar condition was observed in immigrants from Naples in Italy (Eager 1901). Several reports from other parts of the world and, in particular, the observations of Colorado Brown Stain by McKay and Black (1916) eventually, in 1931, led to the discovery that this condition was correlated to the fluoride content in drinking water (Churchill 1931, Smith, Lantz and Smith 1931, Möller 1982).

On the basis of McKay and Black's pioneering work, Dean (1934, 1938a) continued to elucidate the relationship between the occurrences of dental fluorosis and the concentration of fluorides in the drinking water. On the basis of statements in earlier reports - that teeth with dental fluorosis were no more susceptible to dental decay than normal teeth, Dean conducted extensive epidemiological studies which demonstrated the existence of an inverse relationship between the severity of dental caries and the fluoride content in the drinking water (Dean 1938b, 1942). The identification of this relationship led to the idea of adjusting the fluoride content in fluoride-deficient drinking water. Between 1944 and 1947, the first communities to introduce this caries preventive measure were Grand Rapids, Newburgh, Brantford, Sheboygan and Evanston (Möller 1982).

Equations for calculating the optimal fluoride level in drinking water (the level at which the caries reducing effect is the highest, and the risk of provoking dental fluorosis the least) were devised by Hodge (1950), Galagan and Lamson (1953), Galagan and Vermillion (1957) and Richards et al (1967).

The caries preventive efficacy of both ingested and topically applied fluoride has been well established (Backer Dirks et al 1978, Binder et al 1978, Brudevold and Naujoks 1978, Von der Fehr and Möller 1978). The goal has been to provide this efficacy without incurring unwanted side effects, most commonly dental fluorosis.

The effects of acute and chronic fluoride overexposure on tissue and organ systems other than enamel have been reported (Hodge 1979, Smith and Hodge 1983, Hodge and Smith 1986, Newbrun 1987, Whitford et al 1987).

Dental fluorosis was recognised as a phenomenon in the early part of the century by McKay in Colorado. The earlier observations of this relatively common, geographically defined and distinctive pattern of abnormal tooth enamel initiated clinical, epidemiological and laboratory research which identified an association between mottled enamel, which subsequently became known as dental fluorosis, and fluoride (Szpunar and Burt 1987).

Water with a high fluoride content is usually found at the foot of high mountains and in areas with certain geological formations, particularly those of marine origin. Typical examples are belts extending from Turkey through Syria, Jordan, Egypt, Libya and Algeria to Morocco, from Egypt through Sudan and Kenya to Tanzania, and from Turkey through Iraq, **Iran**, Afghanistan, India and northern Thailand to China. Since fluoride, as it occurs in drinking water, is colourless, odourless and tasteless, its presence in excess only becomes evident if chemical analysis is performed or if cases of dental or skeletal fluorosis are found (Phantumvanit et al 1988).

Dental fluorosis still remains a major issue in the mainstream of basic, applied and health services research, because:

1. The clinical condition led to discovery of a trace element with a caries inhibitory property,
2. Fluorosis is a permanent record of abnormal tooth development,
3. The mechanism of fluorosis remains unexplained,
4. Fluorosis has important implications in the community because of its cosmetic, environmental and disease preventive roles .

(Cutress and Suckling 1990).

Bushehr province, with a surface area of about 27,653 square km, is located in south-west Iran. This province is situated between 37 degrees 14 minutes to 30 degrees 16 minutes North latitude, and 50 degrees 6 minutes to 52 degrees 58 minutes East longitude. This province is limited by Khozestan province, Kohkilooyeh and Boyrahamadi province in the North, Fars province in the North-East and East, Hormozgan province in the South-East to the coastal region and the Persian Gulf to the West and South generally. Bushehr city lies in the center of this province with an area of 984.5 sq. km; situated between 50 degrees 51 minutes geographical longitude, and 28 degrees 59 minutes geographical latitude. It is situated between 1 to 5 meters above sea level, and the temperature ranges between 50 degrees centigrade in summer and 1 degree centigrade in winter; humidity ranges between 12 per cent to 98 per cent, with different fluoride concentration (1 to 5 ppm F) in the drinking water. Unfortunately, there is no epidemiological data and clinical data on the prevalence and severity of dental fluorosis at the provincial and national level available. Any use of

fluorides, whether systemic or topical, in caries prevention and treatment in children results in ingestion and absorption of fluoride into the blood circulation. The mineralisation of teeth under formation may be affected so that dental fluorosis may occur (Fejerskov, Manji and Baelum 1990).

Reports have appeared that indicate a trend towards higher dental fluorosis prevalence, occurring in both fluoridated and non fluoridated communities in the USA (Leverett 1986, Szpunar and Burt 1988), in Europe (Woltgens et al 1989), in New Zealand (Cutress et al 1985), and in Hong Kong (Evans 1988).

At the major international meeting on cariostatic mechanisms of fluoride, held in Naples in 1976 (Brown and König 1977), the consensus about the nature and mechanisms of dental fluorosis was that maximum caries reduction would only be obtained through systemic use of fluorides. Mild dental fluorosis was therefore considered an unavoidable **side - effect** which was commonly thought to result from the toxic effect of fluoride on the secretory phase of enamel formation (Fejerskov, Manji and Baelum 1990).

Differentiating between fluorotic and non-fluorotic defects of dental enamel is an important diagnostic decision in epidemiology and public health dentistry. The differential diagnosis of fluorosis is difficult unless there are new developments which provide a better understanding or a new

insight which modifies the present day consensus on fluorosis and its diagnosis (Cutress, Suckling 1990).

This thesis will review the existing knowledge of available fluoride sources, and the evidence for an association between specific fluoride sources and dental fluorosis, metabolism (availability and toxicology), prevalence, etiology and pathogenesis of dental fluorosis, differential diagnosis and indices for measuring dental fluorosis, and treatment method/s for active defluoridation.

This review will be useful to the writer and other health professionals working in Boushehr province in Iran, and will enable the writer to suggest some recommendations for actions that may be necessary in relation to dental fluorosis.

2 SOURCES OF FLUORIDE

The well established inverse relationship between the fluoride concentrations of drinking water and the incidence of dental caries has given rise to extensive research aimed at determining the optimum level of fluoride ingestion in order to obtain the maximum protective effect of this element, with the least risk of provoking adverse effect.

The following is a review, concerning the occurrence of fluorides in the environment and the exposure and metabolism of fluorides in humans.

2.1 INDIRECT SOURCES OF FLUORIDE OCCURRENCE

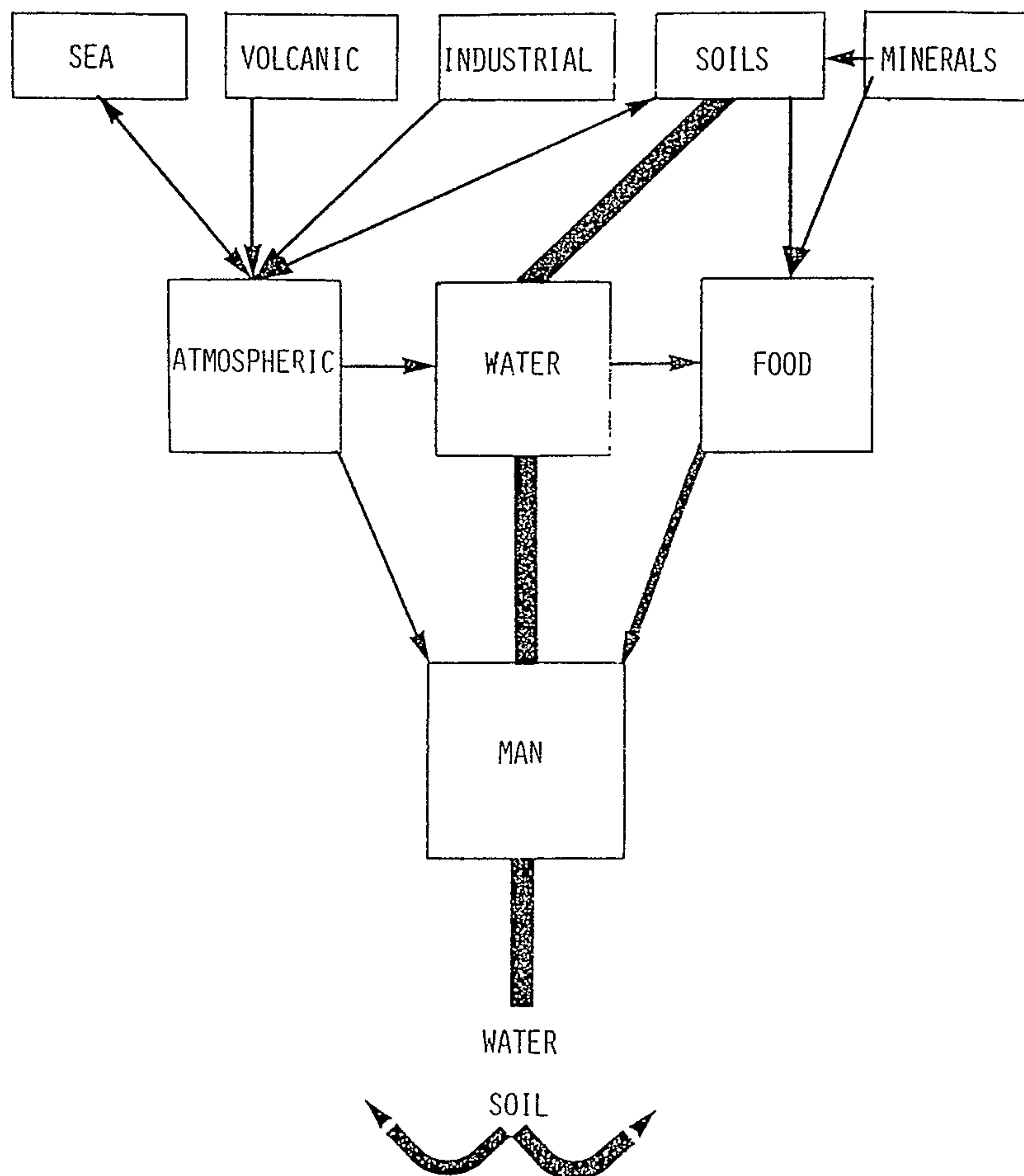
As shown in **Figure 1**, 'geochemicals' are primary and intermediate sources of fluoride. Each source is capable, directly or indirectly, of providing excess fluoride to man and animals. Often, but not always, this excess is delivered via water supplies (WHO 1982).

2.1.1 Volcanic Emission

Fresh volcanic debris has for centuries been directly associated with damage to vegetation and therefore to grazing animals. This geological activity injects a surprising amount of fluoride into the atmosphere, estimates of annual volcanic outflow of fluoride exceeding 7 million tonnes is enough to provide us each with 2g day!

Fortunately, only part returns to earth via precipitation - it is not clear what happens to the remainder (Bartel 1972).

Figure 1 Fluoride sources.
Source: WHO (1982)



2.1.2 Sea Water

Sea water contains appreciable levels of fluoride (0.8 - 1.4 ppm) which recycles via airborne spray although little is known of its fluoride distribution to land, fresh water and soils. There is no evidence of soil fluoride being higher in seaboard areas or islands (WHO 1982).

2.1.3 Industrial fluoride

Industrially produced fluoride which is predominant in pollutant form as gas, aerosol, smoke and dust and is known to damage vegetation, plants, trees, grazing animals and to produce fluorosis. In non-industrial areas, air concentrations are insignificant, whereas industrialised regions can still raise airborne fluoride levels to as high as 2 ug/m^3 in the local atmosphere. Industrial fluoride is considered the major pollutant affecting animals despite strict regulations on its emission (Crissman et al 1980).

Prevailing climatic conditions, vegetation and living conditions will very much dictate the area, people, food and water supplies influenced by the fall-out. There is a wealth of fluoride containing raw materials used in industry. Reviews by Marier and Rose (1971) and Robinson (1972) provide information on the source and volume of airborne fluoride. Particulate fluoride appears to dominate over gas and acid in industrial locations (Dinman 1976) and hence, measurement of fluoride in water (e.g. local pools) can greatly under-assess total fluoride output. It appears that industrial emission of fluoride is predominantly associated with phosphate fertiliser, aluminium and coal burning industries such as glass, steel, bricks, ceramics, and fibreglass (Smith and Hodge 1979).

2.1.4 Soil

Soils themselves vary considerably in fluoride content, reflecting contributions from base minerals, atmosphere, volcanic fall-out and seaspray. This type of accumulation appears to be supported by analyses of soil profiles which show a distinct increase in fluoride content with depth from the surface (Robinson and Edgington 1946, McIntyre 1949).

Particular mention should be made that, industrial fertilisers can contain up to 1 per cent fluoride. Its long-term continual application to soils for intensive agriculture, however, does not appear to be a hazard because of the stability of the fluoride complex, its low solubility and its rapid elusion from soils (Stewart et al 1974).

2.1.5 Minerals

Although the chief fluoride-containing minerals are fluorspar (CaF_2), cryolite (Na_3AlF_6) and fluorapatite ($\text{Ca}_{10}(\text{PO}_4)_2\text{F}_2$), these are not necessarily the main natural contributors to fluoride in water and soils. Various other less common but more soluble inorganic combinations can contribute substantially to biologically available fluoride. For example, rock salt containing sodium fluoride is 1000 times more soluble (42.2 g/l) than fluorspar, (0.04 g/l) and cryolite is only very slightly soluble (WHO 1982).

2.2 DIRECT SOURCES OF FLUORIDE OCCURRENCE

Fluoride experience is directly linked to the drinking water, the food and the air. For the majority of mankind the order of contribution to fluoride history is water, food, atmosphere (air) and pharmaceutical products (WHO 1984).

2.2.1 Water

Epidemiological, analytical and physiological studies so far indicate that with few exceptions, drinking waters are the principal determinant of fluoride availability to man. The subject has been reviewed many times and in essence, the occurrence of fluoride in water varies considerably from very low to very high. It is undoubtedly the availability of dissolved fluoride in water which explains its pre-eminence as the source of fluoride to man. Cutress in 1982 emphasised this because it provides a relatively simple means of identification of biologically available fluoride and its control.

Fortunately, the fluoride content of most potable waters is moderate to low, the higher levels being associated with subterranean water sources and low surface water. The fluoride is mostly present in its soluble form. Soft drinks, beer, milk, tea, coffee, all contribute to the total daily intake of fluoride. Some of these beverages increase the level of fluoride over and above that in the water base. In fact evidence pointed to an increasing daily fluoride intake via the food and beverage chain and that this process will intensify (Marier 1977).

2.2.2 Food

Despite the wealth of data available on various foodstuffs, a degree of uncertainty hovers around the significance of the stated level of fluoride occurrence (WHO 1982).

Preparation and analytical procedures are often complex since it is not clear whether, fluoride levels found represent total fluoride in the food and what proportion of that identified is physiologically available. A simulated physiological extraction procedure might provide a more realistic evaluation of available fluoride.

It is known that virtually all foods contain at least trace amounts of fluoride but daily intake is relatively low (**Table 1**). Most plants seemed to absorb very little fluoride from the soil even when the soil has high fluoride levels, but utilises fluoride from water contact.

Table 1 Concentration of fluoride in foods.
Source: Taves (1983)

Food Type	Fluoride Concentration (ppm)		
	Average	Standard Deviation	Range
Dairy products	0.25	0.38	0.02-0.82
Meat, fish, poultry	0.22	0.15	0.04-0.51
Grain and cereal products	0.42	0.40	0.08-2.01
Potatoes	0.49	0.26	0.21-0.84
Leafy vegetables	0.27	0.25	0.08-0.70
Legume vegetables	0.53	0.05	0.49-0.57
Root vegetables	0.38	0.11	0.27-0.48
Fruits	0.06	0.03	0.02-0.08
Oils and fats	0.25	0.15	0.02-0.44
Sugar	0.28.	0.27	0.02-0.78
Beverages	0.76	0.83	0.02-2.74
Non classifiable	0.59	0.19	0.29-0.87

The explanation of the low occurrence of fluoride in plants (and grazing animals) despite high soil fluoride is that fluoride is present in a stable, insoluble form (Toth and Suga 1980).

2.2.3 Atmospheric

The total airborne fluoride is the sum of wind-blown, seaspray, volcanic and industrial dust and gases. Stoiber in 1971 indicated that, fluoride may be dispersed in a free particulate, inorganic or organic form, as acids, gases or aerosols. Their linear distribution from original sources is very variable depending on climatic conditions and the stability and form of the fluoride. The majority (97%) of

non-urban areas have no detectable atmospheric fluoride, but in urban areas levels may reach almost $2 \mu\text{g}/\text{m}^3$ (Vostal 1971).

2.2.4 Pharmaceutical Products

An increasing number of pharmaceutical products contain fluorides in organic or inorganic form. Some products such as sodium fluoride tablets or combined fluoride-vitamin pills are meant as a means of intended supplement of fluoride for caries preventive use. The fluoride content in other products (such as dentifrices containing fluoride or mouthwashes which are used as vehicles for caries preventive measures affecting the fluoride content of the enamel surface or the biochemistry at the interface between the enamel surface and the oral environment) may be retained in the order 25 per cent. It is normally concluded that the use of fluoridated dentifrices or mouth rinses corresponds to an intake of 0.25 mg fluoride per day (WHO 1982).

2.3 OCCURRENCE IN MAN

Teeth, bones and tissues are derived from ectoderm (epidermis, hair and nails) are the richest in fluoride, whilst tissues of the internal organs, muscles and biological fluids contain much less fluoride and tissues of the eye contain fluoride only in traces (WHO 1982).

Absorption and availability of fluoride to body tissues is dependent on the chemical form of the fluoride and its solubility. Absorption is not confined to ionised fluoride. Organic and inorganic fluorides can pass through cell membranes without decomposition. Absorption is fast, but accumulation in bone and soft tissues is relatively slow (though much faster in children than in adults) and dependent on the amounts ingested, frequency of ingestion and period of past ingestion. Ultimately an approximate fluoride balance (slightly negative) is reached which reflects the sum total occurrence of environmental fluoride for a person.

The occurrence of fluoride in man can be considered with respect to the distribution and concentration as stored fluoride, and transitory fluoride.

Various investigations about stored fluoride in man have conflicting reports on fluoride levels in various organs and tissues. However, it is generally agreed that:

- (1) The bulk of fluoride in man is associated with the skeletal tissues,
- (2) The fluoride content of soft tissues is low,
- (3) Skeletal tissue can contain up to 0.6% wet weight (or 1100 ppm in ash), without signs of physiological hazard (McClure et al 1958),
- (4) In normal subjects, in low-fluoride areas, the range is 200 - 300 ppm fluoride (dry weight), in fluorotic subjects, the range is 700 - 7000 ppm fluoride (Singh et al 1963).

The availability of fluoride does not appear to influence the concentration of fluoride in soft tissues (**Table 2**).

Fluoride in blood, milk, saliva and tissue fluids is of a low order (0.1 - 0.2 ppm) and not much influenced by differences in environmental fluoride levels. Except for transitory elevation corresponding to recently ingested fluoride in fluids, levels remain low and constant (WHO 1982).

Table 2 Concentration of fluoride in human soft tissues (ppm dry weight).

Source: WHO (1982)

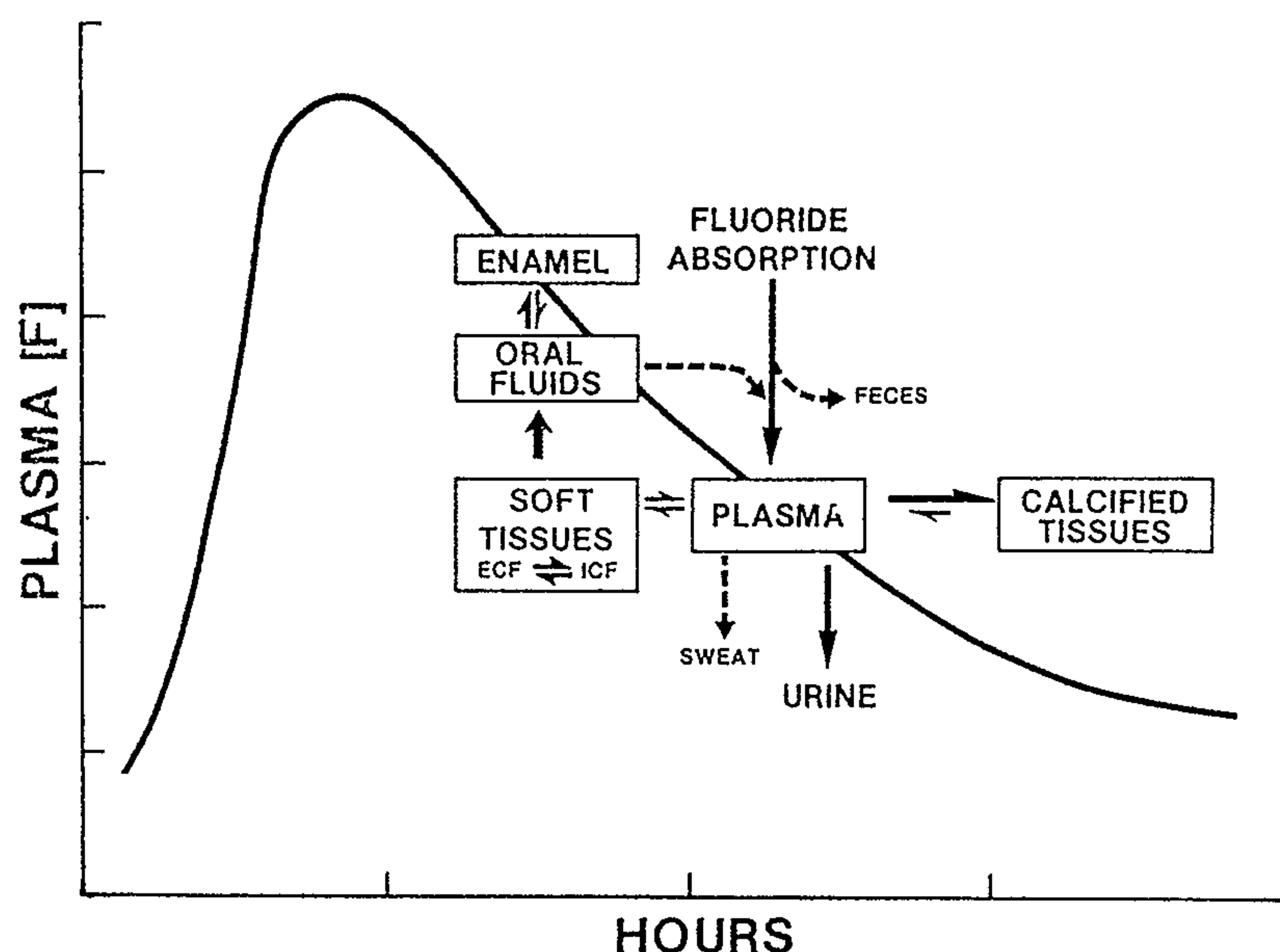
Tissue	Content of F in water	
	0 - 1.0 ppm	1.0 - 4.0 ppm
Heart	2.3	2.8
Liver	2.3	2.3
Lung	5.1	6.2
Kidney	3.3	8.5
Spleen	4.9	3.6
Aorta	41.0	25.1

3 FLUORIDE METABOLISM

Shortly after water-born fluoride was identified as the aetiological factor responsible for the mottled enamel known as "Colorado brown stain" and other descriptive terms, the inhibitory action of the ion on dental caries was recognized. This realisation led directly to the hypothesis that adjusting the fluoride concentration of drinking water to approximately 1.0 ppm would result in an important public health dental benefit. It was also recognized that knowledge of the physiological and toxicological characteristic of fluoride was essential if programs for controlled water fluoridation were to be safely implemented. Thus, interest in the metabolism and toxicity of the ion began in earnest 45 years ago. Based on the research conducted since then, **Figure 2**, shows the general disposition of ingested ionic fluoride as it is understood today (Whitford 1990).

Figure 2 The general scheme of fluoride metabolism superimposed on a typical plasma fluoride concentration curve.

Source: Whitford (1990)



3.1 AVAILABILITY OF FLUORIDE

Fluorine is the most electronegative of all chemical elements (atomic weight 19.0 and atomic number 9). Combined chemically in the form of fluorides, chiefly as fluorspar (CaF_2), fluorapatite ($\text{Ca}_{10}(\text{PO}_4)_6\text{F}_2$) or cryolite (Na_3AlF_6), it is seventeenth in the order of abundance of elements in the earth's crust (Fleischer 1953). Barth (1947) estimated that the earth's crust contained 880 ppm. Most studies have shown that the level of fluoride in soil decreases from below upwards, the mean fluoride content varying between 200 and 290 ppm (Vinogradov 1954). Fluoride is also present in sea water (0.8 to 1.4 ppm) (Thompson and Taylor 1933, Wattenberg 1943, Kappana et al 1962) and in nearly all fresh ground water (concentration varies). The range of fluoride levels in drinking water varies in different parts of the world; in Tanganyika, some areas have been reported with as much as 95 ppm F in the drinking water (Tanganyika Government Chemist 1955); in USA the concentration ranges from 0-16 ppm (WHO 1970); and in England the range is 0-5.8 ppm (Heasman and Martin 1962).

Additional fluorides are widely distributed in the atmosphere originating from the dusts of fluoride containing soils (Williamson 1953), from gaseous industrial wastes (MacIntire et al 1952) from the burning of coal fires in populated areas (Cholak 1959), and from gases emitted in areas of volcanic activity (Noguchi et al 1963). Thus fluoride, in varying concentrations, is freely available in nature.

3.2 INTAKE OF FLUORIDE

The intake of fluoride varies according to the age of persons, geographical location and dietary habits. Assuming a water fluoride content of 1 ppm, the average intake from all sources in the United Kingdom would be about 3.2 and 2.2 mg per day for men and women respectively (Longwell 1957). In a study of daily intake of fluoride from food and drinking water containing 1 ppm fluoride, McClure (1949a) estimated that the total daily intake of a 10-12 year old child would be 0.016-0.069 mg per kg of body weight. Thus for a 50 kg person the total daily intake would be 0.8-3.5 mg. In children aged 5-14 years, the intake is 1.2 mg. McClure (1949a) determined the fluoride content in different types of food. For example, dried mackerel and dried salmon, contain 84.5 ppm and 19.3 ppm respectively, tea leaves contain an average 97.0 ppm fluoride and whole potatoes contain 6.4 ppm fluoride. McClure (1949a) considered that about 0.3-0.5 mg fluoride were provided by the ordinary mixed solid foods of the American adult. Water consumption also depends on dietary habits and socio-economic conditions. Milk is known to have a low and constant level of fluoride being in order of 0.1 to 2.0 ppm. While milk is low in fluoride, formulas made with evaporated milk and tap water containing 1 ppm fluoride can have significant high levels of fluoride, up to 0.9 ppm. Dry formulas prepared with fluoridated water can reach levels about 50 times that of human milk (WHO 1982).

3.3 ABSORPTION OF FLUORIDE

Fluoride is readily absorbed into the body mainly in the stomach and is passive in nature, no active transport mechanism is involved. The maximum plasma fluoride concentration is reached within 30 to 60 minutes. The short interval between intake of oral doses of soluble fluorides and excretion in the urine is also evidence of very rapid absorption (WHO 1982).

Absorption can also occur from the lungs by inhalation of fluoride dusts and gases. A third and very rare route of absorption is through the skin such as when hydrogen fluoride is applied to the skin. However, the resulting burn to the skin is more serious than is the fluoride that is absorbed (Goodman and Gilman 1965).

Soluble fluorides in drinking water will be absorbed nearly completely, regardless of the level of fluoride in the water supply (McClure et al 1945, Largent 1960). The question has been raised whether fluoride in milk is as readily available as it is in water. Ericsson (1958), using ¹⁸fluoride, found that at concentrations of 1 and 4 ppm fluoride, the absorption of fluoride from milk was slower than that from water even though the ultimate percentage absorbed was nearly the same whether fluoride was supplied in milk or in water. Tea is a rich natural source of fluoride. The average fluoride concentration in tea leaves is approximately 100 ppm. Approximately 90 per

cent of the fluoride from this source is extracted after infusion, so that the fluoride concentration of the infusion is around 1 ppm fluoride (Ham and Smith 1950). The absorption of fluoride from food depends on the solubility of the inorganic fluorides in the diet and on the calcium content of the diet. If calcium (as calcium phosphates or calcium carbonate) or aluminium compounds are added, the fluoride absorption is markedly reduced - to about 50 per cent. In such cases, the fluoride is bound in a less soluble form and faecal excretion increases (Hodge and Smith 1965).

3.4 EXCRETION OF FLUORIDE

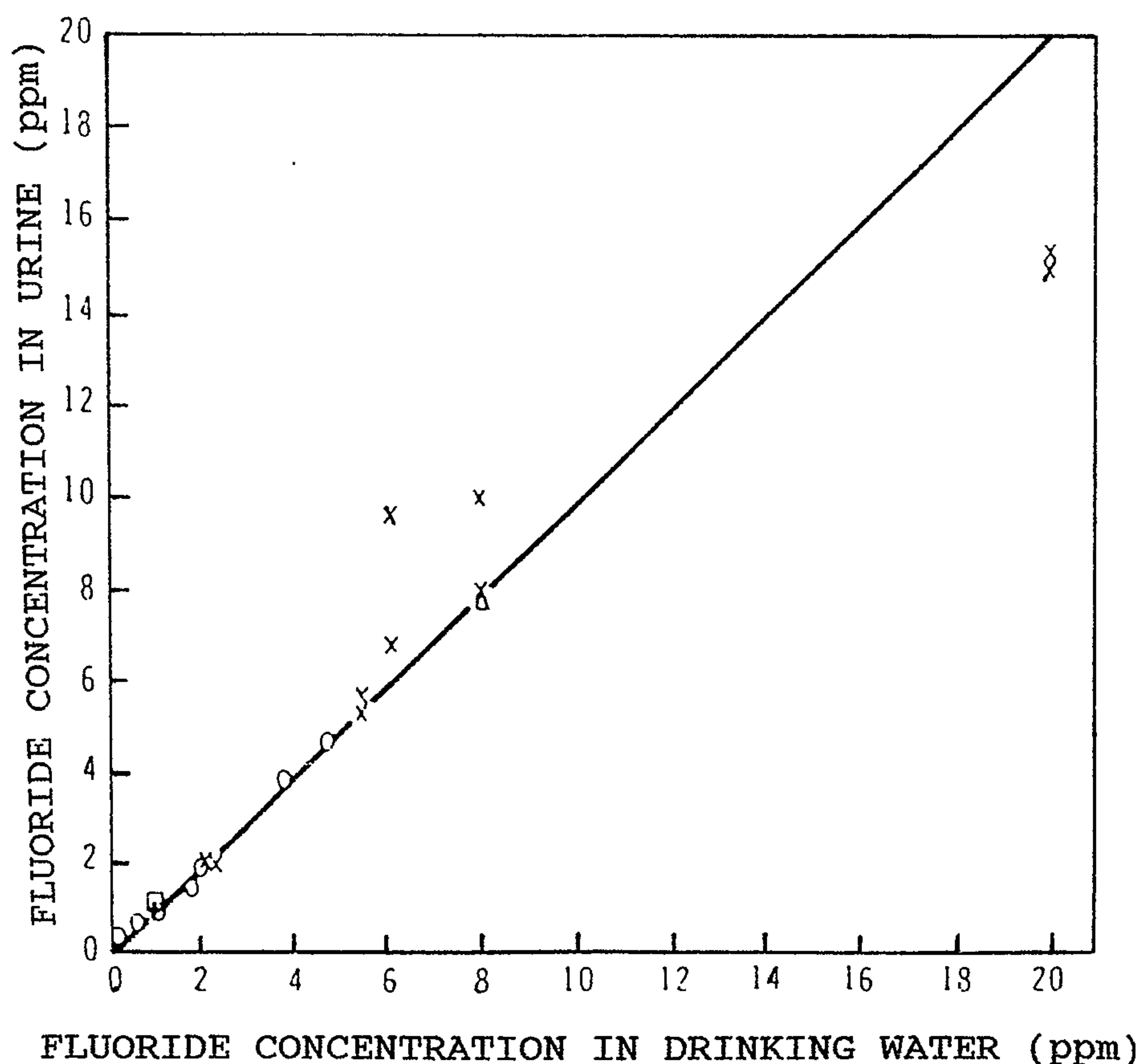
The main route of fluoride excretion is via the kidney. After a glomerular filtration, varying amounts of fluoride are absorbed in the proximal and distal tubules. The mechanism involved is simple passive diffusion. Both urinary flow and pH are involved in regulating the renal clearance of fluoride (Whitford and Pashley 1976, Ekstrand et al 1978, 1980).

This has several practical implications on the metabolism of fluoride. High urinary flow and an alkaline urine will result in a rapid clearance of fluoride from plasma while a low urinary flow and acidic urine will result in slower renal elimination rate of fluoride. In an adult, 40 to 60 per cent of an ingested dose is found in urine. The factor

may vary considerably, since it is influenced by the degree of bone uptake, which in turn is determined by the age of the subject and their present and past fluoride intake. It is therefore evident that the kidney, on a short term basis, is regulating the overall metabolism of fluoride in the organism. Bone, is the corresponding organ for the long term regulation (Möller 1982).

Human urinary fluoride concentrations depend on the drinking water concentration as shown by McClure and Kinser (1944), who measured the urinary fluoride concentration from 1,900 young males from areas where the fluoride concentration in the drinking water varied from 0.5 to 5.1 ppm (Figure 3).

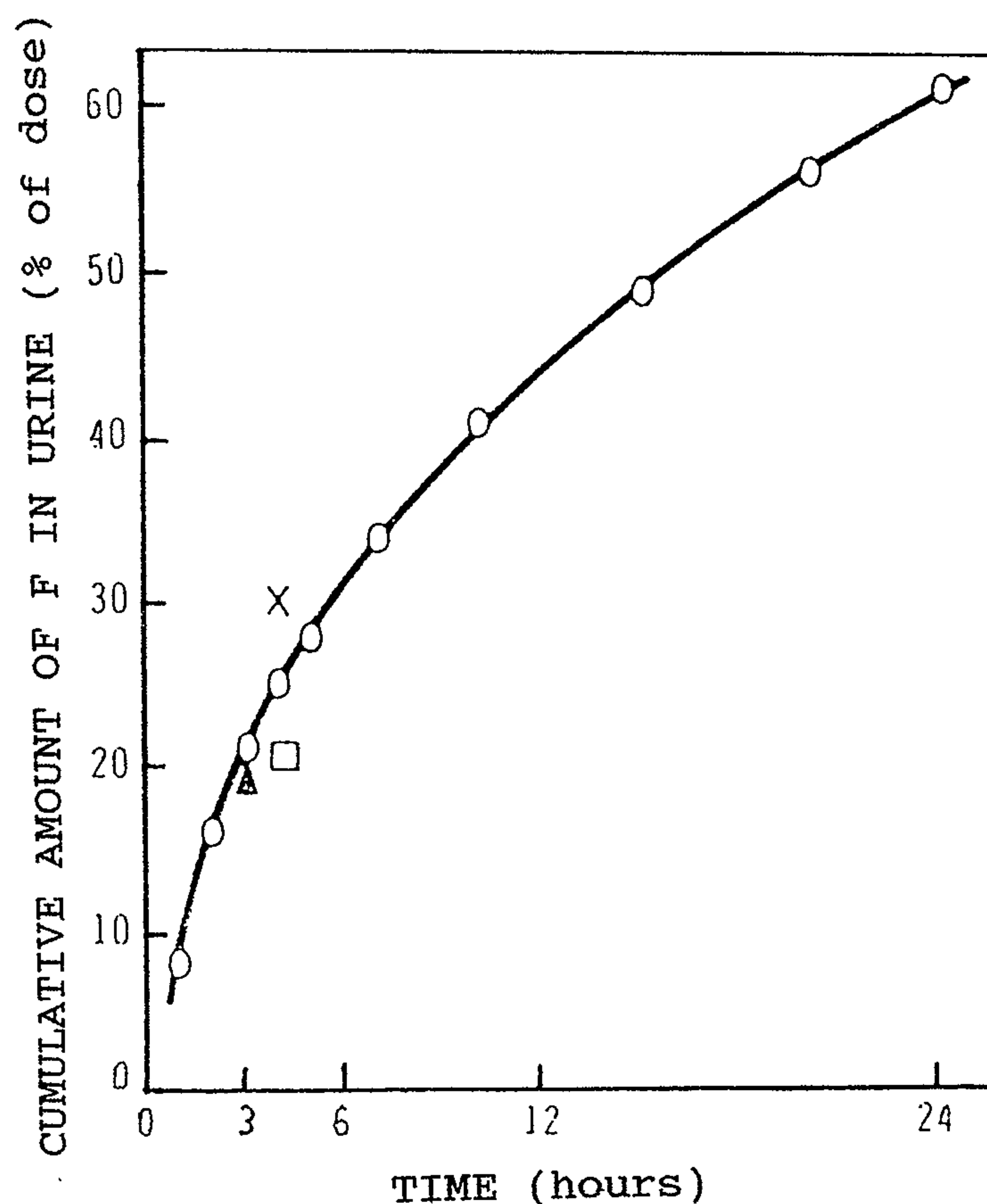
Figure 3 Relation between fluoride concentration in the human urine and that in the water supplies used.
Source: WHO (1970)



Fluoride is rapidly excreted from the body. Even small amounts, 1.5 mg (Hodge and Smith 1965) or 5 mg (Zipkin and Leone 1957), taken in a glass of water, are absorbed and excreted so rapidly that 20 per cent of the fluoride can be found in the urine after three hours (**Figure 4**). Using ^{18}F Carlson et al (1960b) and Ericsson (1958) found that up to 30 per cent of a 1 mg dose was detectable in the urine in four hours. The very rapid rate of excretion is one of the most protective factors in severe fluoride poisoning (death may occur within four hours). The critical period is short since fluoride is rapidly removed from the blood stream and extracellular fluid via the kidney, and skeletal deposition is extremely rapid.

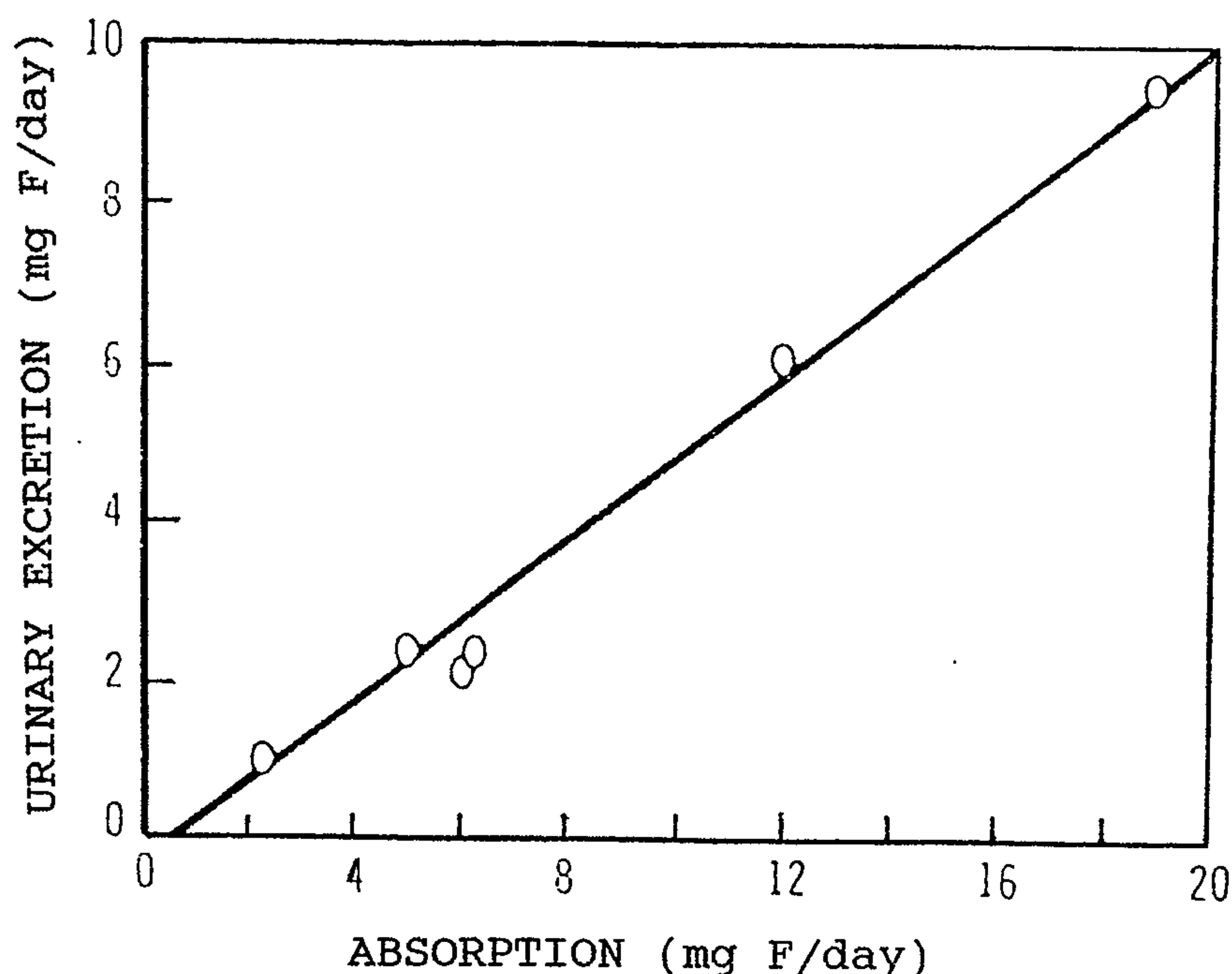
Figure 4 Cumulative secretion of fluoride in the urine of man following oral administration of sodium fluoride.

Source: WHO (1970)



In the relatively unexposed individual about half a single dose of fluoride is excreted in the urine in the following 24 hours and about half is deposited in the skeleton. Largent (1960) collected samples of everything he ate and drank and of everything he excreted, over a period of several months, so that he was able to make definitive measurements of the retention of fluoride when daily doses of 1-18 mg of fluoride were ingested. This fluoride balance study done by Largent showed that fluoride retention followed closely a simple straight line indicating storage of 50 per cent of the absorbed fluoride (Figure 5).

Figure 5 Relation between absorption and urinary excretion of fluoride.
Source: WHO (1970)



Younger individuals, who have a greater proportion of their skeleton available to the circulation and who are actively laying down bone mineral, excrete a lower percentage of a given dose of fluoride than do adults. Infants excrete 40 per cent of the fluoride ingested daily (Ham and Smith 1954). Longwell (1957) reported that children in the London area aged 5-6 years excreted half as much fluoride in the urine as did those aged 10-12 years (0.16 mg per 24 hours compared with 0.35 mg per 24 hours).

3.5 STORAGE OF FLUORIDE

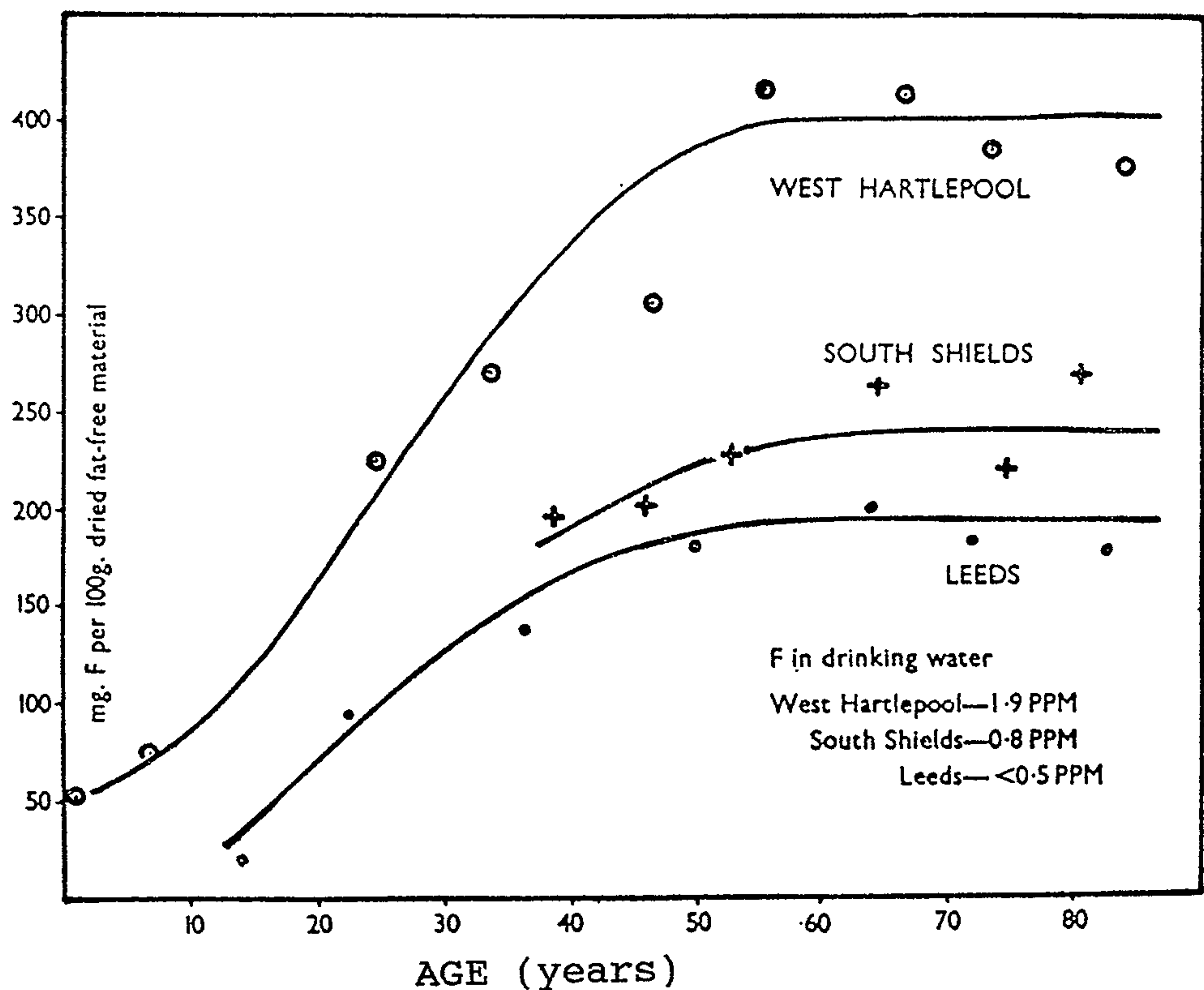
Fluoride is stored in the hard tissues of the body. It has been detected in every specimen of bone or tooth analysed. The extent of fluoride uptake in different parts of the skeleton and dentition depends upon the amounts ingested and absorbed, the duration of fluoride exposure and the type, region and metabolic activity of the tissue concerned. Therefore there is a great disparity in fluoride levels, both between individuals and between different types of mineralised structures. Even within tissues which appear structurally homogeneous, concentrations may vary markedly over distances of only a few microns (WHO 1984).

3.5.1 Fluoride Content of Bone

Fluoride ions are able to enter the hydroxyapatite lattice, and its concentration in living human bone builds up slowly with age reaching a plateau level of fluoride concentration at about the age of 55 years (Smith et al (1953), Jackson and Weidmann (1958). The value of the plateau level depended on the fluoride intake; in West Hartlepool (fluoride content in drinking water is 1.5-2.0 ppm) the plateau level was 4000 ppm, whereas in Leeds (fluoride in drinking water is 0.1 ppm) the plateau level was 2000 ppm (Figure 6 Jackson and Weidmann 1958).

Figure 6 Fluoride content in post-mortem samples of human rib bone in areas with different fluoride levels in the water supply.

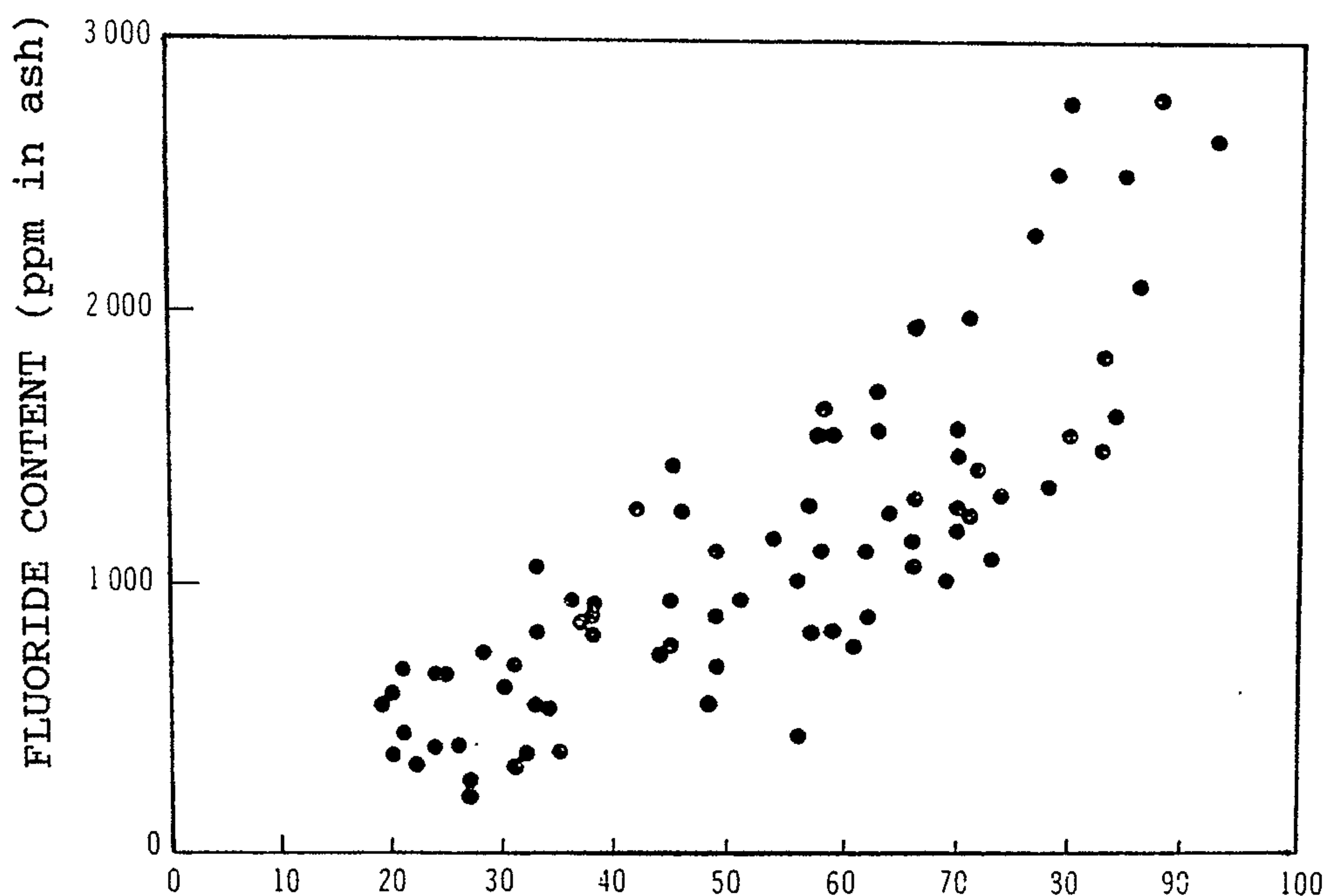
Source: Jackson & Weidmann (1958)



Note: Data on vertical axis is given as mg fluoride per 100g dried fat-free material. Multiply by 10 to get ppm fluoride.

Results of studies by Blayney et al (1962) also showed that the fluoride content of bone was related to the fluoride intake. Weatherell (1966), using compact bone from the femora of persons living in an area containing 0.1 ppm fluoride, suggested that a plateau level was not reached, but the fluoride content increased with age (Figure 7).

Figure 7 Fluoride content of femoral compacta from humans of different ages living in low fluoride areas.
Source: Weatherell (1969)



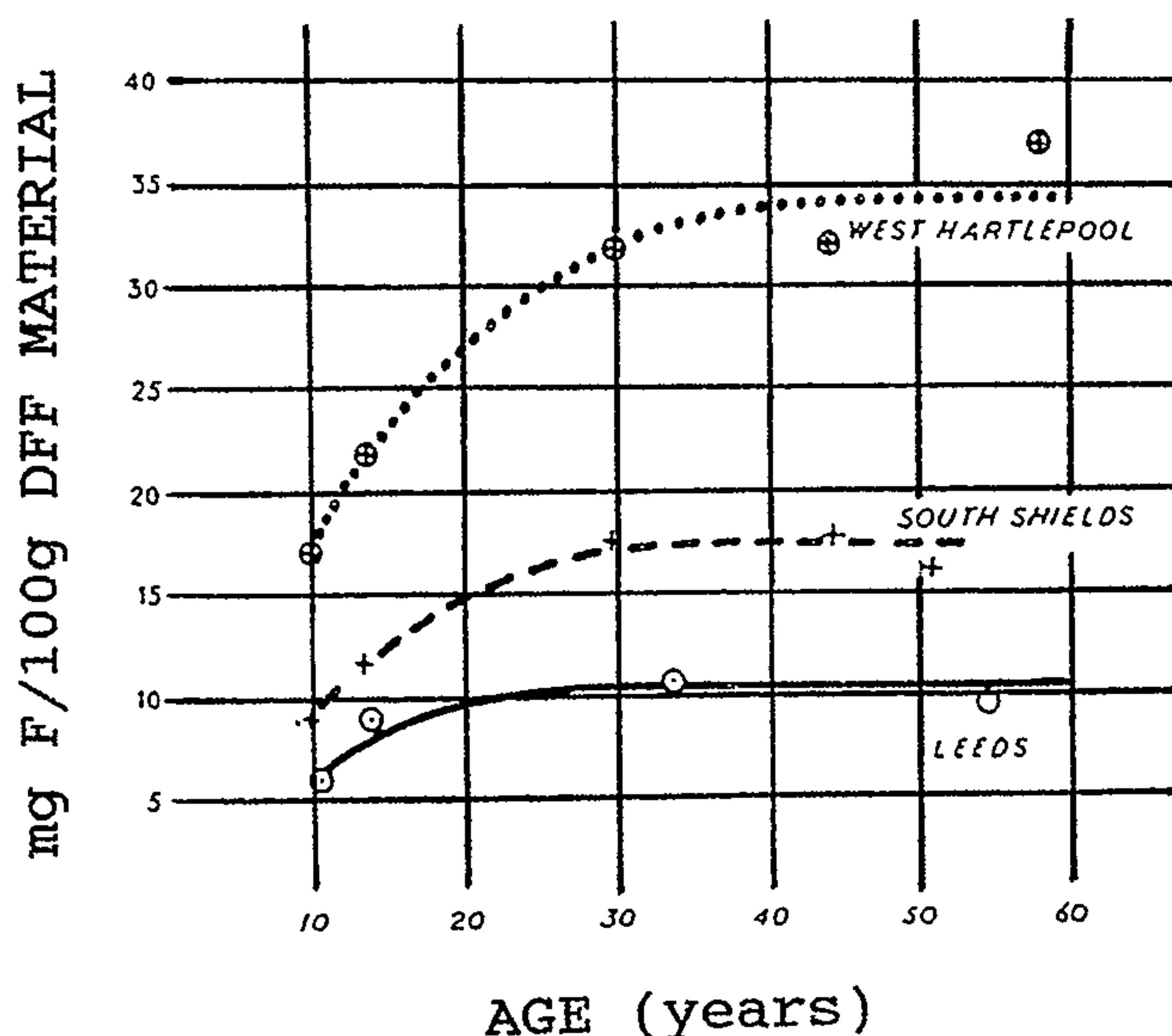
The distribution of fluoride within bone is not uniform. It is highest in those areas of most active growth. Endosteal and periosteal surfaces usually have a higher fluoride content than the central parts of compact bone (Weidmann and Weatherell 1959). Thus, fluoride content of bone increases with age and fluoride content of bone increases with increasing fluoride concentration in the drinking water.

3.5.2 Fluoride Content of Enamel and Dentine

Fluoride uptake in dental tissues increases with age and with increasing fluoride concentration in the water supplies, but the fluoride content of dentine and enamel is lower than that found in bone from the same individual (Murray 1976).

Jackson and Weidmann (1959) showed that in human premolar enamel from West Hartlepool (fluoride = 1.5-2.0 ppm), fluoride concentration increased from 170 ppm fluoride at 10 years and reached a plateau level of 350 ppm at 30 years of age. In contrast, human premolar enamel from Leeds (fluoride = 0.1 ppm) contained 50 ppm fluoride at 10 years and reached a plateau level of 100 ppm fluoride at 30 years of age (Figure 8).

Figure 8 Fluoride content in whole premolar enamel from persons living in areas with different fluoride levels in the water supply.
Source: Jackson & Weidmann (1959)

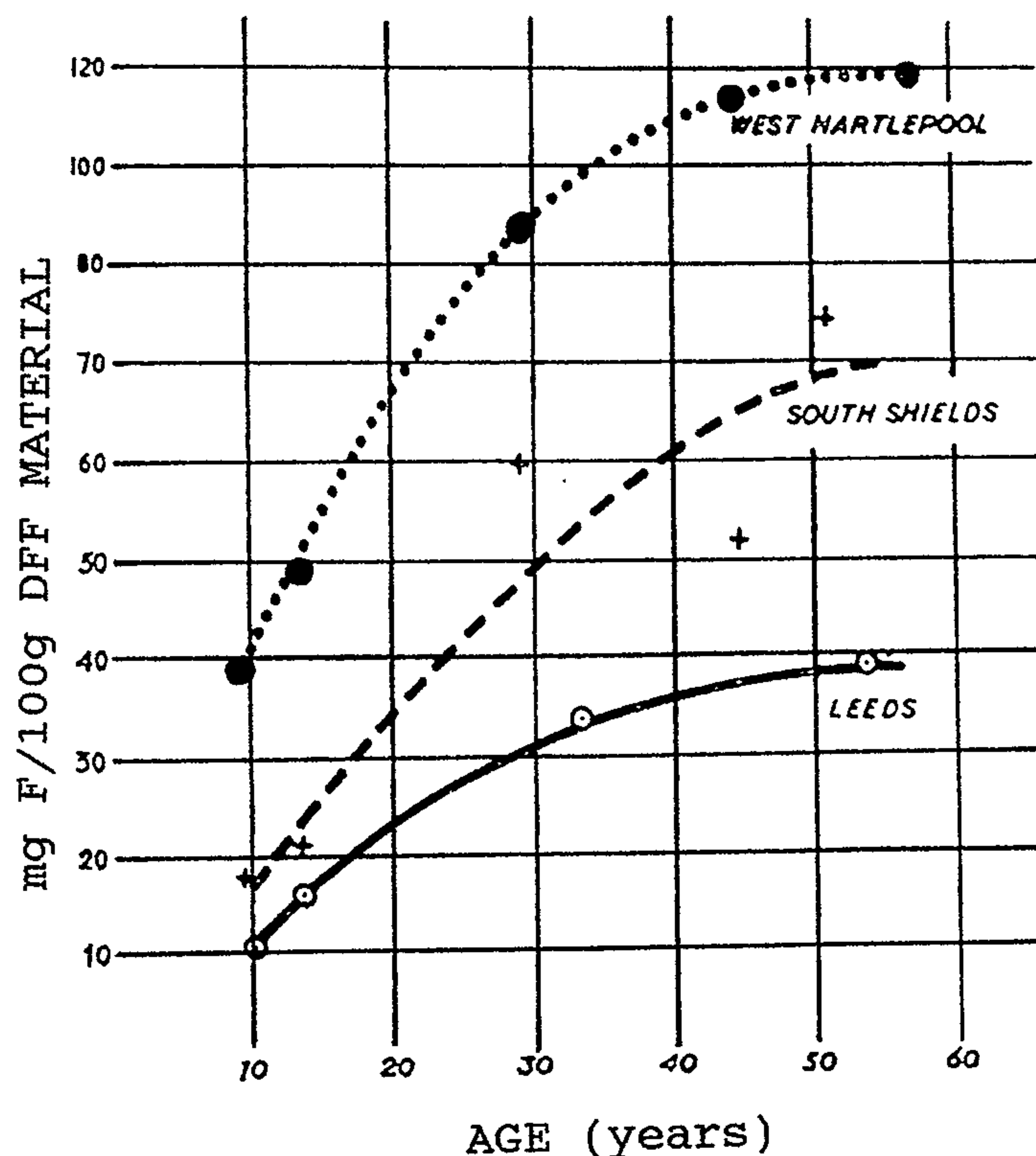


Note: Data on vertical axis is given as mg fluoride per 100g dried fat-free material. Multiply by 10 to get ppm fluoride.

Dentine was found to contain four times more fluoride than enamel. The concentration of fluoride in premolar dentine from West Hartlepool increased from 400 ppm at 10 years to 1200 ppm at 55 years, whereas premolar dentine from Leeds contained 100 ppm fluoride at 10 years and increased to 400 ppm at 55 years (Figure 9).

Figure 9 Fluoride content in whole premolar dentine from persons living in areas with different fluoride levels in the water supply.

Source: Jackson & Weidmann (1959)



Note: Data on vertical axis is given as mg fluoride per 100g dried fat-free material. Multiply by 10 to get ppm fluoride.

However, results of studies using whole enamel and dentine are obscure due to the fact that the fully formed content of these tissues is not uniform. After the tooth has fully formed fluoride is chiefly incorporated at tissue surfaces. The highest concentration of fluoride in dentine is found adjacent to the odontoblastic layer (Yoon et al 1960). The concentration of fluoride in the outer level of enamel is about four to five times higher than in deeper layers. Moreover, this differential was present in unerupted teeth and, at least in low fluoride areas, was independent of age. Isaac et al (1958) concluded that when the fluoride content of the water supply is increased from 0.1 ppm to 5.1 ppm there is an increase in the amount of fluoride deposited in the enamel during tooth formation. Fluoride concentration in the outer layer of enamel in areas with 0.1 ppm fluoride in the water supply increased from 500 ppm fluoride in the under 20-year age group to 1000 ppm fluoride in the over 50-year age group.

In areas with 1 ppm fluoride in the water supply, fluoride concentration in the outermost layer of enamel increased from 900 ppm fluoride in the under 20-year age group to 1600 ppm fluoride in the over 50-year age group. In areas with 5 ppm fluoride in the water supply, the outer level of enamel contained 3200 ppm fluoride and this value did not increase significantly with age, suggesting that this figure represents fluoride saturation of enamel. Studies by Weatherell and Hargreaves (1965) and by Hallsworth and Weatherell (1969) have shown the distribution of fluoride

in enamel and how the concentration of fluorides decreases from the surface inwards to the enamel dentine junction (Figures 10 and 11) .

Figure 10 Distribution of fluoride in enamel of a permanent incisor.

Source: Weatherell & Hargreaves (1965)

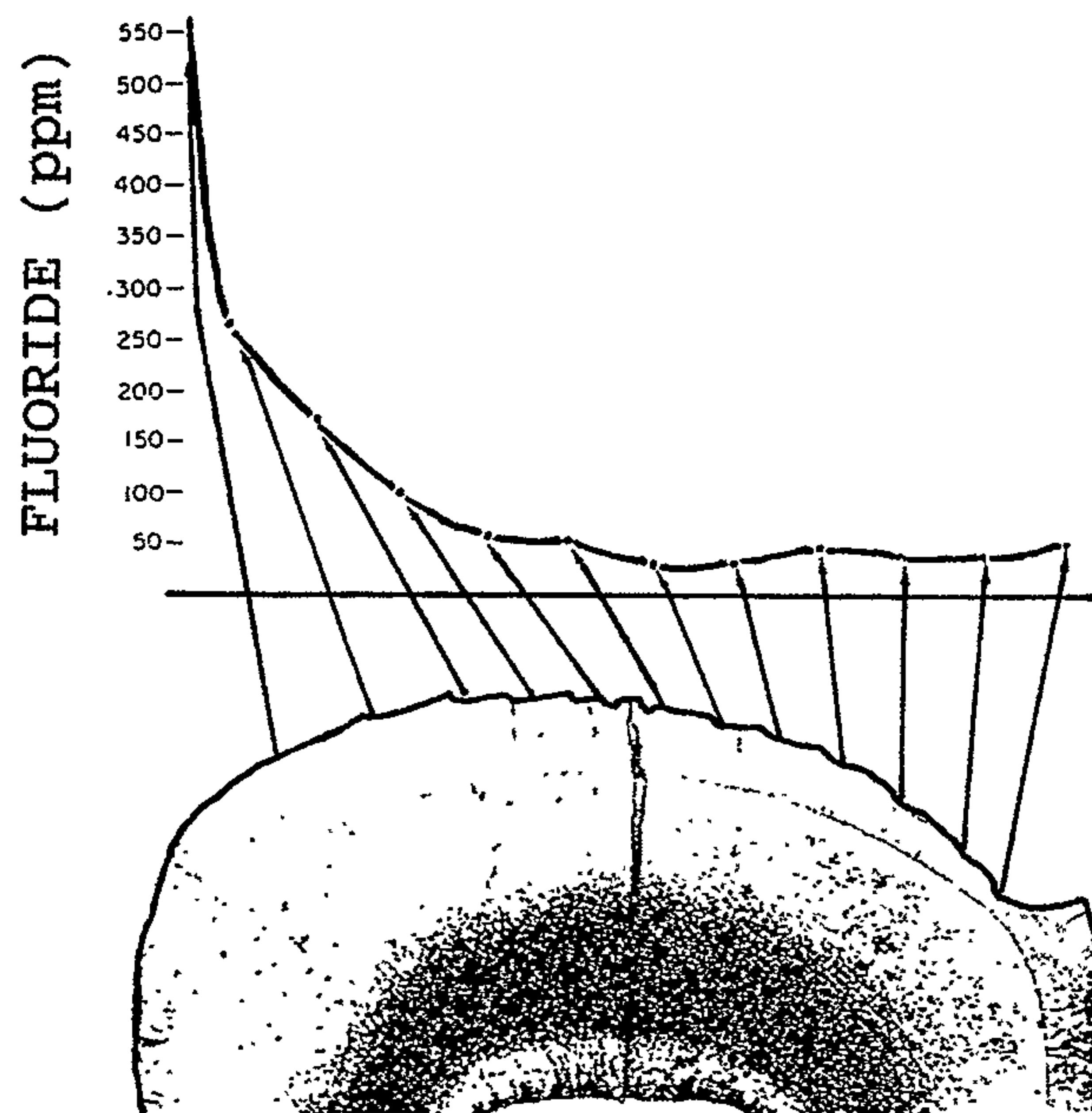
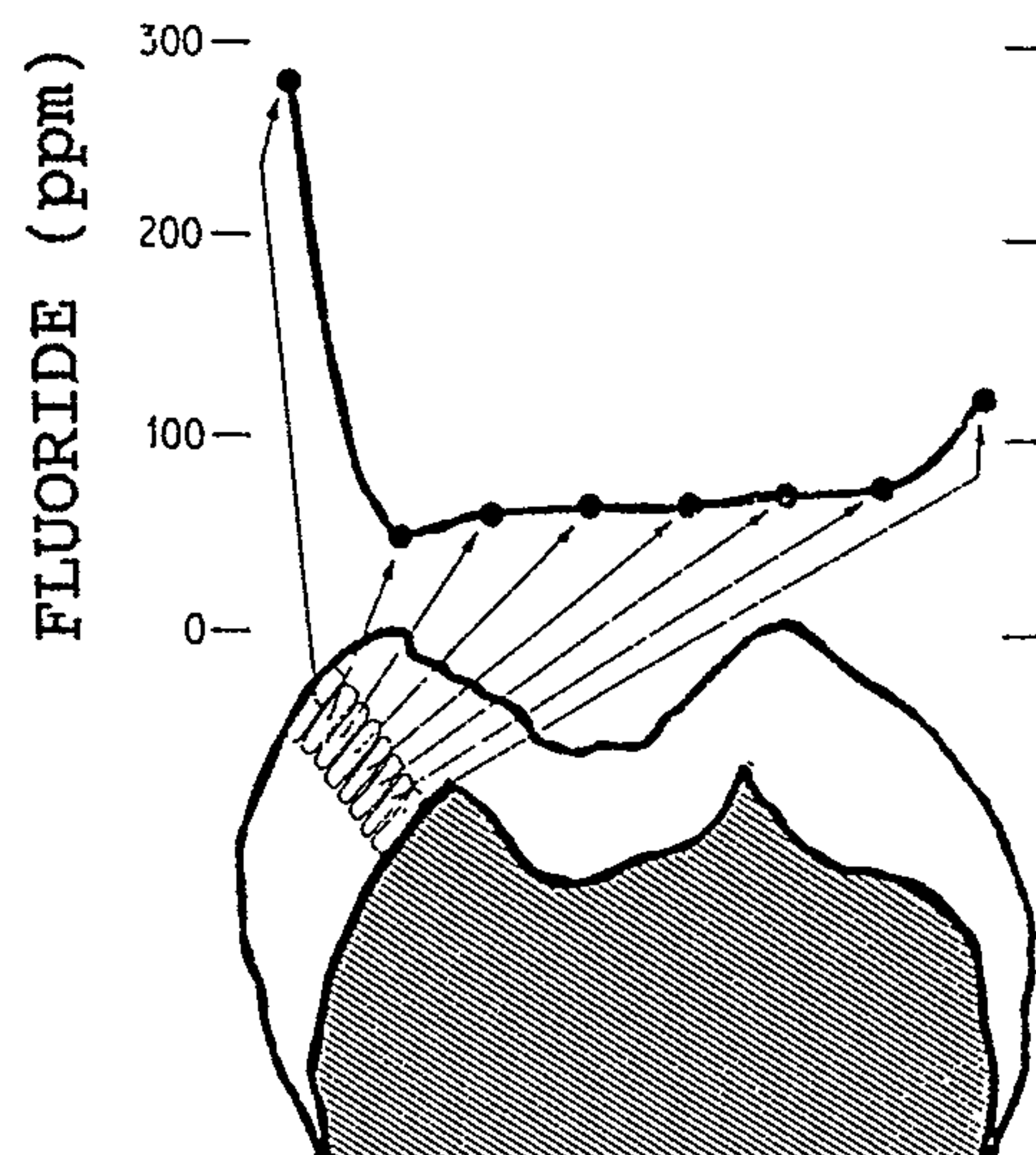


Figure 11 Distribution of fluoride across the cuspal enamel of an unerupted mandibular third molar from a female aged 51 years.

Source: Hallsworth & Weatherell (1969)



Little et al (1967) after examining enamel from people aged 20-50 years living in areas with less than 0.5 ppm fluoride in the water supply, concluded that the marked gradient in fluoride content from surface inward of sound enamel, was found to be primarily limited to discoloured enamel, characterised by a shallow micro radiolucent band just under the surface.

The suggestion that fluoride tended to occur in enamel defects, including early carious cavities, was made by Dowse and Jenkins (1957), who applied ^{18}F to the surface of teeth. In a later report, Jenkins (1963) stated that enamel ground from "white spots" (which he equated with early carious lesions) contained over twice the fluoride content of a similar sample from intact enamel in the same teeth (**Table 3**) and he concluded that under normal physiologic conditions fluoride might accumulate in early cavities and exert its effects just where it could be most beneficial. Similarly, Little et al (1967) suggested that as most of the natural accumulation of fluoride is into the areas of lower mineral content the beneficial effect of fluoride in reducing caries may be in preserving surfaces subject to demineralisation.

**Table 3 Fluoride content of normal and carious enamel
from towns with 0 ppm F and 2 ppm F in the water
supply.
Source: Jenkins (1963)**

	Non-carious Enamel		Carious Enamel	
	0 ppm	2 ppm	0 ppm	2 ppm
F ppm	250	440	640	950
No. of teeth	108	112	108	112

3.5.3 Fluoride in Blood

Estimations of fluoride content of blood are normally made on blood plasma. Approximately three-quarters of the total blood fluoride is in the plasma and one-quarter in the red blood cells (Carlson et al 1960b). Regulatory mechanisms operate within the body to maintain the plasma fluoride content within narrow limits (Smith et al 1950, Singer and Armstrong 1960, 1964). The mechanisms are effective under substantial variations of dietary fluoride intake (Carlson et al 1960a). The regulation of plasma fluoride concentration is due, firstly, to the large volume of extracellular body fluid which dilutes the absorbed fluoride, secondly by deposition of fluoride in the skeleton and thirdly by excretion of fluoride in the urine, and particularly in hot climates, by perspiration (Murray 1976).

The fluoride content of blood plasma of residents in five areas where the fluoride content of drinking water varied from 0.15 to 5.4 ppm has been examined (Singer and Armstrong 1960). In four centres where the drinking water contained 0.15-2.5 ppm fluoride the mean plasma fluoride concentration lay within the range 0.14-0.19 ppm. In the community with 5.4 ppm fluoride in the drinking water, the plasma fluoride concentration was 0.26 ppm. These quantities refer to total fluoride content of plasma. Studies by Taves 1966, Singer and Armstrong 1969 suggested that plasma contains two forms of fluoride: one form is free and ionic, the other is bound to albumin and is non-ionic. It is the free and ionic fluoride, which accounts for 15-20 per cent of the total plasma fluoride, which can be expected to participate in physiological reactions. (Table 4).

Table 4 Normal concentration of fluoride in human blood.
 Source: Singer & Armstrong (1969)

Fluoride Concentration in blood, ppm	Comment (in drinking water supply, ppm)
0.14 (plasma)	0.15
0.15 (plasma)	1.10
0.19 (plasma)	1.10
0.16 (plasma)	2.50
0.26 (plasma)	5.40

3.5.4 Fluoride in Placenta and Fetus

Studies have indicated that human placenta acts as a partial barrier to the transfer of fluoride from maternal blood to fetal blood (Gardner et al 1952, Feltman and Kosel 1955, Ziegler 1956).

Gedalia et al (1961, 1964) showed that when fluoride intake was low (0.1 ppm fluoride), the fluoride content of the placenta was lower than that of maternal or fetal blood, but when fluoride intake was raised, the fluoride content of the placental tissue and of the maternal blood was higher than that of the fetal blood. This suggested that the placenta has a limited permeability to the increased concentration of fluoride ions and that the placenta plays a role in the transfer of fluoride from mother to fetus.

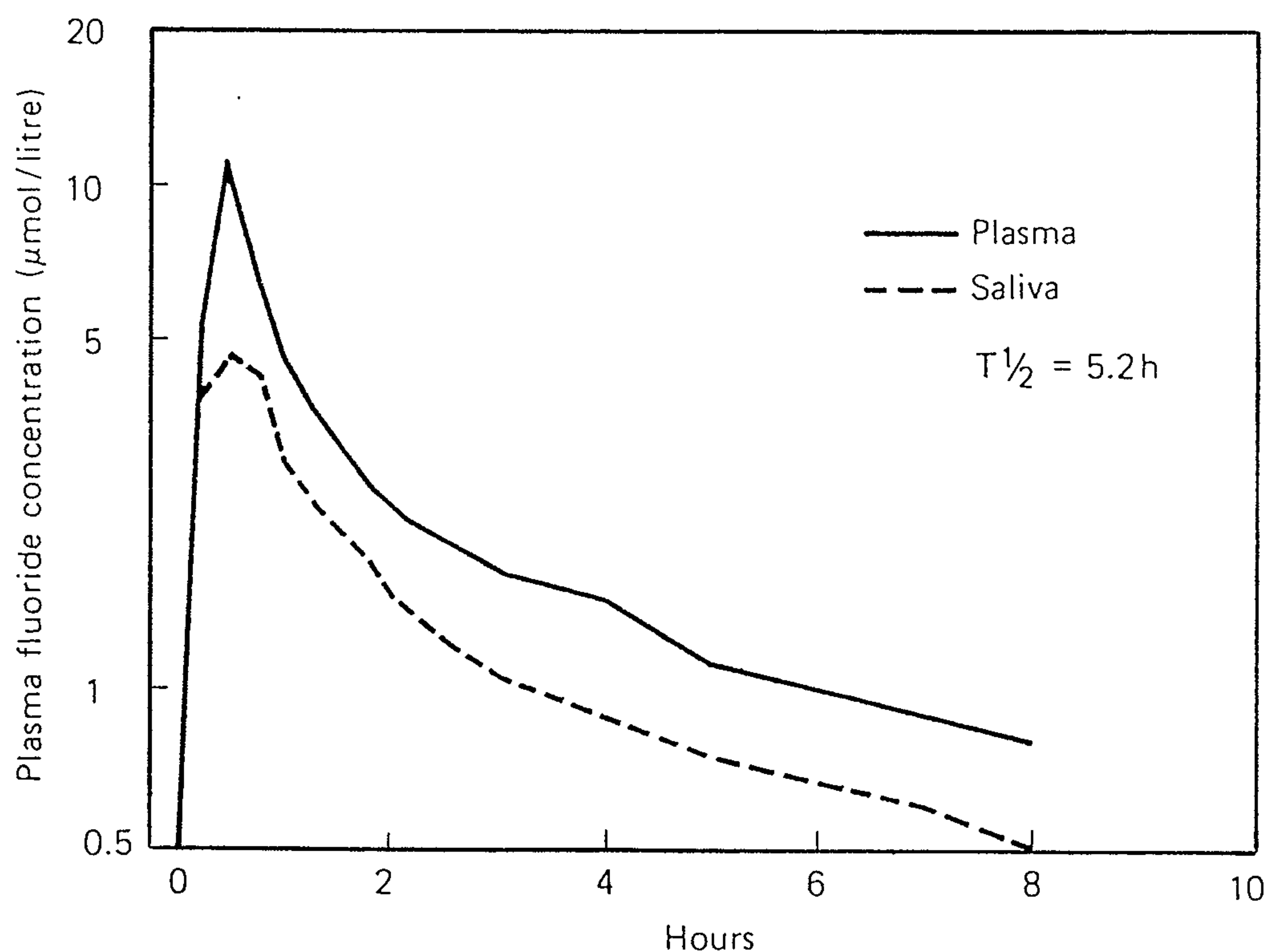
Ericsson and Malmnas (1962) studied the transfer of ^{18}F across the placenta of patients undergoing therapeutic abortions. The ^{18}F content of fetal blood was found to be very low 5-30 minutes after intravenous injection, always less than one-third of the simultaneous ^{18}F concentration in the maternal blood. The ^{18}F concentration of the placenta was between that of the maternal and that of the fetal blood suggesting that fluoride does cross the placenta and is incorporated into fetal bones and teeth undergoing calcification, but the placenta may act as a partial barrier to fluoride, thus protecting the fetus from toxic amounts (Gedalia et al 1964).

3.5.5 Fluoride in Saliva

The fluoride content of saliva has been found to be comparable with plasma levels (**Figure 12**). Carlson et al (1960a,1960b) in their experiments with ^{18}F found that saliva from the parotid duct contained slightly lower concentration than plasma, but showed a similar but slower rise after ingestion of ^{18}F .

Figure 12 Fluoride concentration in plasma and parotid saliva after intake of 3 mg of fluoride as NaF tablets.

Source: Ekstrand et al (1977a)



4 FLUORIDE TOXICOLOGY

"In the dose lies the poison" (Paracelsus).

4.1 CHARACTERISTICS OF FLUORIDE TOXICOLOGY

Fluoride found early use as a "household poison" because rats and cockroaches were susceptible (Newbrun 1986). Fluoride was first added to public drinking water in the 1940s and in the 1950s. Fluorides have been incorporated into tooth paste, other dental products and dietary supplements. The inappropriate use of these products can contribute significantly to total fluoride intake (US Department of Health and Human Services 1991).

Toxicologists classify substances in six categories (toxicity ratings or classes) by probable lethal dose (taken at once) as shown in **Table 5** (Newbrun 1986).

The lethal dose for sodium fluoride in the human for a 70kg adult is 1 to 2 teaspoonsful (5-10 gm); fluoride is therefore classed as "very toxic". It would be expected that no adult who ingested fluoride as (sodium fluoride or another readily soluble salt) in that dose range would survive (WHO 1984, Whitford 1990).

Table 5 Categories of toxicity.
Source: Newbrun (1986)

Category	Probable Oral Lethal Dose (Human)	
	Dose	For 70 kg person (150 lbs)
6 Super toxic	< 5mg/kg	< 7 drops (a taste)
5 Extremely toxic	5-50 mg/kg	7 drops-1tsp
4 Very toxic	50-500 mg/kg	1 tsp-1 oz
3 Moderately toxic	0.5-5 g/kg	1 oz-1pt (or 1 lb)
2 Slightly toxic	5-15 g/kg	1 pt-1qt
1 Practically nontoxic	> 15 g/kg	> 1qt (2.2 lbs)

Toxicologists use a "rule of thumb" to predict roughly what the therapeutic dose of a new chemical may be if only the lethal dose is known. As a first guess, the rule of ten may be used. The rule of ten assumes tenfold increases between asymptomatic, therapeutic, toxic, and lethal doses. The pitfalls of applying this assumption are illustrated in **Table 6**. The rule applies to morphine. Salicylate does not fit the rule; the lethal dose is much less than predicted. Digitoxin is so toxic as to be in a class by itself. Fluoride is much less toxic and lethal than predicted (Newbrun 1986).

Table 6 Magnitude of increase between asymptomatic and lethal dose levels of four well-known substances.
Source: Newbrun (1986)

	Morphine Sulfate (mg)	Salicylate (g)	Digitoxin (mg)	Fluoride (mg)
Asymptomatic	0.3	0.1	0.1	0.1
Therapeutic	1.6-3.0	0.3-3.6	1.0	1.0
Toxic	30-60	7-13	1.2	< 264
Lethal	200-400	30-40	2.5	2500

4.2 ACUTE FLUORIDE POISONING

Knowledge of the quantity of fluoride which, when actually ingested, is likely to cause serious toxic signs and symptoms is important from several points of view. It allows a judgment to be made about what treatment should be given when a known dose has been ingested. Fluoride ion in the acidogastric juice of the stomach becomes un-ionised F molecule and crosses membranes speedily. From the blood, many soft tissues reach their maximal concentrations in minutes (US Department of Health and Human Services 1990).

The lethal oral dose of sodium fluoride for a 70-kg man lies between 5000 and 10,000 mg (1 to 2 teaspoonsful corresponds to 2500-5000 mg of fluoride). Clinical toxicologists class sodium fluoride as a very toxic compound, along with such widely used drugs as aspirin, phenobarbital, boric acid and quinidine (WHO 1984, Newbrun 1986).

The signs and symptoms of acute fluoride poisoning include:

1. extremely severe nausea, vomiting and diarrhoea; blood was often present in the vomitus and stools.
2. abdominal burning and cramping followed by
3. general collapse and shock; pallor, weakness, absent or thready pulse, shallow unlaboured respiration, weak heart tones, wet cold skin, cyanosis and equally dilated pupils
4. death occurs as early as two to four hours

Convulsions were usually absent with paralysis of the muscles of deglutition, carpopedal spasm and spasm of the extremities. Histological findings indicate irritation of the mucosal surfaces, congestion and edema of viscera, dilation of the right cardiac chambers, slight edema of the brain, and no fatty changes in the liver.

Successful treatment of acute fluoride poisoning depends on **early initiation** of the following:

1. cardiac monitoring, preparations for endotracheal intubation and for direct-current cardioversion;
2. prompt and frequent blood analyses, especially for calcium, magnesium potassium, pH;
3. IV infusion of fluids to restore blood volume, of calcium gluconate or chloride, and of magnesium, potassium, and bicarbonate to restore calcium and magnesium levels to normal ranges and to correct other imbalances observed in serum analyses or inferred from electrocardiographic signs;
4. alkaline diuresis to speed up excretion of fluoride;
5. appropriate treatment of severe cardiac arrhythmias.

Favourable response to treatment may be prompt, usually within 24 hours (Newbrun 1986, Whitford 1990).

4.3 CHRONIC FLUORIDE POISONING

In contrast with acute lethal poisoning in man, which is characterised by rapid responses, *chronic* poisoning from exposures up to 100-200 mg daily causes a well-known pattern of abnormalities, especially in bony tissues, depending on the daily fluoride intake and the duration over months or years.

Möller and Gudjonsson's in 1932, described an occupational disease, "osteosclerosis" (radiographically diagnosed), in cryolite workers handling crushed sodium aluminium fluoride mineral in Copenhagen.

Osteosclerosis arising from the prolonged excessive intake of fluoride (osteofluorosis) has been reported from widely distributed geographic areas (USA, Argentina, China, India, the Persian Gulf region, South Africa, and Tanzania). The condition is related directly to the intake of fluoride, which in turn is related to the concentration of fluoride in the drinking water and the amount of water ingested. Nutritional status, presence of other diseases, and whether or not the individual performs hard physical labor are frequently associated with the appearance of osteofluorosis, but it is not known if these confounding factors have a specific role. In Spain, the condition has been described among heavy drinkers of wine to which sodium fluoride had been added as a preservative (WHO 1984 and Newbrun 1986).

In the USA, detectable but not severe osteofluorosis was seen in about 10 per cent of the population who had resided for at least 15 years in Bartlett, Texas (8 ppm fluoride in the water). A few instances of osteofluorosis have been seen in persons using water supplies containing 4 ppm.

Singh et al (1963) reported that the drinking water in one Indian village contained 16 ppm fluoride. Hot (115°-120° F) days of heavy labor demanded an average daily intake of 5 litres of water. Although total intake was not estimated, just the water would contribute up to 84 mg fluoride daily, so that "the total fluoride is enormous." From shallow wells, if the water were boiled, unknown amounts of fluoride might be added from suspended particulates of mud (granite and auorite) containing 0.1 per cent fluoride.

The effects of fluoride ingestion are dependent on the dose and the duration of intake. These effects are summarised in Table 7.

Table 7 Summary of the effects of fluoride ingestion.
Source: Newbrun (1986)

Concentration or Dose of Fluoride	Medium	Time	Effect
0.5 ppb (0.0005 ppm)	Air	Lifetime	Limit for susceptible plants
1 ppm	Water	Lifetime	Less dental caries ^a
≥ 2 ppm	Water	During tooth formation	Dental fluorosis ^a
5 ppm	Water or air	Yrs	No osteosclerosis ^a
8 ppm	Water	Yrs	10% osteosclerosis ^a
≥ 20-80 mg/day	Water or air	Yrs	Crippling fluorosis ^a
40 ppm	Feed	5 Yrs	Body weight loss ^b
50 ppm	Food or water	Yrs	Thyroid changes ^c
100 ppm	Food or water	Mos	Kidney injury ^c
2.5-5.0 gm	Acute dose	2-4 Hrs	Death ^d

^a Man

^b Cattle studies

^c Animal studies

^d 70-kg adult man

5 PREVALENCE OF DENTAL FLUOROSIS

A variety of factors have been identified which are thought to alter the extent to which individuals respond to the toxic effects of fluoride on mineralising tissues. Some of the differences in susceptibility of individuals within the same population can be explained, of course, by "biological variations" between individuals (Szpunar and Burt 1987).

After the considerable research on dental caries and dental fluorosis that occurred during the 1930 to mid 1950s period, little epidemiologic research on the occurrence of dental fluorosis has been carried out, especially in areas with optimal or above optimal level of fluoride in water. Typical examples are Turkey, Syria, Jordan, Egypt, Libya, Algeria, Morocco, Sudan, Tanzania, Iran, Iraq, Afghanistan, and northern Thailand and China (Szpunar and Burt 1987).

The ingestion of fluorine through drinking water and food, by Indians, like other population groups in various parts of the world, is well established. Fluorine has been found in drinking water from various parts of India. The relationship of fluorine ingestion and dental fluorosis or skeletal fluorosis has been established during the last 25 to 30 years. In 1957, manifestation of fluorine ingestion in India was observed in the Nellore district South of India. This observation showed stiffness and pain in the spinal regions and in various joints in some of the residents of certain villages. The kidney function was

impaired in a majority of these cases and the urine contain amounts of fluorine much above the upper normal limits. The biochemical examination of serum, inorganic phosphate and serum phosphatase indicated favourable conditions for abnormal deposition of bone. The radiographical findings showed excessive calcification of tendon, ligaments and fascia, the production of osteophytic formations from various bones and almost complete synostosis of various joints especially of the vertebral column. The degree of disability and the time of onset of the symptoms of the disease were related to the concentration of fluorine in the water and length of time of ingestion, with a concentration of fluorine in drinking water of 9.2 to 11.8 ppm. The symptoms of fluorine intoxication also appeared in immigrants, who had resided in this area from one year to four years (Shourie 1957).

The prevalence of dental fluorosis in different areas in Sri Lanka indicated that, in endemic zones, the prevalence of dental fluorosis among children age 12, was 51 to 78 per cent while in non-endemic area it was 5.4 per cent. There were no significant sex differences in the prevalence of dental fluorosis (Warnakulasuriya et al 1990).

The prevalence of dental fluorosis in Kenya among children 12-15 years of age, with 2 ppm fluoride in their drinking water was 100 per cent, 95 per cent of all teeth exhibit a marked opacity or appear chalky white and 50 per cent have loss of the outermost enamel in irregular areas, and

less than half the surface is so involved (Baelum and Fejerskov 1986). The remaining intact enamel is opaque. This study showed the relationship between the exposure to fluoride from drinking water and the increase in the prevalence of dental fluorosis (Manji et al 1986a).

Norms for an optimal dosage of fluoride, in terms of total intake, as well as in drinking water and dietary supplements, were established empirically. Original recommendations for dietary fluoride supplementation were based on an attempt to mimic, by dietary supplementation, total fluoride intake that would occur naturally in an optimally fluoridated community. The dosage schedule for dietary fluoride supplements by the American Academy of Pediatrics came as a result of the observation of the relationship between dietary fluoride supplementation at the higher levels and objectionable amounts of dental fluorosis (Leverett 1986).

Aasenden and Peebles in 1974 looked at the effect of consuming dietary supplements on various parameters, including dental fluorosis in permanent teeth. Their observations were pivotal in the subsequent decision to reduce the recommended dosage of fluoride for children from birth to age two years (Leverett 1986).

The availability of fluoride from multiple sources has raised question about the potential for increases in the incidence of dental fluorosis. Aasenden and Peebles in 1974 found that 67 per cent of the children who ingested

0.5 mg fluoride from birth to three years of age and 1.0 mg daily thereafter had very mild to mild dental fluorosis and 14 per cent had moderate fluorosis (Kumar et al 1989).

Leverett in 1986 suggested that fluorosis has become more prevalent among children in the USA, in both fluoridated and non fluoridated areas, as a result of ingestion of fluoride from sources other than drinking water. Sources included certain infant foods and formulas, fluoride containing dentifrices, and dietary fluoride supplements. Dietary fluoride supplements were first accepted by the US Council on Dental Therapeutics in 1958. They represent an alternative source of fluoride that can be made available to children who don't have access to optimally fluoridated water supplies (Woolfolk et al 1989).

Prevalence of dental fluorosis in children aged 8 to 16 years who were lifelong residents of areas with negligible, optimal, and above optimal fluoride concentrations in drinking water presenting with severe fluorosis, also have a significantly higher caries experience than do children with lesser degrees of fluorosis. Additional evidence showed that teeth with severe fluorosis have higher percentages of carious lesions and restorations than do teeth with lesser degrees of fluorosis or without fluorosis (Driscoll et al 1986).

Studies in East Africa, Kenya, have indicated that populations living at relatively high altitudes are more susceptible to the toxic effects of fluoride in developing

dental fluorosis. This is a new factor which has been shown to be associated with a higher prevalence and severity of dental fluorosis in very low fluoride areas. People living in an area with 0.04 ppm F exhibit a high prevalence of dental fluorosis. As the areas in which these unexpected findings occurred tended to be in warmer climatic zones, people in such zones may consume more water each day than those living in more temperate climate (Manji et al 1986b, Fejerskov et al 1988).

6 ETIOLOGY, APPEARANCE AND PATHOGENESIS OF DENTAL FLUOROSIS

6.1 ETIOLOGY

The cause of fluorosis is the ingestion of high amounts of fluorine during the period of formation of the teeth. The main source of fluorine ion which causes the teeth to be affected with mottled enamel is drinking water. With the consumption of water containing less than 0.5 mg/l fluorine ion there is absolutely no fluorosis damage to the teeth. With water containing from 0.8 to 1.2 mg/l fluorine ion, the teeth are affected with fluorosis in 10-12 per cent of the population. With a concentration from 1.2 to 1.5 mg/l, 20-30 per cent of the people are affected, with a fluorine content from 1.5-1.8 mg/l 40 per cent, and with the concentration in the water over 2-2.5mg/l more than 50 per cent of the population is affected. With an even greater increase in the fluorine content, the per cent of those affected with fluorosis increases sharply, almost the entire population consuming water from this sources (Gabovich and Ovrutskiy 1969).

6.2 APPEARANCE

The severity of dental fluorosis increases with the amount of fluoride ingested during the period of tooth formation. The first signs of fluoride-induced enamel changes appear as fine striae of accentuated perikymata, evenly distributed over the surface and most easily detected by viewing the tooth surface tangentially. In more affected teeth, the fine lines become broader and more pronounced, occasionally merging to produce irregular, scattered, cloudy or paper white areas. Whenever such scattered opacities contrast with the surrounding enamel, they may resemble the so-called idiopathic enamel opacities. With increasing severity the irregular opaque areas merge until extensive areas appear chalky white. Pitting can occur, either as a considerable number of minute depressions, or as single or multiple, circular holes indicating a loss of the outermost surface enamel. Horizontal bands of circular pits are often observed which, in more severely affected teeth, tend to merge into large "corroded" areas. In severely damaged teeth, almost the entire enamel surface appears to be "corroded"; there is an extensive loss of surface enamel, a part from a cervically intact rim of enamel which is markedly opaque. Varying degrees of discolouration can be seen, but as this is somewhat dependent upon post-eruptive events, it should not be used as part of a clinical score system. In general, increased levels of dental fluorosis are accompanied by a reduced resistance of abrasion which is often very extensive in high fluoride areas (Fejerskov et al 1977).

Dental fluorosis is a subsurface hypomineralization of dental enamel (Baelum et al 1986). The degree of hypomineralisation or tissue porosity combined with the depth of the porous enamel determines the clinical appearance of the fluorotic tooth. The surface defects of the enamel which may be seen in severe cases of dental fluorosis have been considered a direct toxic effect of fluoride on the secreting ameloblasts and hence designated hypoplasia. However, pits are posteruptive changes resulting from the fact that the teeth following eruption are subjected to a variety of traumas. Depending on the degree of preeruptive hypomineralisation the teeth may develop surface damage to a varying extent. Light and electron microscopic studies have shown that the pits are not hypoplasia (Baelum et al 1986).

Dental fluorosis comprises areas of diffuse hypomineralisation, or porosity in the subsurface enamel deep to a well mineralised surface layer. In severe cases many regions of the enamel exhibit 25 per cent or more pore volume located deep to the enamel surface (Fejerskov et al 1975).

The qualitative microradiography has been unable to demonstrate the changes occurring in dental enamel exhibiting slight degrees of dental fluorosis. Polarised light microscopy, is able to detect a large variety of minute changes when used in a combined qualitative and quantitative way. With increasing severity of fluorosis

the porous changes extend deeper into the tissue and a definite highly mineralised surface zone is observed. The changes are not restricted to the outer third of the enamel, although it is in this part that the most severe hypomineralisation or porosity is found. A widening of the innermost microporous part of the enamel indicates that the entire enamel is affected by the fluoride (Thylstrup and Fejerskov 1977).

The lesions in primary teeth appear similar to those described for permanent enamel. From a pathogenic point of view this is of great importance, particularly as the primary incisors are affected as well. This is of interest as it supports that placenta dose not act as a barrier. However, dentin is not affected in human dental fluorosis. Baud and Alami in 1970 described the presence of hypomineralised layers parallel to the counter line in the dentin, although the dentin is apparently normal in many fluorosed teeth which present enamel changes, furthermore extensive area of interglobular dentin are often recorded (Fejerskov et al 1977).

6.3 PATHOGENESIS

The pathogenesis of fluorosed enamel is most often described as a direct effect of fluoride ions (Weatherell et al 1975).

Although the effect of fluoride leading to dental fluorosis may be of very complex character, some basic possibilities in terms of pathogenic mechanisms should be considered. The principal stages which may be affected by fluoride are:

1. Effect on ameloblasts
 - a. Secretory phase: diminished matrix production, change of matrix composition on possible ion transport mechanisms.
 - b. Maturation phase: diminished withdrawal of protein and water on possible ion transport mechanisms.
2. Effect on nucleation and crystal growth in all stages of enamel formation.
3. Effect on calcium homeostasis with dental fluorosis being an indirect result.

(Fejerskov et al 1977)

Most of the hypotheses on pathogenic mechanisms in dental fluorosis are based on animal experiments, receiving a wide range of fluoride doses.

6.3.1 Effect of Fluoride on the Secretory Stage in Amelogenesis

The effect of acute and chronic doses of fluoride on dental hard tissue formation was studied by Neiman and Elsenmann 1975, Patterson and Kruger 1975, and Shinoda 1975.

Acute doses of fluoride have given rise to a zone of

hypermineralisation in both dentin and enamel. However, fluoride differs from all other ions by creating a hypermineralised zone followed by the zone of hypomineralisation resulting from a disturbance of the function of the secretory ameloblast by the fluoride.

In acute fluorosis, in animals that had been given a single injection of 0.3 ml of 2.5 per cent sodium fluoride, the changes were observed in the posterior region of the incisor confined chiefly to the ameloblastic layer, which was in the secretory stage. These cells contained globules which stained intensely with haematoxylin and were located at the irregular surface of the organic enamel matrix (Fejerskov et al 1977), thus the hypermineralised zone is a result of instant formation of apatites in all mineralising tissues caused by the high concentrations of fluoride in plasma immediately following injection (Larsen et al 1976).

6.3.2 Effect of Fluoride on the Enamel Matrix

Experimental data indicate that fluoride may interact with the cellular processes during enamel matrix secretion. However, biochemical and ultrastructural data from chronic fluoride experiments are needed to ascertain whether the pathogenesis of dental fluorosis can be explained in this way only (Fejerskov et al 1977).

6.3.3 Effect of Fluoride on the Maturation Stage in Amelogenesis

There is little known about the possible effect of fluoride on the cellular events taking place during the maturation stage in amelogenesis, impairment of the processes in this stage may lead to a type of lesion similar to dental fluorosis. Allan in 1963 suggested that these are the result of a direct fluoride effect on the complex ameloblast function during maturation, or on crystal growth. Also fluoride may be present in much higher concentrations during certain stages of amelogenesis, this means that those parts of enamel which are in the stage of enamel maturation for the longest period of time may be the most susceptible to exhibit fluoride dependent changes. (Weatherell et al 1977)

6.3.4 Effect of Fluoride on Dentin Formation

Dentin exhibits striations, hypoplastic defects, hypomineralised interglobular spaces, and in severe cases gross deformations of the external outline of the dentin. The number and severity of these changes increase with increasing levels of fluoride and with increasing time of experiments. Dentin in human fluorosed teeth exhibits changes compatible with those of experimental animals, and from a pathogenesis point of view it is therefore important to stress that **dental fluorosis is characterised by changes in both enamel and dentin** (Fejerskov et al 1977).

6.3.5 Effect of Fluoride on Nucleation and Crystal Growth

The porosity of fluorosed human enamel might be a result of a decreased rate of nucleation and or crystal growth. A possible effect of fluoride on crystal growth will take place during both secretion and enamel maturation stages of amelogenesis. The exact mechanisms of mineralisation *in vivo* and the effect of fluoride on these are not as yet explainable from a physiochemical point of view. The decreased nucleation and or crystal growth may be compensated for in dentin due to the continuous mineralisation of this tissue throughout life (Desteno and Feagin 1975).

6.3.6 Effect of Fluoride on Calcium Homestasis

A number of data indicate that both acute and chronic high doses of fluoride may cause a decrease in serum calcium concentration, this leads to a compensatory increase in parathyroid hormone concentration. In human beings it is an open question whether the relatively small doses of fluoride consumed in drinking water are able to induce such effects. These problems are extremely difficult to solve due to biologic variation and analytic limitations (Fejerskov et al 1977).

7 INDICES FOR MEASURING DENTAL FLUOROSIS

Dental fluorosis is a hypoplasia or hypomineralisation of tooth enamel or dentin, produced by the chronic ingestion of excessive amounts of fluoride during the period when teeth are developing. The major determinant of the prevalence and severity of dental fluorosis has been shown to be the concentration of fluoride in the water consumed by infants and children during the first five years of life. Mineralisation of permanent teeth, other than third molars, occurs from about the time of birth until about five years of age. After that time, teeth other than third molars are completely mineralised and can not be affected by fluorosis because of excessive fluoride ingestion (Horowitz 1986).

Although both primary and permanent teeth may be affected by fluorosis, under uniform conditions of fluoride availability, fluorosis is greater in permanent teeth than in primary teeth because:

1. Most of the mineralisation of primary teeth occurs before birth and the placenta serves as a passive barrier to the transfer of high concentrations of plasma fluoride from a pregnant women to her developing fetus, thus controlling, to some extent the delivery of fluoride to the developing primary dentition.
 2. The period of enamel formation for primary teeth is shorter than for permanent teeth and the enamel of primary teeth is thinner and has greater opacity than that of permanent teeth, which makes detection of fluorosis more difficult.
- (Horowitz 1986).

They are several classification system or indexes that record all defects in enamel were developed. These are Dean's Index, Tooth Surface Index of Fluorosis (TSIF) and Thylstrup and Fejerskov Index (TFI).

7.1 DEAN INDEX

In **Table 8** and **Figure 13**, the classification system which Dean recommended in 1942 is shown.

Table 8 The Dean index
Source: Möller (1982)

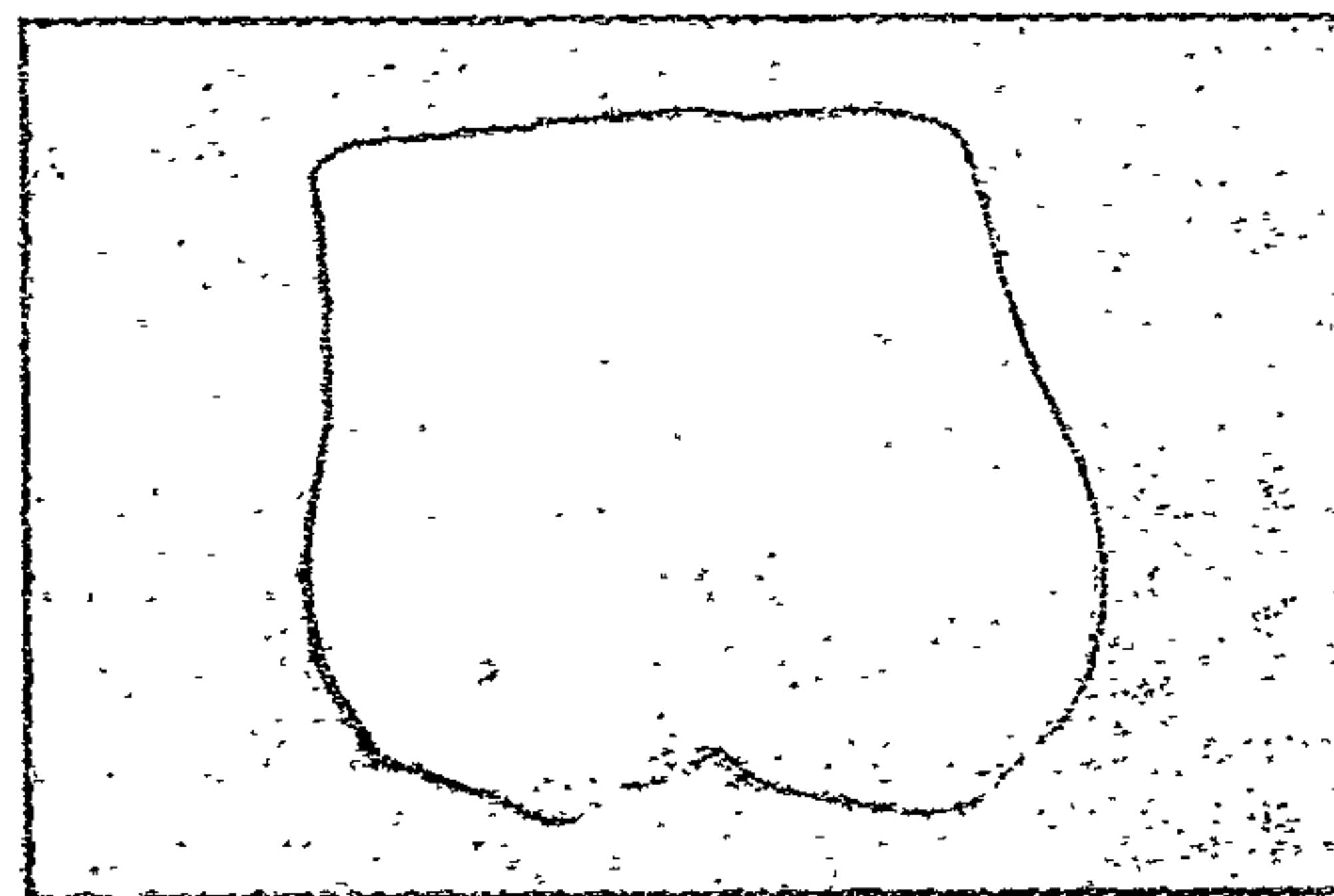
1.1	Normal	The enamel represents the usual translucent semi-vitriform type of structure. The surface is smooth, glossy, and usually of a pale, creamy white colour.
1.2	Questionable	The enamel discloses slight aberrations from the translucency of normal enamel, ranging from a few white flecks to occasional white spots. This classification is utilised in those instances where a definite diagnosis of the mildest form of fluorosis is not warranted and a classification of "normal" not justified.
1.3	Very mild	Small, opaque, paper white areas scattered irregularly over the tooth but not involving as much as approximately 25% of the tooth surface. Frequently included in this classification are teeth showing no more than about 1-2 mm of white opacity at the tip of the summit of the cusps of the bicuspid or second molars.
1.4	Mild	The white opaque areas in the enamel of the teeth are more extensive but do not involve as much as 50 % of the tooth.
1.5	Moderate	All enamel surfaces of the teeth are affected, and surfaces subject to attrition show marked wear. Brown stain is frequently a disfiguring feature.
1.6	Severe	All enamel surfaces are affected and hypoplasia is so marked that the general form of the tooth may be affected. The major diagnostic sign of this classification is the discrete or confluent pitting. Brown stains are widespread and teeth often present a corroded-like appearance.

Figure 13

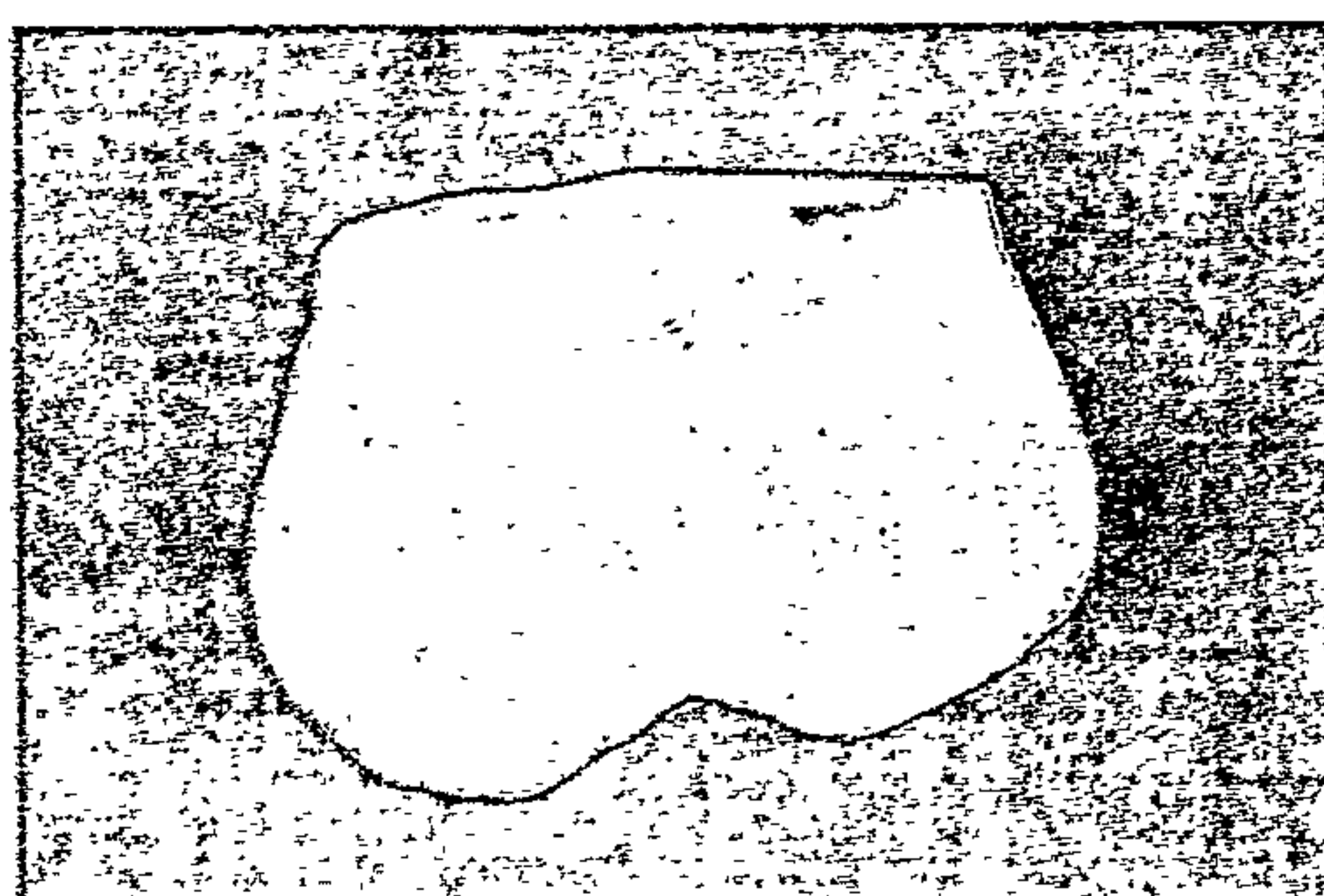
Teeth with dental fluorosis
(Dean's Classification)
Source: Möller (1982)



1.1



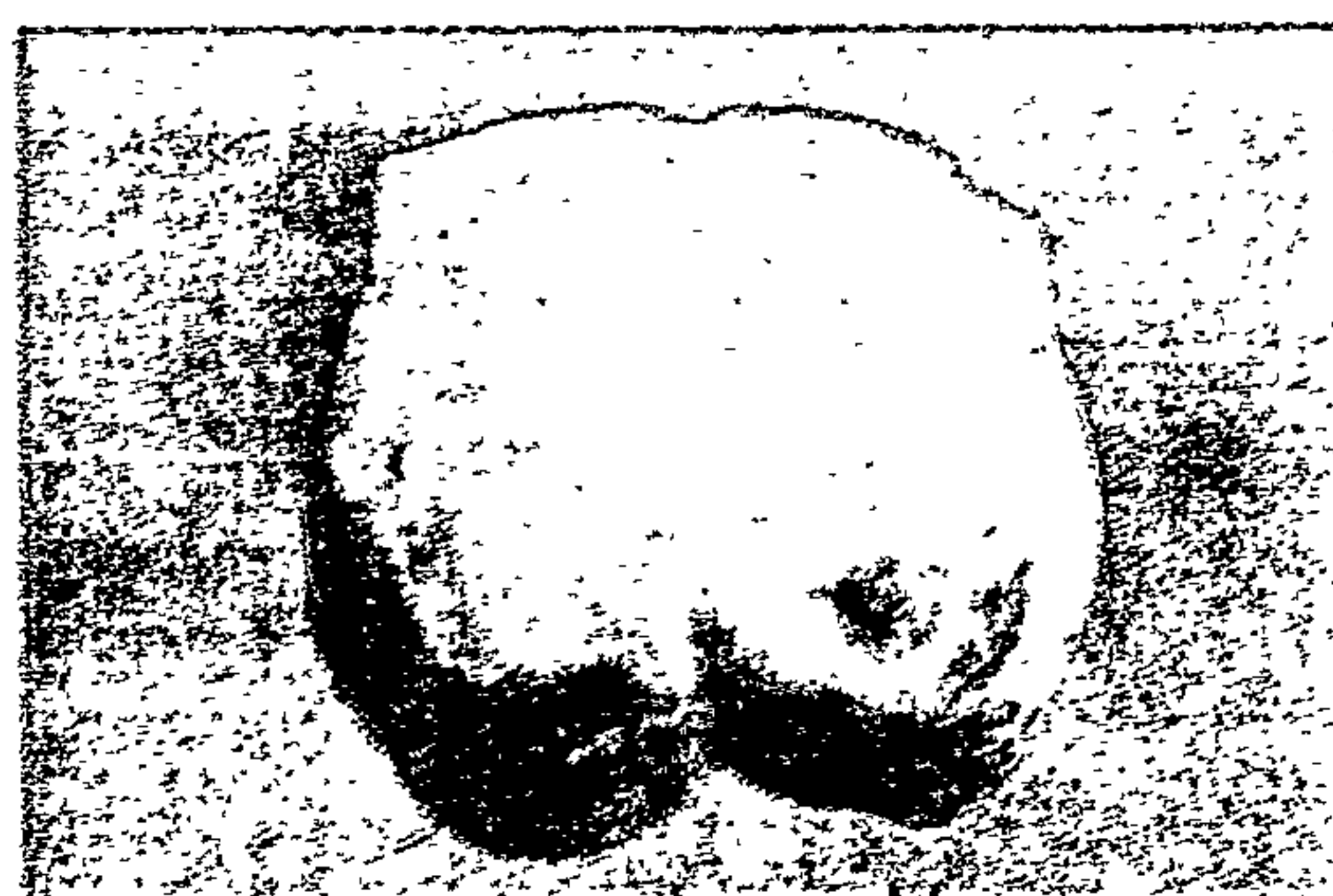
1.2



1.3



1.4



1.5



1.6

7.2 TOOTH SURFACE INDEX OF FLUOROSIS (TSIF)

The Tooth Surface Index of Fluorosis (TSIF) was applied in a survey to assess the prevalence of dental caries and dental fluorosis in communities having optimal and above optimal concentrations of naturally occurring fluoride in drinking water. With the TSIF, a separate score is given to each unrestored anterior tooth surface **from the labial and lingual aspects** and three to each posterior tooth, **from the buccal, lingual and occlusal aspects**. Only fully erupted tooth surfaces are scored with the TSIF, and air is not used to dry the teeth. Because teeth are normally kept moist by saliva, they should be examined in their natural state to determine whether they display fluorosis under condition that more closely approximate normal social interactions. Because the TSIF is not an interval scale, scores are not averaged, they are arranged in various frequency distributions, which can be compared by non parametric statistical test **Table 9**.

**Table 9 Descriptive Criteria and Scoring System for the
Tooth Surface Index of Fluorosis (TSIF)
Source: Horowitz (1986)**

Numerical Score	Descriptive Criteria
0	Enamel shows no evidence of fluorosis
1	Enamel shows definite evidence of fluorosis, namely areas with parchment-white colour that total less than one-third of the visible enamel surface. This category includes fluorosis confined only to incisal edges of anterior teeth and cusp tips of posterior teeth.
2	Parchment-white fluorosis totals at least one-third of the visible surface, but less than two-thirds.
3	Parchment-white fluorosis totals at least two-thirds of the visible surface.
4	Enamel shows staining in conjunction with any of the preceding levels of fluorosis. Staining is defined as an area of definite discolouration that may range from light to very dark brown.
5	Direct pitting of the enamel exists, unaccompanied by evidence of staining of intact enamel. A pit is defined as a definite physical defect in the enamel surface with a rough floor that is surrounded by a wall of intact enamel. The pitted area is usually stained or differs in colour from the surrounding enamel.
6	Both discrete pitting and staining of the intact enamel exist.
7	Confluent pitting of the enamel surface exists. Large areas of enamel may be missing and the anatomy of the tooth may be altered. Dark-Brown stain is usually present.

7.3 THYLSTRUP AND FEJERSKOV (TF) INDEX

According to the the clinical appearance, dental fluorosis may be arranged into ten classes, **Table 10**, ranging from a score of 0 to 9, reflecting increasing degrees of severity. Since, at the time of eruption, all surfaces of a given tooth are equally affected, the classification system need not be applied to all surfaces. The facial (buccal or labial) surface will reflect the degree to which all surfaces of each tooth are affected. This classification is based on the TF index proposed by Thylstrup and Fejerskov (1978).

In order to facilitate the use of this simple index the principle features are shown diagrammatically in **Figure 14** and **Plates 1-14** in the Appendix.

Each single score in the TF index represents a measurement on an ordinal scale and should therefore only be considered as an arbitrary point along a continuum of changes of the enamel. Indeed each score represents a small spectrum of changes associated with dental fluorosis which will be encountered in the course of a survey.

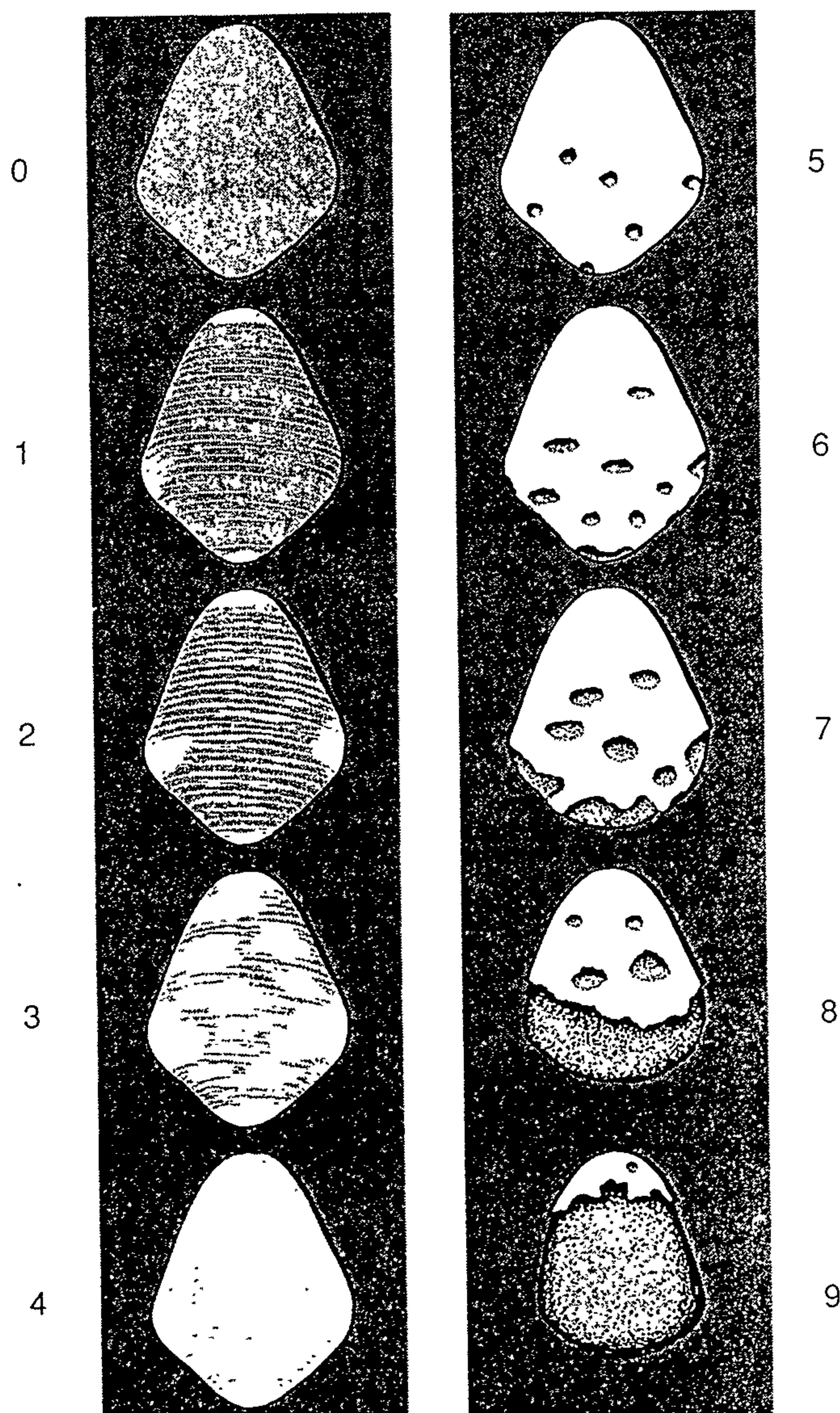
Table 10 **Classification of the characteristic clinical appearance enamel.**

Source: Thylstrup & Fejerskov (1978)

Score	Clinical Appearance
TF score 0	The normal translucency of the glossy creamy white enamel remains after wiping and drying of the surface.
TF score 1	Thin white opaque lines are seen running across the tooth surface. Such lines are found on all parts of the surface. The lines correspond to the position of the perikymata. In some cases, a slight "snowcapping" of cuspal incisal edges may also be seen.
TF score 2	The opaque white lines are more pronounced and frequently merge to form small cloudy areas scattered over the whole surface. "Snow-capping" of incisal edges and cusp tips is common.
TF score 3	Merging of the white lines occurs, and cloudy areas of opacity occur spread over many parts of the surface. In between the cloudy areas white lines can also be seen.
TF score 4	The entire surface exhibits a marked opacity, or appears chalky white. Parts of the surface exposed to attrition or wear may appear to be less affected.
TF score 5	The entire surface is opaque, and there are round pits (local loss of the outermost enamel) that are less than 2 mm in diameter.
TF score 6	The small pits may frequently be seen merging in the opaque enamel to form bands that are less than 2 mm in vertical height. In this class are included also surfaces where the cuspal rim of facial enamel has been chipped off, and the vertical dimension of the resulting damage is less than 2 mm.
TF score 7	There is a loss of the outermost enamel in irregular areas, and less than half the surface is so involved. The remaining intact enamel is opaque.
TF score 8	The loss of the outermost enamel involves more than half the enamel. The remaining intact enamel is opaque.
TF score 9	The loss of the major part of the outer enamel results in a change of the anatomical shape of the surface/tooth. A cervical rim of opaque enamel is often noted.

The range of scores available in the TF index makes it a versatile classification system enabling it to be used to measure the effects of fluoride exposure in populations exhibiting the mildest to the most severe forms of dental fluorosis. The TF index has the benefit of being clear, precise and sensitive for the measurement of dental fluorosis under most circumstances. (Fejerskov et al 1988)

Figure 14 Diagrammatic illustration of the clinical features of dental fluorosis from its mildest form to the most severe. Compare with Plates 1-14 and the description of the TF index.
Source: Fejerskov et al (1988)



8 DIAGNOSIS OF DENTAL FLUOROSIS AND THE PROBLEMS OF DIFFERENTIAL DIAGNOSIS

Differentiating between fluorotic and non-fluorotic defects of dental enamel is an important diagnostic decision in epidemiology and public health dentistry (Fejerskov et al 1988) .

The commonly accepted diagnostic criteria for fluorosis discriminate between non discrete symmetrical and asymmetrical distributions of opacities of dental enamel. These criteria appear to identify most cases of dental fluorosis. However, it is not yet confirmed that the pattern and distribution of dental fluorosis are unique phenomenon. Metabolic, physiological, other trace element, and malnutrition have been reported to induce bilateral symmetrical developmental enamel opacities (Cutress and Suckling 1989). The causes of opacities, hypoplasias and discolouration of human enamel, excluding fluorosis are listed in **Table 11**.

Table 11 Causes of opacities, hypoplasias, discoloration of human enamel, excluding fluorosis.

Source: Pindborg (1990)

Systemic Causes

Genetically determined
Amelogenesis imperfecta as isolated phenomenon.
Amelogenesis imperfecta associated with other lesions. Epidermolysis bullosa, Pseudohypoparathyroidism. Taurodontism, curly hair, sclerotic bones.
Other genetically determined diseases.
Ehlers-Danlo's syndrome.
Chromosomal anomalies, Trisomy 21.
Congenital defects.
Heart disease, Unilateral facial hypoplasia, Inborn errors of metabolism, Galactosaemia, Phenylketonuria, Alkaptonuria. Erythropoietic porphyria. Primary hyperoxaluria. Neonatal disturbances. Premature birth. Hypocalcaemia, Haemolytic anaemia, Allergy, Infectious diseases, Viral, Rubella Bacterial, Syphilis, Tetanus, Neurological disturbances. Tuberous sclerosis. Endocranopathies. Hypothyroidism. Hypoparathyroidism. APEC-syndrome. Diabetes.
Nutritional deficiencies.
Nephropathies.
Nephrotic syndrome.
Infections of urinary system.
Enteropathies.
Coeliac disease. Lymphangiectasia.
Liver diseases, Intoxications, Tetracyclines, Thalidomide. Vitamin D, Pica.

Local causes

Local acute mechanical trauma
Neonatal mechanical ventilation, Falls, Gunshot, Surgery, Ritual mutilation, Electric burn, Irradiation, Local infection, Periapical osteitis, Acute neonatal maxillitis, Regional odontodysplasia, Local infection

8.1 THE DIAGNOSTIC PROCEDURE

Any examination of dental enamel should begin with an overall view of the general features of the teeth present. Examination for dental fluorosis is no exception to this rule. Dental fluorosis almost always affects the entire dentition. All surfaces of an affected tooth are equally affected at the time of eruption into the mouth. Although there may be slight differences in the post-eruptive changes occurring on different surfaces of the same tooth, it is sufficient to record the score only of the facial (buccal or labial) surfaces. Homologous teeth will invariably be affected to a similar degree, though each tooth in the mouth should be classified and its score recorded.

If the examination for dental fluorosis is part of a survey in which dental caries is being recorded, it is recommended that the examination for caries be carried out first, since such an examination usually requires removal of the plaque from the enamel surface before diagnosis. By the time the caries examination of the individual has been completed, the tooth surfaces will usually have dried out sufficiently to enable a proper diagnosis of dental fluorosis (Fejerskov et al 1988).

If the examinations are being carried out only for recording dental fluorosis it will be necessary to clean and dry the buccal surfaces of the teeth using either cotton wool or gauze. The tooth surfaces should then be

allowed to dry for approximately 1-2 minutes before examination starts. Of course, if the examinations are being conducted in a clinic then drying of the teeth may be more rapidly achieved by the use of pressurised air. Cleaning and drying of tooth surfaces is particularly important in areas of low endemicity of dental fluorosis, whereas in studies performed in higher fluoride areas these procedures will not be of so great an importance as the enamel changes will be much more obvious (Fejerskov et al 1988). Once the teeth have been dried it is not vital that the teeth remain dry as the rate at which saliva regains access to the pores of the enamel is quite slow.

The importance of plaque removal for the diagnosis of milder forms of dental fluorosis, is that, the removal of plaque will result in the disclosure of a much more extensive involvement of the enamel surface than was apparent before the plaque was removed (**Plate 19 and Plate 20**).

Plate 21 and Plate 22 show the importance of drying the enamel surface. In Plate 21, the tooth was photographed immediately before drying and in Plate 22, approximately 1 minute after, with the result that the surface opacities were markedly enhanced. Drying of the tooth surface, especially when dealing with the milder forms of dental fluorosis, transforms the diagnostic process from being a hazy guess to a precise diagnosis and classification. Once the facial surfaces of the teeth have been wiped clean and dried, each tooth present is scored (Fejerskov et al 1988).

Incisors are the least, and the premolars amongst the most, severely affected teeth in the mouth. Therefore, when conducting surveys to obtain rough estimates of the prevalence and severity of dental fluorosis, it is possible to record data only for the upper central incisors and upper first or second premolars. However, this method is not recommended since the recording of data for all teeth is not a laborious procedure (Fejerskov et al 1988).

8.2 POST-ERUPTIVE CHANGES

In the milder forms of dental fluorosis, mechanical attrition, resulting from the use of abrasive toothpaste, will overtime cause an apparent "remission" of the fluorotic lesion most likely due to surface enamel removal. The upper incisors are particularly susceptible to such environmental modification. The consequence is that there sometimes seems to be a gradual "regression" of the degree of severity of the milder forms of dental fluorosis (TF scores 1 to 3 and, sometimes, 4) after the tooth has erupted into the mouth (Fejerskov et al 1988).

Teeth which at the time of eruption exhibit extensive porosities (TF score 4) may, shortly after eruption, become pitted or experience extensive flaking off of the surface enamel. The resulting features are characteristic of TF scores 5 to 9 (**Plates 8-14**).

A further characteristic of post-eruptive changes in the more severe forms of dental fluorosis is the tendency for

occlusal surfaces to experience marked attrition. This is because porous hypomineralised enamel can easily become worn away by occlusal forces (**Plate 23**). It is important to be aware of this fact as the hypomineralised enamel on the facial surface, bordering with the occlusal surface, frequently flakes off as well. Such loss of enamel near the cusps has resulted partly from physical forces at the occlusal surface. Indeed, the same forces may also cause pitting to occur further away from the occlusal surface.

Attrition at proximal sites of the teeth often results in the development of wear facets. As the exposed porous enamel at these sites frequently becomes stained, the discoloured enamel may sometimes be mistaken for proximal carious lesions.

The discolouration of the upper central incisors exhibiting TF score 3 may occur, especially on the mesio-incisal part of the facial surface. Such discolourations are often more pronounced in TF scores 4 and above as there is often marked wear of the incisal part of these teeth, (**Plate 24**). Moreover, the labial surface of the upper central incisors are frequently discoloured in cases where incomplete lip closure exposes the lower part of the incisors to the air. The dried portion of the tooth surface often becomes rapidly stained. The resulting brown stains on the labial surface of the incisors is often taken by both lay persons and inexperienced health workers as the principal characteristic of "real" dental fluorosis.

In some populations having severe dental fluorosis, it is customary, especially for girls, to grind the discoloured or disfigured facial surfaces of the upper incisors for cosmetic reasons (using either some kind of abrasive or files) to remove the discoloured, porous, outer surface enamel. The resulting induced post-eruptive changes which such teeth exhibit (**Plate 25, Plate 26**) may make it difficult for an examiner to diagnose or classify the affected surfaces appropriately.

The grinding of the facial surface of the teeth usually involves the removal of the incisal two-thirds of the outer porous and stained enamel. This grinding eventually exposes the underlying less porous enamel. However, the border between the inner almost normal enamel and the outer fluorotic enamel is gradually moved towards the cervical margin, resulting in a half-moon shaped area of exposed porous enamel that rapidly takes up stains from the mouth. It is this combination of features which give these particular types of induced post-eruptive changes their characteristic appearance.

Where such teeth are encountered, it may be best not to assign a score to the surface, as there is no way of being sure how severely affected the enamel surface was before the grinding was carried out (Fejerskov et al 1988).

8.3 PROBLEMS OF DIFFERENTIAL DIAGNOSIS

8.3.1 Non-Fluoride Opacities

The possibility of diagnosing the early stages of dental fluorosis with any certainty is difficult (Jackson 1961, Small and Murray 1978, Suckling and Pearce 1984).

Some of these difficulties may include enamel opacities of non-fluoride origin (Fejerskov et al 1988). However, if the criteria of the TF index (TF score 1) are applied properly the earliest sign of dental fluorosis should not be confused with any of the non-fluoride-induced lesions. This is not to say that the presence of other types of enamel opacities cannot complicate the diagnosis of dental fluorosis (TF scores 1-2) when both types of enamel changes are present at the same time on a tooth surface. However, if the following criteria for differential diagnosis between non-fluoride-induced opacities and the milder forms of dental fluorosis are applied (**Table 12**) there should rarely be grounds for any doubt (Fejerskov et al 1988).

Table 12 Differential diagnosis, milder forms of dental fluorosis and enamel opacities of non-fluoride origin.

Source: Fejerskov et al (1988)

Character-istics	Dental fluorosis	Enamel opacities
Area affected	The entire tooth surfaces, often enhanced on or near tips of cusps or incisal edges.	Usually centered in smooth surface of limited extent.
Lesion shape	Resemble line shading in pencil sketch which follow incremental lines in enamel (perikymata). Lines merging, and in score 3, a cloudy appearance. At cusps/incisal edges formation of irregular white caps "snow cap".	Round or oval
Demarcation	Diffuse distribution over the surface of varying intensity.	Clearly differentiated from adjacent normal enamel.
Colour	Opaque white lines or cloudes, even a chalky appearance "snow caps" at cusp/incisal edges. Score 3 may become brownish discoloured at mesio-incisal part of central upper incisors after eruption.	White opaque or creamy-yellow to dark reddish-orange at time of eruption.
Teeth affected	Always on homologous teeth. Early erupting teeth, incisors/1st molars least affected. Premolars and second molars and third molars most severely affected.	Most common on labial surfaces of single or occasionally homologous teeth. Any tooth may be affected but mostly incisors.

Plate 27 is an example of a single enamel opacity of non-fluoride origin in an upper incisor which also exhibits dental fluorosis corresponding to a TF score of 1. Single opacities frequently occur in the lower incisors. Of all teeth in the permanent dentition, these teeth are, the least affected by dental fluorosis. The features of such opacities are quite distinct from all features of dental fluorosis, there should be no difficulty in making a differential diagnosis. Rarely, opacities may occur on homologous teeth. In **Plate 28** two distinct, well-defined opacities with clearly demarcated borders are seen.

The enamel opacities of non-fluoride origin (**Plate 29**), illustrates a case in which yellow-brown opacities are superimposed on milder forms of dental fluorosis. Such a lesion cannot be confused with any of the signs of dental fluorosis as the two features on the same tooth surface are quite distinct.

8.3.2 Cases of Limited Periods of Exposure to Fluoride

Cases which may cause differential diagnostic problems are those in which individuals have been exposed, during the long-lasting period of tooth-formation, to a limited and well defined period of fluoride ingestion. In such cases, only certain tooth groups or parts of the tooth surfaces exhibit the signs of dental fluorosis. These features may be seen in populations in which there have been substantial migrations in or out of high fluoride zones, or in cases in which, fluoride tablet regimes have been terminated for

one reason or another in the midst of the period of tooth-formation. However, if the principles underlying the TF classification system are properly applied in such cases, it should be possible to make a diagnosis, even if tentative, which can then be confirmed by taking a careful history of the individual concerned.

8.3.3 The Early Caries Lesion

In certain cases early "white spot" caries lesions occurring along the cervical margin may sometimes be mistaken for the features of mild dental fluorosis. As shown in **Plate 30**, such early lesions are usually characterised by having a more pronounced opacity than the opaque lines of dental fluorosis which can be seen on the remaining parts of the surface in TF scores 1 and 2. The location of the lesion at or just above the gingival margin helps in establishing a differential diagnosis.

8.3.4 "Idiopathic" Dental Fluorosis

As seen in **Plate 31**, at present, there is nothing about the distribution of such cases in any population, except that they are extremely rare. A few case reports have been presented of patients where no apparent history of any significant fluoride exposure existed, and yet the teeth exhibited the characteristics of dental fluorosis corresponding to the TF scores 2 to 5. The etiology of these cases is still unknown (Fejerskov et al 1988).

8.3.5 Enamel Hypoplasias

It would not be possible to mistake a classic enamel hypoplastic lesion, even of systemic origin, with any of the severe forms of dental fluorosis. Systemic hypoplasias affect homologous teeth and frequently several groups of teeth are involved. The classic features vary from a grooved line across the tooth surfaces to a wider band of faulty deformed enamel. These are always characterised by having smooth rounded and well demarcated borders as seen in **Plate 33**. Hypoplasias should be distinguished from opacities of the enamel. The form and shape of the enamel is always affected by hypoplasias, whereas opacities reflect only a disturbance of mineralisation, and do not result in any alteration in the shape or form of the tooth surface (Fejerskov et al 1988).

8.3.6 Amelogenesis Imperfecta

Of the various types of amelogenesis imperfecta, only the subgroups commonly referred to as the hypomineralised types can present a diagnostic problem. In **Plate 32**, an example is presented. There is an extensive brownish discolouration of the entire tooth, which in this case also exhibits a certain disfigurement of the crown. These features are very distinct from any of the forms of severe dental fluorosis (Pindborg 1982).

8.3.7 **Dentinogenesis Imperfecta**

In **Plate 33**, an example of a case of dentinogenesis imperfecta is shown. In such cases the discolouration of the tooth is a result of a developmental disturbance of unknown origin of the pulpo-dentinal organ. The discolouration is due to the fact that the pathologically disturbed pulpo-dentinal organ can be seen through the translucent enamel. Once again no differential diagnostic problems should exist (Fejerskov et al 1988).

8.3.8 **Tetracycline Stains**

Tetracycline-induced changes are characterised by a spectrum of discolouration, **Plate 34**. Such discolorations are very different to those associated with the post-eruptive changes seen in dental fluorosis. If an examiner should be in doubt, tetracycline induced changes can be differentiated from all other types of enamel changes by ultraviolet light (Fejerskov et al 1988).

9 THE TREATMENT OF DENTAL FLUOROSIS

The clinical pattern of mottled enamel study, has shown that because teeth remain fully adequate, both functionally and cosmetically, mild forms of mottling (first degree) require no treatment.

Moderate and severe forms of mottled enamel, in which the teeth are significantly disfigured, most often the front teeth, need special treatment. Mottled enamel with pronounced pigmentation so disfigures the teeth that those afflicted with these forms of fluorosis, especially young people, try to remove the defect with powdered brick, emery paper or else they cover the teeth with artificial (metal) crowns.

The effectiveness of treating fluorotic spots depends essentially on whether the symptoms in moderate and severe mottled enamel are reversible. Two things must be taken into consideration. Firstly, the mottling which we see occurs before the tooth erupts, in the period of its formation. Secondly, it depends on diagnostic possibilities. Mottled enamel can be diagnosed only after the tooth erupts, in the period when the enamel has acquired a mature and stable structure.

The **treatment of mottled enamel** as a whole consists of counteracting disfiguring spots and is accomplished by local action on affected tissues of the tooth. There are many known methods of treating mottling (removal of spots),

with the bleaching effect of medicaments releasing free oxygen (perhydrol, hydrogen peroxide), as the basic method.

9.1 CHEMICAL TREATMENT

The most effective treatment proven to treat **dental fluorosis with mechanical or chemical damage to the integrity of the enamel coating** is by chemical treatment (Ovrutskiy 1962).

The method of treatment consists of the following:

1. The tooth being treated is isolated from saliva with cotton cylinders, carefully wiped with a dry cotton ball and then dried with a cotton ball soaked in ether
2. Then the outer layer of enamel is ground off the labial surface of the tooth. This is done with a fine grinding carborundum stone or paper disks used for polishing fillings
3. A small cotton tampon soaked in a solution of perhydrol is applied to the "fresh" enamel surface for 10 minutes; as it dries it is moistened
4. After the tampon with perhydrol is removed, the cotton balls must be replaced by dry ones. If they are not, it is possible that the mucosa of the lip and gum will be burned by the perhydrol with which the tampons have been soaked. Teeth covered with dry cotton balls are radiated with a quartz lamp for 3-4 minutes
5. The session concludes by rubbing the treated (usually the labial) surface of the enamel with 75 per cent fluorine paste.

Treatment by this method can be completed in 2-3 sessions, but it is usually prolonged to 5-6 sessions so a lot of enamel is not ground off in one session. The interval between two sessions should be not less than 5-7 days, the minimum time necessary for regeneration processes.

If the integrity of the enamel coating was disturbed by chemical means, with lactic or acetic acid, the number of sessions should be kept to a minimum, not more than three.

It is better, to remove spots according to the described method without the application of acids. After acid treatment the enamel becomes dull. In one case after lactic acid was used on a 22 year-old patient, the treatment of mottled enamel was complicated by severe hyperaesthesia around the neck of the tooth. Results have shown that treatment by this method was permanent and showed no side effects (Ovrutskiy 1962).

Discovering the etiology of fluorosis, makes it possible to direct prevention of this disease towards standardisation of the fluorine content in the environment, particularly in drinking water supplies. Therefore, the basis of dental fluorosis prevention consists of regulating the amount of fluorine in drinking water, the replacement or defluoridation of water supplies with high fluorine levels.

9.2 METHODS FOR ACTIVE DEFLUORIDATION

Instead of replacing the water supply to prevent dental fluorosis, several methods for active defluoridation of drinking waters have been developed. Most of these methods are based on the principles of absorption or ion exchange such as the use of:

- 1 Lime softening
- 2 Alum
- 3 Alum and lime (Nalgonda technique)
- 4 Activated alumina
- 5 Activated carbon
- 6 Natural bone
- 7 Bone char
- 8 Bone char and charcoal (ICOH Defluoridator)
- 9 Synthetic bone
- 10 Bauxite
- 11 Commercially produced ion-exchange resins
- 12 Electrodialysis
- 13 Reverse osmosis
- 14 Various other treatment agents

In the following, therefore, only those methods which have proven to have a certain cost-effectiveness will be dealt with in more detail (Commins 1985, Phantumvanit et al 1988).

9.2.1 Lime Softening

It is possible to remove fluorides as a side reaction to the lime softening of water if the water contains high levels of magnesium (>50 mg per litre) and moderately low levels of fluoride (3-4 mg per litre). Strict control of pH and alkalinity during the process is important. The method is only feasible when both hardness and fluoride

removal are needed. Ordinary commercial home water softeners have been shown to have only limited fluoride-reducing capacity (Commins 1985).

9.2.2 Alum

Alum (aluminium sulphate) has a high fluoride absorption capacity and it is only suitable for treating certain types of water. The method can reduce the fluoride concentration from 7.5 ppm to 0.8 ppm after 2 hours at pH between 7.2 and 8.4. With the high alum dose needed there is a risk that residual aluminium levels will exceed the WHO guideline value (0.2 mg per litre) (Commins 1985).

9.2.3 Alum and Lime

This method, known as the Nalgonda technique, involves adding in sequence sodium aluminate or lime followed by alum. Bleaching powder may also be added for disinfection purposes. This method is based on flocculation, sedimentation and filtration. To be effective the method requires careful control of the alkalinity of the water which can be difficult if the raw water contains more than about 10 ppm of fluoride. This method has been used with success in several areas in India.

The capital cost for a medium-sized defluoridation plant would be in the order of US\$ 100 per m³ water and the annual cost of defluoridation at a rate of 50 litres per capita per day is approximately US\$ 5.

The Nalgonda technique can also be modified for use in individual households. A mixture of alum and calcium carbonate is added in a specific ratio for each home, depending on the fluoride content of the water. When adding the mixture to the water, a white flocculent precipitate is formed, which settles down in about 10 minutes leaving the supernatant ready for household use (Commins 1985).

9.2.4 Activated Alumina

Activated alumina is a special form of acid-treated alumina. When fluoride is very high, levels of 10 ppm F^- in water can easily be reduced to around 1 ppm F^- . Levels as low as 0.1 ppm F^- can be achieved if desired. Control of optimum pH (5 to 6) as well as carefully operated regeneration procedures are important. The capacity of the medium decreases with an increase in pH or alkalinity.

The defluoridation equipment is normally installed in connection with an existing water treatment plant. The defluoridation equipment can be operated by the ordinary water plant operator if on-the-job instruction in the chemical and engineering aspects of defluoridation is provided (Rubel 1983).

For a town of approximately 2,000 inhabitants, the capital investment for a defluoridation plant using activated alumina would be in the order of US\$ 60,000, with an operation and maintenance cost of US cents 3-5 per m^3 of water.

Most defluoridation plants using activated alumina have been installed in developed countries. In specific cases, however, other forms of treatment may be more cost effective (Commins 1985).

9.2.5 Activated Carbon

Carbon prepared from different carbonaceous sources, have a significant fluoride removal capacity with alum. The fluoride removal capacity is pH dependent. At pH 3, a level of 8 ppm F^- can readily be reduced to 1 ppm. However, the method can be costly to operate since the pH of the water initially needs to be low, and it must be raised again to make the water suitable for drinking purposes. The fluoride removal capacity is also dependent on the material from which the activated carbon has been produced (wood, paddy-husk, cotton, coffee or coconut waste). Because of the relatively high operation costs and its limitations, activated carbon as the only ingredient in a defluoridation plant would seem to be of limited value (Commins 1985).

9.2.6 Natural Bone

Dried and crushed natural bone has a significant fluoride removal capacity. However, bacteriological problems and taste problems associated with the treated water would seem to make this method less attractive compared with the use of bone char or synthetic bone (Commins 1985).

9.2.7 Bone Char

Bone char is produced from natural animal bone which is degreased, dried and pulverised to 400 mesh size, and carbonised in a close furnace at a temperature of 600°C for 20 minutes. Back-washing by caustic soda is used for regeneration. Excess caustic soda is removed by rinsing with raw water (Commins 1985).

9.2.8 Bone Char and Charcoal (ICOH defluoridator)

The ICOH defluoridator for individual households developed in Northern Thailand (Phantumvanit et al 1987) uses a combination filter with 1000 g of bone char and 300 g of charcoal. The filter has been shown to reduce the fluoride content in 480 litres of water from 5 ppm F⁻ or more to less than 1 ppm F⁻ at a flow rate of 4 litres per hour before regeneration or discharge of the filter is needed. The ICOH-defluoridator is assembled from materials generally available for plumbing purposes and so simple in its design that the villagers themselves can produce it.

9.2.9 Synthetic Bone

Synthetic bone is a mixture of tricalcium phosphate and hydroxyapatite produced by reacting phosphoric acid with lime. Synthetic bone is used and regenerated in a similar way to bone char. This method has only been used in a few plants in the USA, and does not seem to be suitable for developing countries (Commins 1985).

9.2.10 Bauxite

Bauxite has been shown to have a reasonably good fluoride removal capacity. However, it has a narrow optimum pH range (3.0-5.5) unless a very long contact time and large amounts of bauxite are employed. In one study more than 90 per cent of the fluoride was retained with a contact time of 4 hours at pH 5.5 to 7.0 with an absorbent dose of 25 g bauxite per litre. Bauxite is not generally locally available and this method, therefore, would seem to be less feasible for developing countries (Commins 1985).

9.2.11 Commercially Produced Ion-Exchange Resins

Methods using ion-exchange resin are normally rather expensive since most of these resins are commercially produced. The commercial names of some of these ion resins are: Amberlite IRA-400, Amberlite XE-75, Carbion, Deacolite FF-1P, Defluoron 2, Florex, Lewatite MIH-59, Tulsion A-27, Vyrodex, Waso 14, Zeokarb 225, and others. The capital costs of using these methods can range from US\$ 500 to US\$ 2,000 per m³ water with an annual treatment cost of US\$ 8 to US\$ 90 at the rate of 50 litres per capita per day. Most of the resins mentioned above are very effective but the cost-effectiveness is so poor that these methods will hardly be of interest for developing countries except perhaps Defluoron-2 which has been used with some success in India. Defluoron-2 is a commercial cation exchange medium prepared from bituminous coals by partial sulphonation. Alum is used to regenerate the material. It is capable of reducing fluoride levels of 3-10 ppm F⁻ to

less than 1 ppm F^- . Even though the method has been used in India it is considered expensive in operation and regeneration. Skilled operators are needed to maintain effective control of the method, which would limit its use, particularly in developing countries (Commins 1985).

9.2.12 Electrodialysis

This method is based upon a membrane separation electrodialysis technique, mostly suitable for brackish water. It requires high technology and is rather expensive, for which reasons it is not likely to be appropriate for developing countries (Commins 1985).

9.2.13 Reverse Osmosis

This method is based on the utilisation of a semipermeable membrane which retains dissolved solids in the water to be treated. There are many different types of such semipermeable membranes but they all seem to require strict control and maintenance and the efficiency of the process is both pH and temperature dependent. Some of them involve multistage processes with special pre-treatment of the raw water. Even though this method is highly efficient it is complicated and expensive and, therefore, at present hardly of any relevance to developing countries (Commins 1985).

9.2.14 Various Other Treatment Agents

A number of other treatment agents for fluoride removal have been proposed: ferric ions, magnesium salts, calcium phosphate, bentonites, Fuller's earth, diatomaceous earth,

serpentine, silica gel, sodium silicate, and strychnos potatarum, which is available locally in Sri Lanka. Under experimental conditions most of these have shown moderate to high fluoride removal capacity. However, little is known regarding their general usefulness and applicability. Some of these agents require the pH of the water to be adjusted to a low value (less than pH 3) to provide effective fluoride removal.

Table 13 shows a comparison between the most commonly used methods which might assist the responsible bodies to make tentative and alternative choices. When the most suitable method has been chosen, a feasibility study should be performed to ensure that the method would really provide the expected result under the conditions prevailing in the area in question.

Table 13 A comparison of the most commonly used methods
for defluoridation of drinking waters
Source: Fejerskov et al (1988)

[illegible]

10 DISCUSSION AND CONCLUSION

Dental fluorosis occurs as a result of long-term intake of fluoride during the period of tooth formation. It is characterised by increasing porosity of the surface and subsurface enamel, causing the enamel to appear opaque. Several factors are involved in the occurrence of dental fluorosis. From the review of literature these are, water, foods, industrial and pharmaceutical products. Teeth, bones and tissues are the richest in fluoride. Fluoride is absorbed mainly in the stomach and excreted via the kidney. Dental fluorosis is symmetrically distributed in the mouth, but not all teeth are equally affected. The first sign of dental fluorosis appears as fine striae of accentuated perikymata, but in severely damaged teeth, almost the entire enamel surface appears to be corroded. Incisors and premolars are the most severely affected and first molars exhibit marked post-eruptive changes. Due to the difficulties in the diagnosis of early stages of dental fluorosis new indices have been developed. The prevalence and the severity of dental fluorosis is increasing in both fluoridated and non-fluoridated areas specially those near the sea. The basis of dental fluorosis prevention consists of regulating the amount of fluorine in drinking water by the most suitable method, and study should be performed to ensure that the method would really provide the expected result.

Clinically, dental fluorosis has been observed among the people who are born and brought up in the Province of Boushehr in Iran. Due to the lack of knowledge of fluoride and dental fluorosis among the professionals, research communities, public health workers, and government decision makers, no epidemiological and clinical data are available. Therefore, a survey should be carried out by well trained personnel. According to the literature reviewed the best groups to include in this survey are those 12 to 15 years of age and TF Index is recommended as a classification system, because measures the mildest to the most severe forms of dental fluorosis and has the benefit of being clear, precise and sensitive for the measurement of dental fluorosis. The bone char and charcoal (ICOH-defluoridator), alum and lime (Nalgonda technique), and bone char, have good fluoride removal capacity, are commercially available and cost-effective (overall), and are suitable methods to regulate or defluoridate the level of fluoride in a community water supply.

The following recommendations have been suggested for Boushehr province in Iran:

1. Increase the knowledge of fluoride and dental fluorosis in the professionals, research communities, public health workers and government decision makers.
2. Identify current levels of fluoride in community water supplies.
3. Identify the prevalence and severity of dental fluorosis in the population aged 12-15 years who have been born and brought up in the Boushehr province.

4. If the prevalence of dental fluorosis is high in that population, studies need to be done to identify the causes.
5. As a index of choice, the Thylstrup and Fejerskov (TF) Index is recommended for use as a classification system to measure the affect of fluoride exposure in the population.
6. The level of dental fluorosis in the population needs to be monitored regularly.
7. If the level of fluoride in drinking water is too high suitable methods should be chosen to regulate or defluoridate the level of fluoride in community water supplies.

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12 APPENDICES

The appendices are 34 colour plates of the TF index and diagnostic procedures for fluorosis.



Plates 1 and 2. TF score 1. Fine white lines across the entire tooth surface.

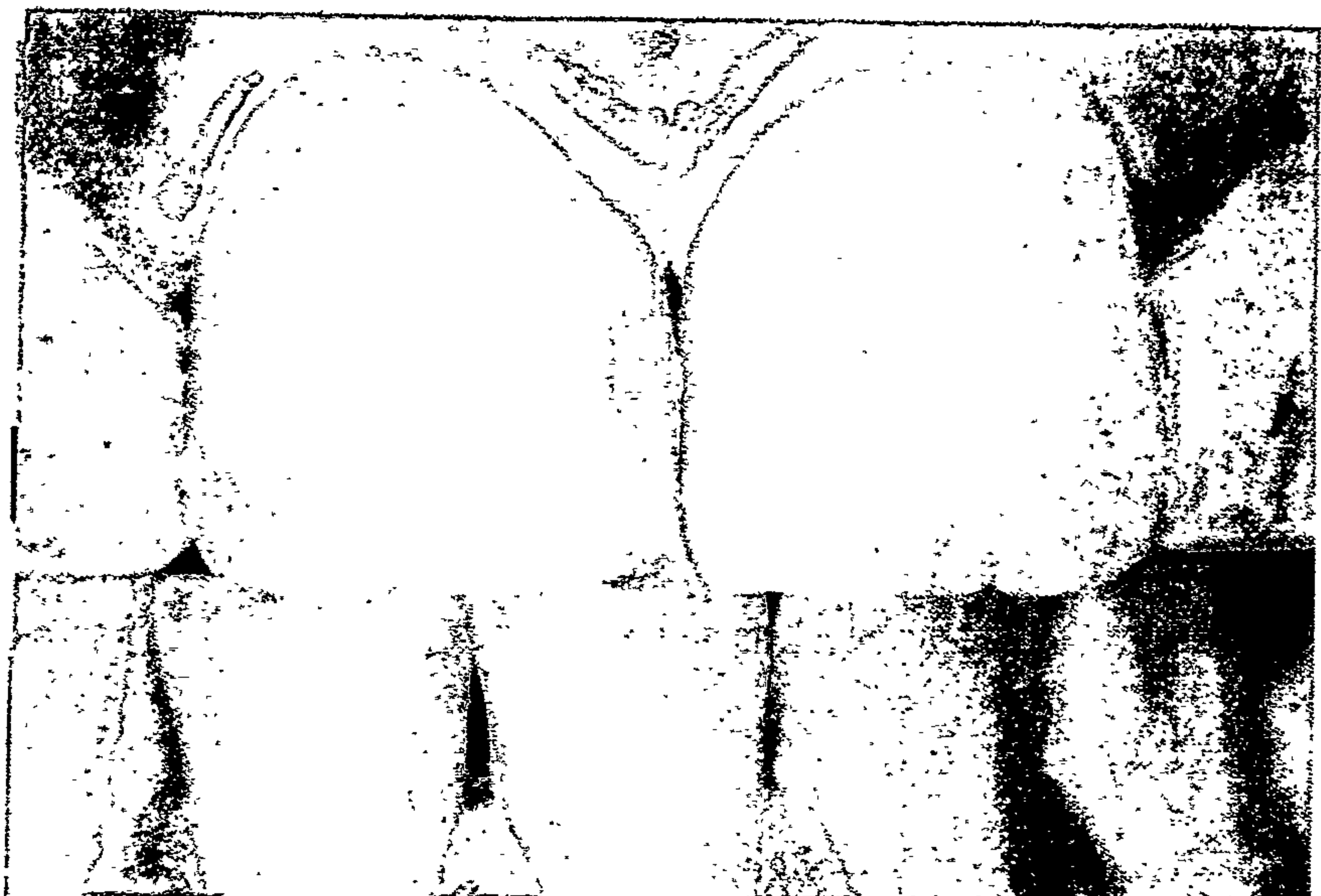
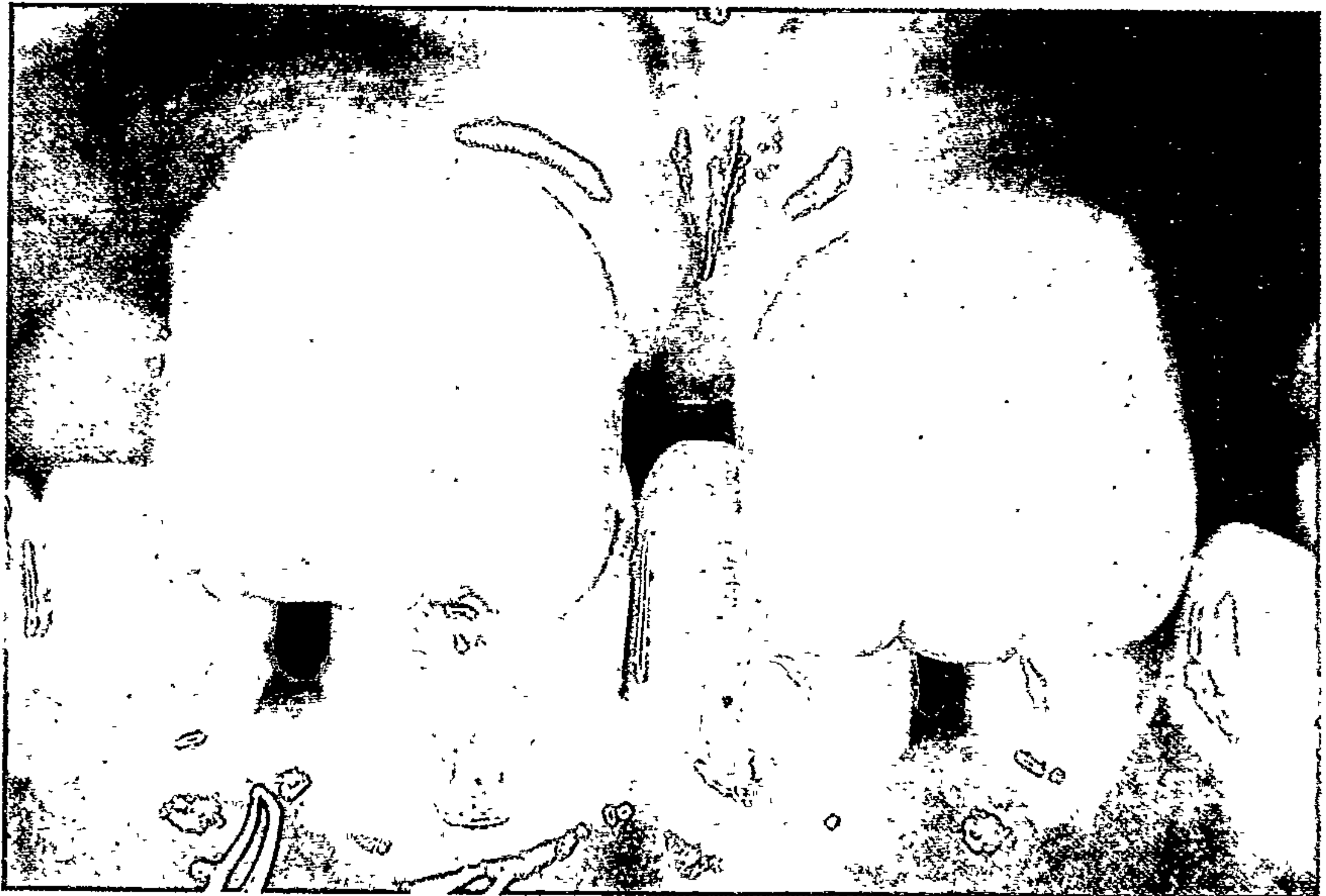


Plate 3. TF score 2. Fine white lines frequently merge.



Plate 4. TF score 3. Irregular cloudy white areas.



Plate 5. TF score 3 with brownish discoloration.



Plate 6. TF score 4 (right incisor) and score 5 (left incisor).



Plate 7. At time of eruption teeth are chalky white even in severe cases of dental fluorosis.



Plate 8. TF scores 4 to 7.



Plate 9. TF scores 4 to 7. Note the loss of outermost enamel to a varying extent.



Plate 10. TF score 7 with uptake of stain in exposed porous enamel. The remaining intact enamel is white, opaque.



Plate 11. TF score 7.



Plate 12. TF scores 8 and 9.



Plate 13. TF scores 7 to 9.



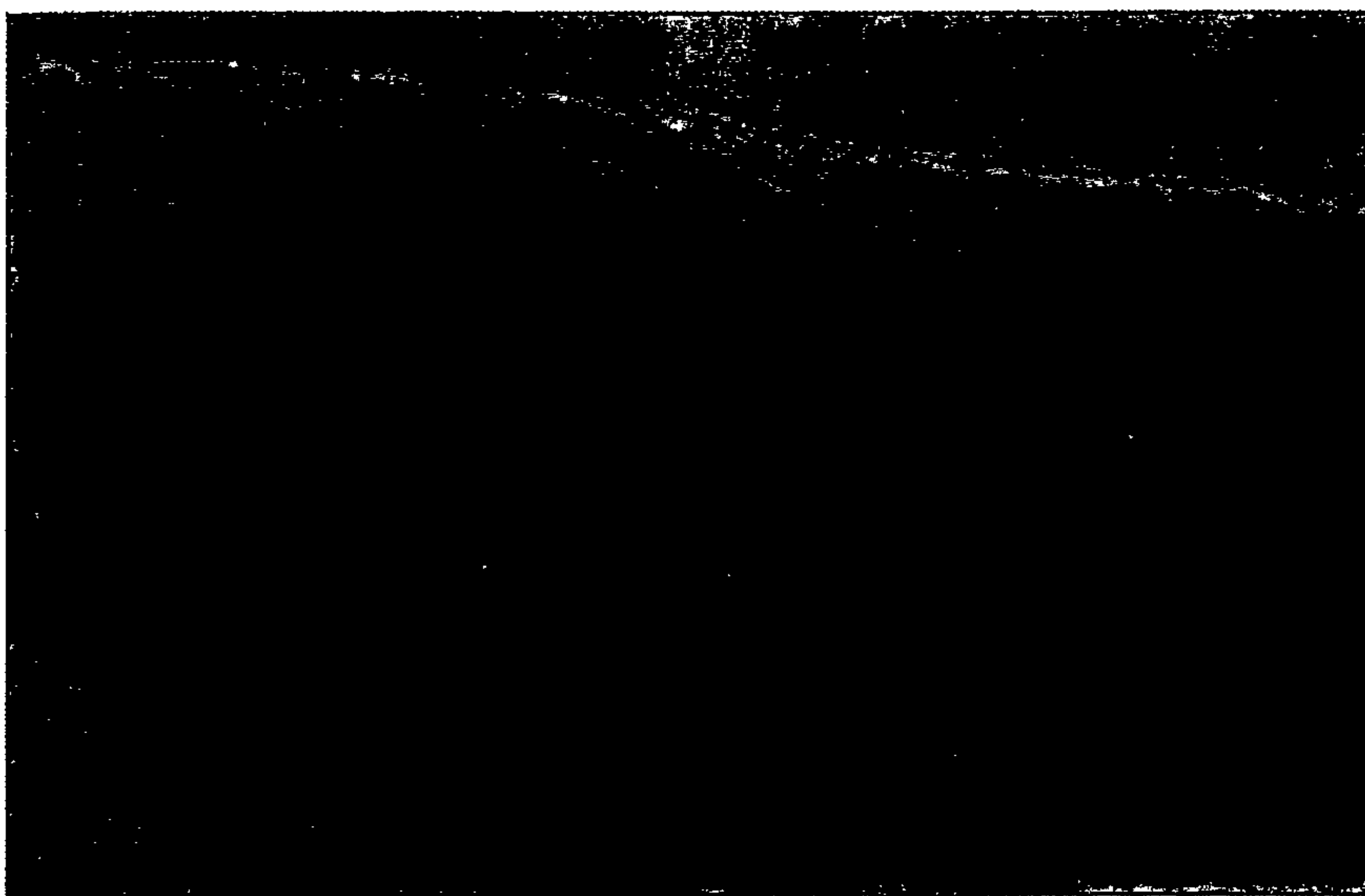
Plate 14. The later in life a tooth is formed and erupts the more severe is the degree of dental fluorosis. TF score 9.



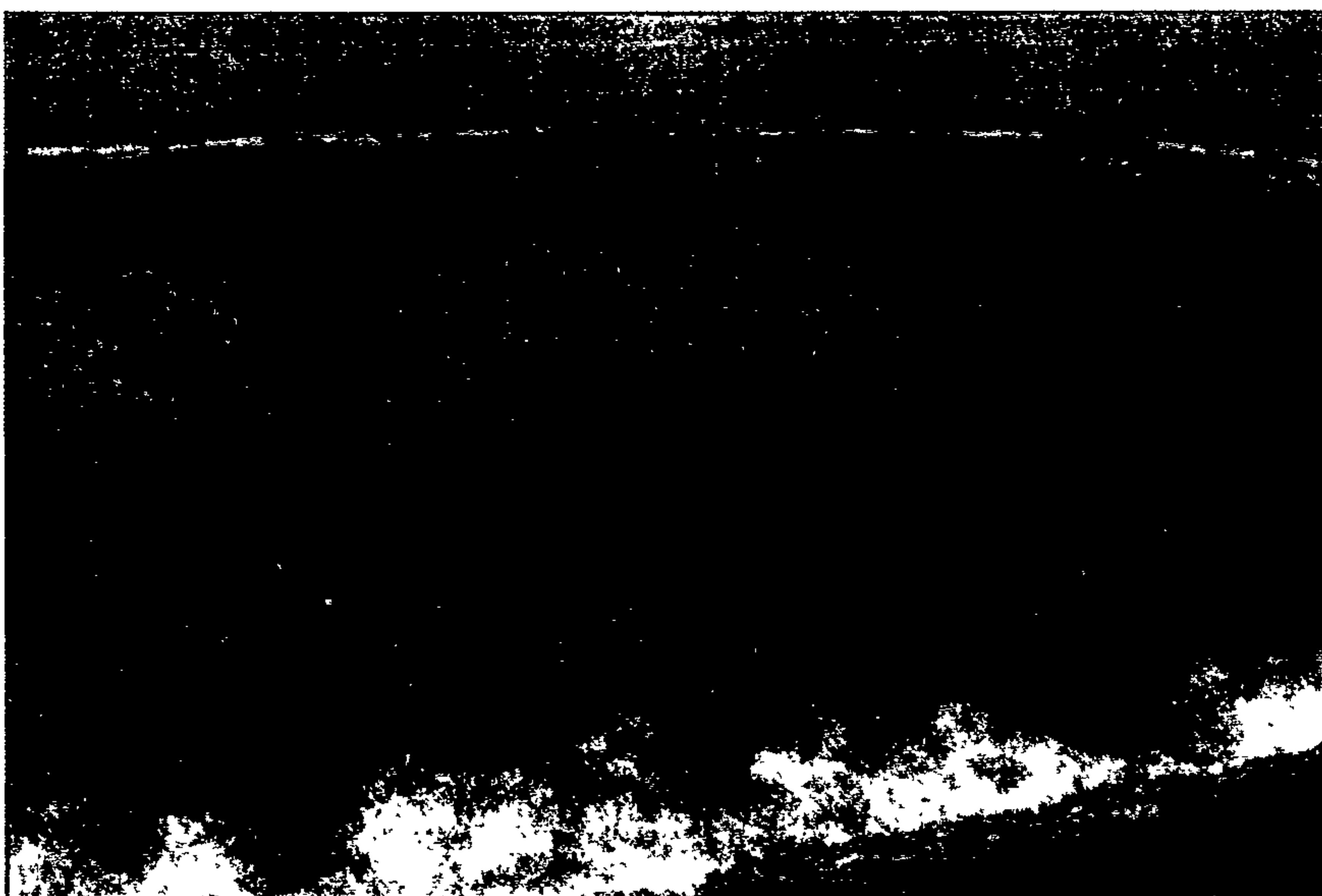
Plate 15. Dental fluorosis in anterior deciduous teeth.

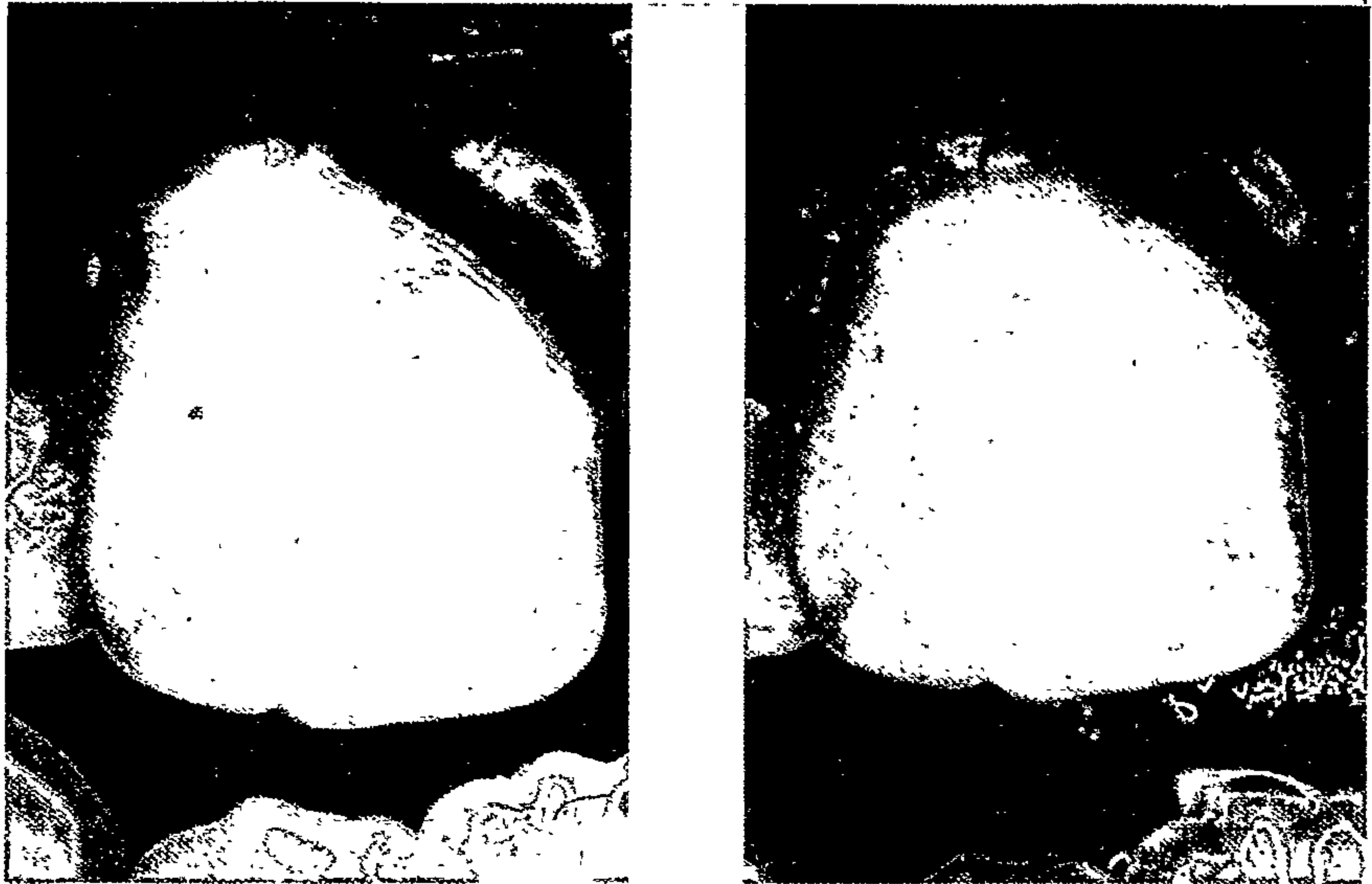


Plate 16. Post-eruptive damages of fluorotic primary and permanent molar.



Plates 17 and 18. Microphotographs of very mild (17) and severe (18) fluorotic enamel. Green colour indicates normal tissue. Reddish/brownish colour represents porosity.





Plates 19 and 20. The effect of plaque removal on diagnosis of dental fluorosis.



Plates 21 and 22. The effect of drying the teeth in order to visualize the porosities clinically.



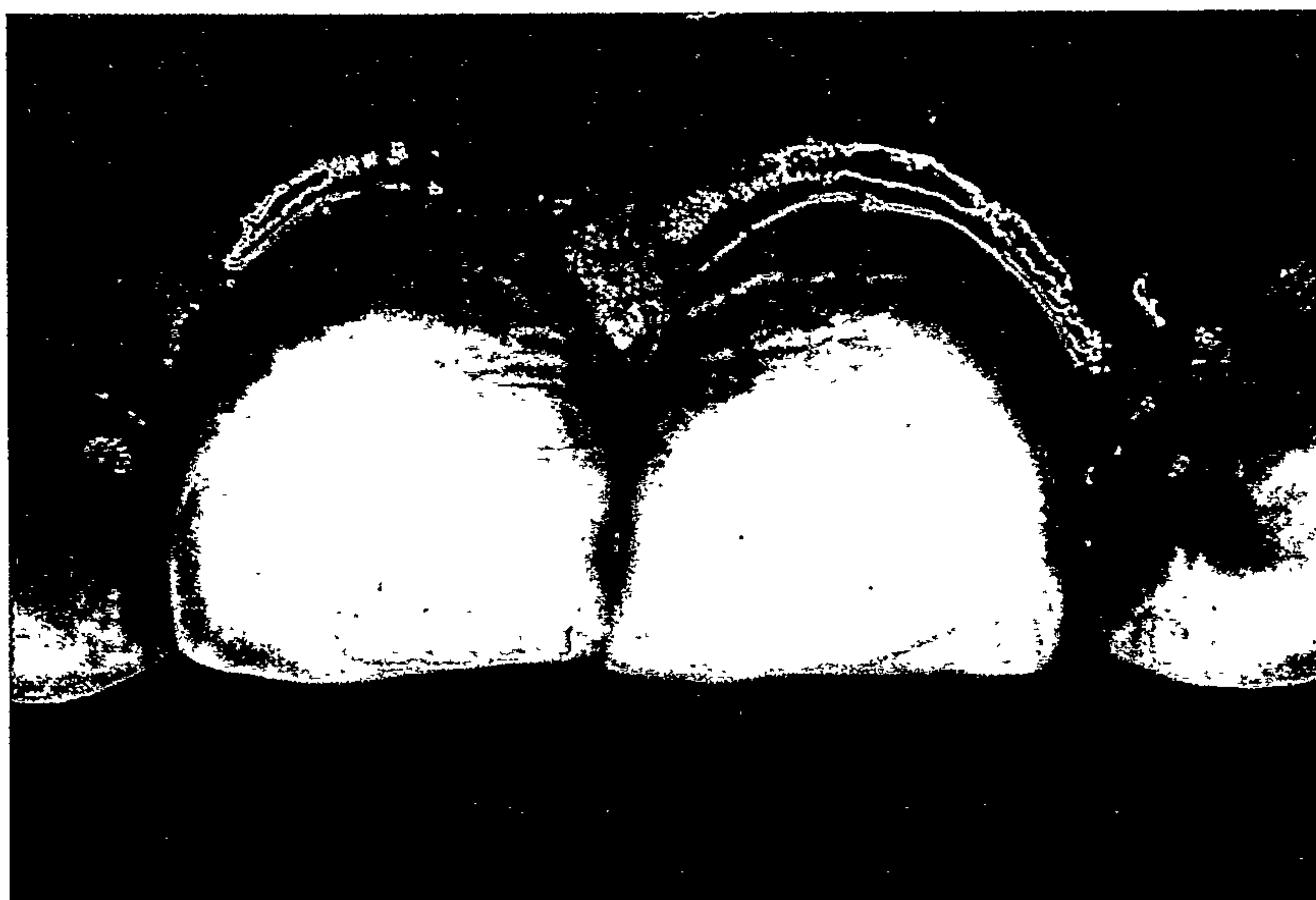
Plate 23. Occlusal abrasion of a porous fluorotic molar.



Plate 24. Extensive discoloration corresponding to position of upper lip.



Plates 25 and 26. The effect of grinding the outermost porous enamel away on central incisors.



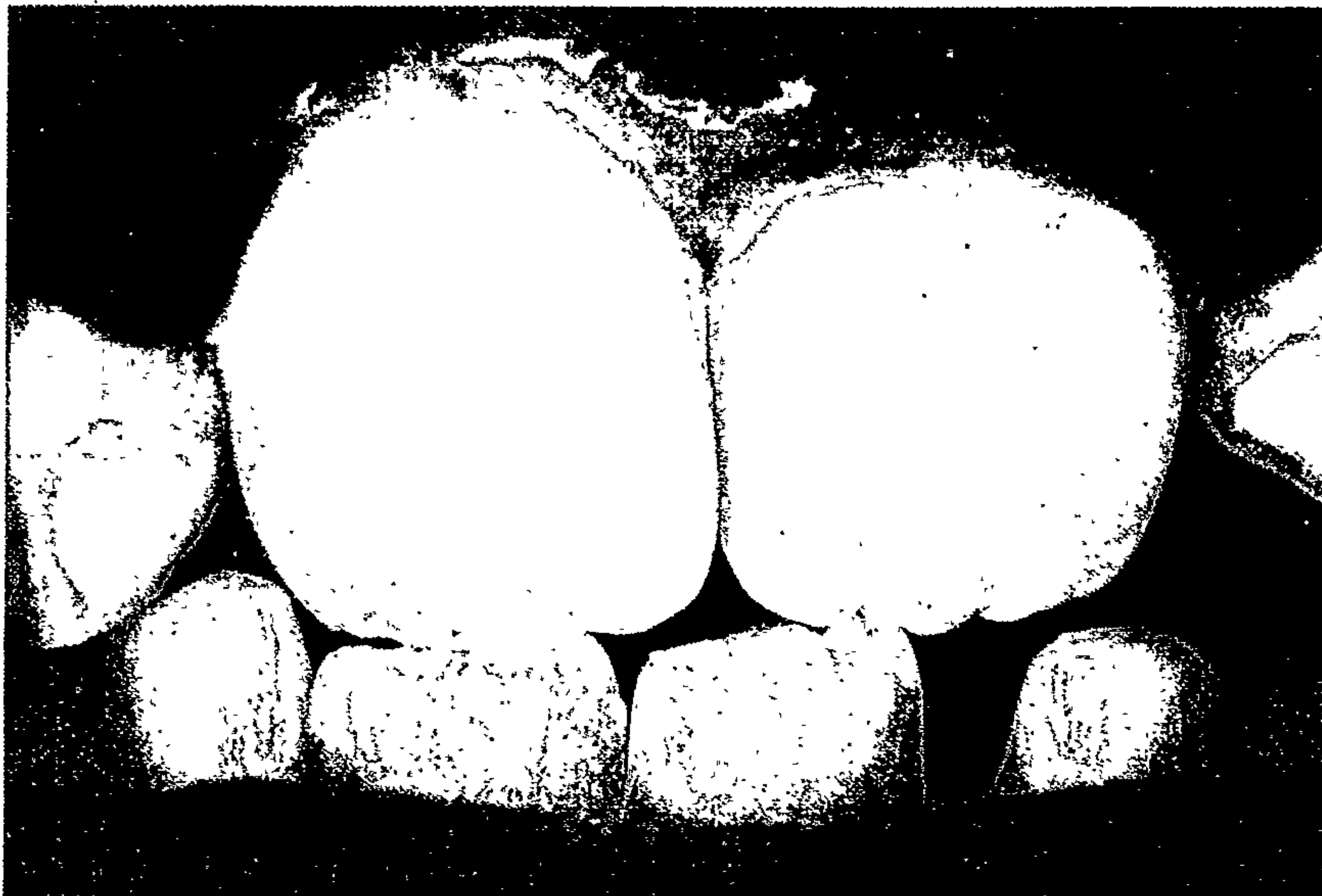


Plate 27. Well-demarcated enamel opacity on central incisor with TF score 1.

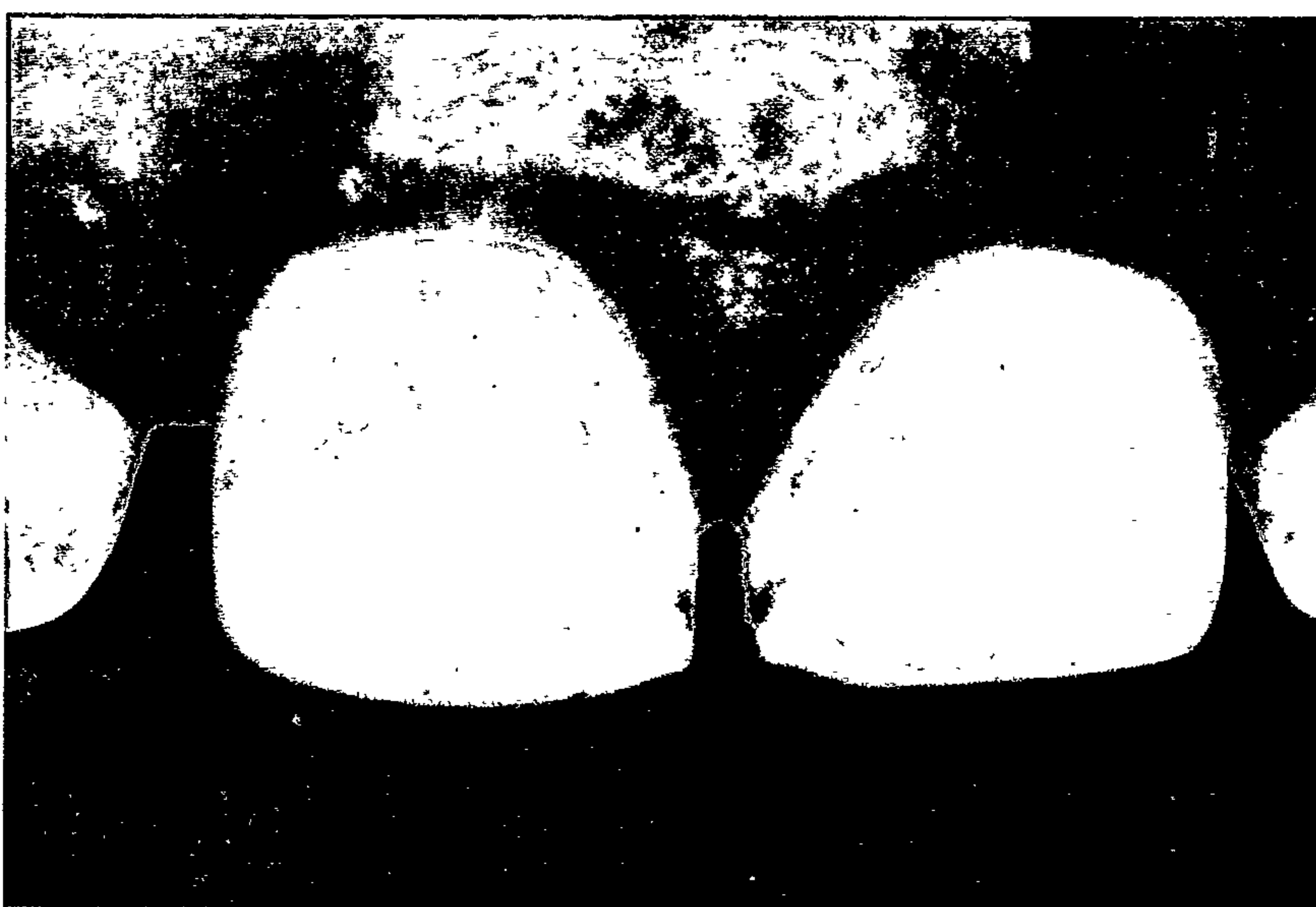


Plate 28. Non-fluoride-induced enamel opacities on homologous teeth.

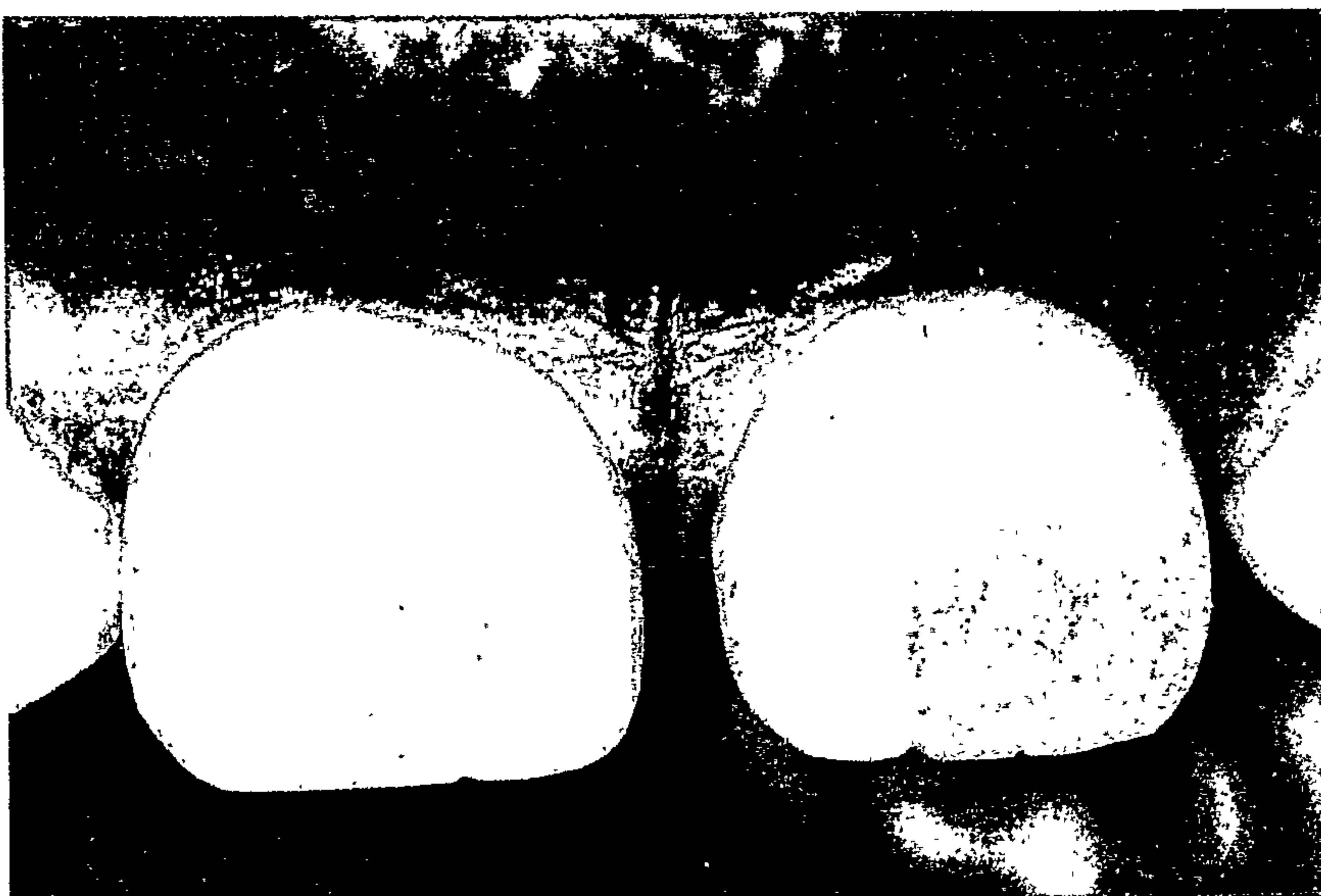


Plate 29. Well-demarcated brown opacities in teeth with TF score 2.



Plate 30. TF score 2 with "white spot" caries lesions along the gingiva of the central incisors.



Plate 31. Rare case of idiopathic dental fluorosis. Compare with Plate 5.



Plate 32. Amelogenesis imperfecta of the hypomineralized type.



Plate 33. Dentinogenesis imperfecta. In addition the lower incisors exhibit a band of enamel hypoplasias.



Plate 34. Tetracycline staining of permanent teeth.

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