INCIDENCE OF DYSTROPHIES CAUSED BY FLUORINE IN ORGANS REGULATED BY THE PARATHYROID GLANDS

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(With Plate 4)

A recent investigation into the symptomatology of chronic fluorine poisoning (fluorosis) revealed, amongst other findings, that organs originating in the ectoderm and regulated by the parathyroid glands, namely, the skin and its appendages, the nails, teeth and hair, were affected (Spira, 1942 c, d). The object of the present paper is to describe the incidence of these lesions as found in a new series, one consisting of 1700 people taken at random, namely, 850 men and 850 women. They belonged to different spheres of life and came from various parts of the country. Their average age was 25 years, namely, 29 years for the men and 21 years for the women.

THE NAILS

The nail is formed in an epidermic invagination, the upper fold of which, the nail wall, covers the proximal end of the nail plate, whereas the lower fold, the nail matrix, constitutes its only reproductive part. Laterally the nail rests on the nail grooves. A normal nail is oblong in shape, and is curved in its transverse axis. Its outer, convex surface is smooth, shiny and pinkish in colour. It grows from the matrix to the free edge in from 130 to 160 days, the average growth being about 3 mm. per month, and it consists mainly of an albuminoid substance called keratin and lipids in the form of cholesterol. The latter, probably the product of cell metabolism, help to maintain the elasticity of the nail. Nails deprived of cholesterol, such as occur in the course of systemic disturbances, become scaly, dry and brittle.

It was shown by the previous investigation referred to above that 185 out of 1099 people with mottled teeth, that is to say 17 %, complained of their finger-nails being brittle. A deviation from the normal finger- and toe-nails was, however, also encountered very frequently amongst those classified as not having mottled teeth. Thus brittleness of the finger-nails was complained of in as many as 31 % of all the 1700 people examined in the present series.

As seen from Table 1, the changes met with most frequently in both the finger- and toe-nails were onychorrhexis and Beau's lines. Onychorrhexis is a condition in which the nail plate, instead of being smooth, presents a longitudinal striation of varied degree, a striation caused by ridges and depressions running parallel to each other. They are frequently described as corresponding to similar longitudinal depressions and ridges produced on the nail-bed by the papillae of the corium. When onychorrhexis is advanced, these parallel ridges in the nails are not only visible but also palpable on the outer surface of the nails, and their pathological significance becomes

obvious. In the finger-nails the longitudinal striation was frequently, but in the toe-nails always, accompanied by Beau's lines, which are transverse elevations alternating with depressions and giving the affected nail an undulated appearance. On the other hand, when Beau's lines were present they were invariably associated with the longitudinal striation. They occurred more frequently, and were more pronounced, in the big toes than in the thumbs. Nor was longitudinal striation, even when associated with Beau's lines, always accompanied by leukonychia.

As is implied in its name, leukonychia is a condition characterized by opaque paper-white decalcified specks and patches or horizontal bands. The horizontal bands are sometimes so wide as to replace a considerable part or even the entire surface of the nails. According to the shape and the extent of these chalky white markings the condition is spoken of as leukonychia punctata, striata or totalis. Pl. 4, fig. 1 depicts the nails of a woman showing leukonychia affecting nearly their whole surface, together with those of her 17 months old child showing a pronounced degree of longitudinal striation and leukonychia punctata. In the finger-nails leukonychia punctata was met with far more often than leukonychia striata; in comparison with either of them leukonychia totalis was of rare occurrence, though not nearly so rare as the few cases described in the dermatological literature would suggest. In contrast to the finger-nails, in the toe-nails leukonychia punctata or striata were observed on a few occasions only. Most of the cases of leukonychia in the toe-nails exhibited that form of the dystrophy in which the paper-white decalcified zone extended over a considerable part of the nail plate.

In many cases tiny shallow round pits were encountered on the surface of one or more finger-nails. They were either solitary or multiple (Pl. 4, figs. 7, 8). Occasionally they were found to become confluent, and thus form an irregularly shaped pit of a larger size. If the lesion extends over a large part of the nail, it produces onychia. When allowed to grow excessively, the nails of both the fingers and toes tend to curl round their tips in a claw-like manner. In the toe-nails the transverse curvature was exaggerated in many cases, and the frequency with which the toe-nails grew sideways was striking. This often led to the nails becoming ingrown. Onychiauxis and onychogryphosis were of comparatively rare occurrence, these conditions being confined to the toe-nails, especially the big toes. On one occasion only, and that in a case outside the present investigation, did I come across a thumb-nail which exhibited this dystrophy, along with the tendency to grow sideways. The affected nails are

characterized by their being hypertrophied, thickened and raised by a brittle, greyish or brownish corneous accumulation which fills the subungual space. This accumulation, even when only slight in amount and insufficient to produce onychogryphosis, gives the nail a discoloured appearance, the discoloration being a dull dirty grey-brown. In koilonychia the nail plate of one or more fingers, instead of being convex, is concave. It is for this reason that they are popularly called 'spoon nails'. Their occurrence was rare, and the dystrophy was never found to affect the toe-nails.

The degree of the onychodystrophies described varied, of course, considerably. In the majority of cases they presented nothing more than an unsightly appearance, one that could be successfully concealed by nail polish.

The various forms of mottled nails improved promptly when the sources of fluorine poisoning were cut off and the amount of the poison accumulated in the body was eliminated by means of charcoal and an aperient. All the described onychopathies, even when they occur singly, as they occasionally do, or even without the other organs originating in the ectoderm being affected, thus point to the presence of a systemic condition, one which could not, in the present state of our knowledge, be easily detected. Since it has been clinically demonstrated (Spira, 1942*c,d*) that the parathyroid glands are in some way affected by fluorine, the present investigation into the incidence of the nail dystrophies permits of conclusions being drawn as to the extent to which the population of this country is exposed, in a varying degree, to fluorine poisoning. It

Table 1. *Incidence of onychodystrophies*

		Numbers examined					
		Total 1700		Men 850		Women 850	
		Incidence	%	Incidence	%	Incidence	%
Onychorrhaxis	Fingers	1632	96	819	96	813	96
	Toes	1662	98	820	96	842	99
Beau's lines	Fingers	1118	66	552	65	566	67
	Toes	1674	98	828	97	846	100
Leukonychia	Fingers	1189	70	644	76	545	64
	Toes	125	7	76	9	49	6
Pitting	Fingers	708	42	378	44	330	39
	Toes	—	—	—	—	—	—
Claw-shape	Fingers	936	55	516	61	420	49
	Toes	704	41	424	50	280	33
Koilonychia	Fingers	30	2	24	3	6	1
	Toes	—	—	—	—	—	—
Exaggerated transverse curvature	Fingers	—	—	—	—	—	—
	Toes	358	21	215	25	143	17
Growing sideways	Fingers	—	—	—	—	—	—
	Toes	1660	94	774	91	816	96
Onychogryphosis	Fingers	—	—	—	—	—	—
	Toes	107	6	67	8	40	5
Brittleness	Fingers	527	31	195	23	332	39
	Toes	—	—	—	—	—	—

When advanced, and especially when accompanied by dystrophies of other organs regulated by the parathyroid glands, they indicated at a glance that the calcium metabolism was disturbed, since it is a well-established fact that it is the parathyroid glands which exercise their regulating influence over the calcium metabolism of the body. It has already been shown (Spira, 1943) that the various onychopathies are closely similar to mottled teeth, a lesion caused by the ingestion of toxic amounts of fluorine. In fact, it was this close similarity of the changes in the two structures which led the described nail dystrophies to be attributed to chronic fluorine poisoning, and the suggestion to be made that they should be designated simply as 'mottled nails', in analogy with the generally accepted term 'mottled teeth'.

confirms the endemic nature of the disease in this country. To consider this pathological condition to be a physiological one on account of the fact that it has been found to be present in very large numbers is a fallacy to be avoided. Moreover, the presence of mottled nails is a considerably more reliable sign of fluorosis than are mottled teeth, since mottling of the nails occurs at any time in life during exposure to fluorine, whereas mottling of the teeth is the result of ingestion of toxic amounts of fluorine only during the period of their calcification. In addition, the rarity with which deciduous teeth are mottled, and the extraction of permanent 'mottled teeth' involve the loss of a very valuable sign of fluorosis; 'mottled nails', on the other hand, are, whilst present, a definite and easily detectable sign of exposure to toxic amounts of fluorine.

THE TEETH

In the first report on the results of the survey made on 5019 men and women, it was shown that 22 % of those examined were afflicted with mottled teeth (Spira, 1942 *a, b*). Emphasis was, however, laid on the fact that this incidence did not give a complete picture, the true incidence being, in fact, considerably higher, since not only were all the edentulous cases and those with a neglected oral hygiene classified as being free from the affliction, but also all the doubtful cases were left out altogether. Amongst this latter category there were numerous instances of the dystrophy which, according to the state of our knowledge, were generally attributed to 'hypoplasia', rickets, infectious diseases of childhood or avitaminosis. This differentiation between the causations of the dystrophy is, however, not warranted, since the appearance of them all is identical in practically every respect. It is all the more unjustified as mottled teeth, too, are described as a 'hypoplasia': one which is caused by fluorine. It appears that the term 'hypoplasia', as used in dental pathology, is at variance with that used in general pathology, and that a different meaning is being attached to it not only by different writers, but on different occasions by the same writer. If, however, the term 'hypoplasia' is correctly restricted to mean a condition of developmental arrest, such a disturbance of dental development would appear to be incongruent with the conception of a dental lesion, as produced by fluorine or any other toxic agent during the period of calcification of the teeth. 'Hypoplasia' indicates suppression of developmental progress: retarded development. A defect or erosion, on the other hand, as encountered in mottled teeth, is a lesion produced by a noxious agent. Nor can the suggestion made by Fossum (1928) to the effect that mottled teeth are a 'hypoplastic' dental condition caused by infectious diseases such as measles, scarlet fever, diphtheria, chickenpox or smallpox be accepted. Although taken up and often repeated by textbooks, such findings have never been confirmed. The suggestion has been finally disproved by Smith, Lantz & Smith (1932). We should not lose sight of the fact that all these infectious diseases may be accompanied by fragility of the hair and dystrophies of the finger- and toe-nails.

In view of our thus rectified interpretation of pathological conditions, and the fact that Erdheim, in his classical work on tetany (*Tetania parathyreopriva*), reported (1906) that the removal of the parathyroid glands produces not only rickets and osteomalacia but also a dental lesion, one which is identical with that caused by fluorine, the several causes of mottled teeth can now be reduced to one, namely to a disturbance of the action of the parathyroid glands. Both the chemical substance and the surgical operation interfere with their internal secretion. There is a divergence of opinion with regard to the rôle which avitaminosis plays in producing dental lesions. Such lesions have been observed in experimental rats fed on diets deficient in vitamin A (Wolbach & Howe, 1925, 1928, 1933 *a, b*), vitamin C (Howe, 1920, 1922; Höjer, 1924; Wolbach & Howe, 1926) and vitamin D (Mellanby, 1929, 1930; Becks & Ryder, 1931; Rosebury & Foley, 1934), as quoted by Thoma (1941). The teeth lose their normal orange pigmentation, acquire a chalky white appearance and become brittle; they may

become so loose that they can be pulled out with the fingers. All these changes are indicative of a process of defective calcification. Macroscopically such dental changes thus resemble those produced by a lowered function of the parathyroid glands, whether resulting from surgical damage or from chemical destruction. On the other hand, Smith *et al.* (1935) and Smith (1936) demonstrated that mottled teeth, which are the result of decalcification produced by the destructive action of fluorine, cannot be prevented either by means of dietary improvement or by the addition of vitamins A, C or D. Yet, the facts that rickets is cured by vitamin D, and that tetany, even when resulting from damage done to the parathyroid glands in the course of strumectomy, is capable of being held in check by the administration of cod-liver oil or irradiated ergosterin, even without the addition of parathyroid extract (Holtz, 1933; Holtz, Gissel & Rossmann, 1934), or with its addition in small doses (Boothby, 1931, 1932; Boothby, Haines & Pemberton, 1930, 1931; Boothby & Woltman, 1935); these facts and several experiments based on the principle of substituting vitamin D for the parathyroid extract which has become deficient in parathyroidectomized animals (Jones, 1926; Urechia & Popoviciu, 1928; Brougher, 1928; Shelling, 1930; Comel, 1930; Taylor, Weld, Branion & Kay, 1931; Spreter von Kreudenstein, 1937, 1938; all quoted by Thoma, 1941), would seem to indicate a similarity in the actions of vitamin D and the parathyroid hormone. Both regulate the calcium metabolism, and each of them is capable of replacing the other, should such a replacement become necessary as a result of its deficiency. Since vitamin D is found to be able to replenish in a vicarious manner the amount of calcium of which the body has been deprived by the decreased function of the parathyroid glands, the divergent views concerning the relationship between the dental lesions caused by parathyroidectomy and chronic fluorine poisoning on the one hand, and the calcium regulating influence of vitamin D on the other, would thus appear to be reconciled.

In the present survey of the incidence of mottled teeth amongst the 1700 people examined this altered conception of the causation of the dental lesion has been taken into account. The large number of people who were classified in the first survey (Spira, 1942 *a, b*) as not having mottled teeth, because their dental lesion may have been due, according to the then prevailing state of our knowledge, to 'hypoplasia', rickets, infectious diseases or avitaminosis, have now been classified under one heading, namely 'mottled teeth' (Pl. 4, figs. 2-3). No cognisance has been taken this time of the degree of mottling, and only the presence or absence of the lesion has been noted. The result of this survey reveals, as seen in Table 2, the not altogether astonishing fact that as many as 48 % of the 1700 people examined exhibited some degree of mottling of their teeth. Even with this more accurate analysis the incidence of the dental lesion does not fully reveal the true picture. There still remains the number of people who lost their teeth. No doubt some of these teeth were mottled. As many as 10 % of the people examined were completely edentulous. Another 10 % were deprived of all their upper teeth; a few teeth, however defective they may have been, when left in the lower jaw of the people belonging to the latter category, did not

qualify them as completely edentulous. The aggregate number of completely edentulous and nearly edentulous people thus amounted to 20 %. Furthermore, it was reported already on former occasions (Spira, 1928, 1933) that severe forms of gingivitis similar to, if not identical with, alveolar pyorrhoëa were one of the more frequent signs produced by an irritant contained in the drinking water and aluminium cooking utensils. The co-existence of mottled teeth with gingivitis has also been observed by Ainsworth (1933), Gaud, Charnot & Langlais (1935), Dean (1936) and Dean & Elvove (1936). In the present investigation as many as 17 % of those examined exhibited gingivitis in a variable degree sufficient to produce bleeding of the gums. The extent to which people are losing their teeth not on account of decay but because of this affection is well known. No attempt is being made to touch here upon the problem of the utter neglect of oral hygiene which has been frequently encountered in the course of the investigation. It is needless to emphasize that this neglect contributes markedly towards the justification for the oral condition of the population of this country being proverbial. Suffice it to say that no fewer than one person in five has been found from the

Table 2. *Incidence of dental dystrophies*

	Numbers examined					
	Total 1700		Men 850		Women 850	
	Inc.	%	Inc.	%	Inc.	%
Mottled teeth	821	48	409	48	412	48
Gingivitis	286	17	155	18	131	15
Toothless maxilla	160	10	109	13	51	6
Completely edentulous	160	10	121	14	39	5
'Horse-teeth'	165	10	70	8	95	11
Separated teeth	172	10	76	9	96	11

Inc. = Incidence.

present survey to have to depend on a complete set of artificial teeth or, at least, on one which replaces all the maxillary teeth. This contingent will in due course be replenished from those 17 % of the people examined who were found to suffer from gingivitis. The large number of those in whom only a few teeth were missing and replaced by artificial dentures have not been recorded.

An appearance of the teeth, said to be characteristic of the Anglo-Saxon race and known as 'horse-teeth', has been investigated in the present survey along with another anomaly in which the teeth, mostly those in the maxilla, are spaced widely apart (Pl. 4, fig. 4). Whereas the former has, so far as I know, never before been thought of as a dystrophy due to fluorine poisoning, the latter has been described as a condition frequently co-existent with misshapen teeth and with koilonychia (Weech, 1929), and with dystrophies of the nails and hair (Jacobsen, 1928; Clouston, 1929; Hill, 1933), and attributed to endocrine disturbances or to a congenital ectodermal defect of unknown origin (Goeckermann, 1920; MacKee & Andrews, 1924). 20 % of the people examined in the present series exhibited one or the other of these dental dystrophies. Malalignment of the teeth, due to crowding, with the resulting impaction, tilting and rotation of their axis, was a frequent feature. Such a malposition has been regarded as a result of endocrine disturbances (Gilford, 1904; Touraine & Soulignac, 1937).

THE HAIR

The hair originates from an invagination in the Malpighian layer, which is placed between the epidermis and dermis. This invagination pushes down into the dermis forming the follicle. At the base of the follicle the papilla of the hair develops. For a middle-aged man to start to lose his hair to an extent which indicates approaching baldness has for time immemorial been considered to be a 'natural' process to which nobody would give a thought. When, however, alopecia affects young men it assumes a pathological significance, but reference to its aetiology is scarce in the dermatological literature, and, accordingly, also the results of its treatment are modest. It was only when loss of hair was found to accompany other conditions, already known to be due to endocrine disturbances, that alopecia, too, was suspected as being caused in the same way. Yet, the frequency with which alopecia was found to occur simultaneously with mottled teeth was too striking to be missed. Pl. 4, fig. 9 shows this association distinctly. As many as 317 out of 1099 men and women, that is to say 29 % of those found to have mottled teeth in the first survey (Spira, 1942c), complained of loss of hair. Amongst them a few young men were found to be completely bald. A great number of the victims of alopecia showed the beginnings of sparse-

Table 3. *Incidence of hair dystrophies*

	Numbers examined					
	Total 1700		Men 850		Women 850	
	Inc.	%	Inc.	%	Inc.	%
Alopecia	445	26	224	26	221	26

ness of the hair, such as would appear to be 'natural' if they were about 20 years older. In some cases alopecia was associated with brittleness and fragility of those hairs which still remained. In the present inquiry, as will be seen from Table 3, 26 % of the people examined stated that they were losing their hair. An association of alopecia with dystrophies of the teeth was described by Weech (1929); and with onychopathies by Nicolle & Halipré (1895), White (1896), Eisenstaedt (1913), Goeckermann (1920), Friedmann (1921), Clouston (1929), Brain (1930), Broekema (1933) and Boothby & Woltman (1935).

THE SKIN

The co-existence of affections of the skin with those of the teeth, nails and hair has been demonstrated on former occasions (Spira, 1928, 1933, 1942c, d). Pardo-Castello (1941) described several dermatoses accompanied by onychodystrophies, and quoted numerous writers on the subject. None of these, however, appears to have ever made any suggestion as to the cause of either the dermatoses concerned, or of the onychodystrophies, or of the co-existence of both these lesions. But when the co-existent lesions of the skin and nails were found to be accompanied by those of the hair and teeth (Thurnam, 1848; MacKee & Andrews, 1924; Tobias, 1925; Jacobsen, 1928) the possibility of these trophic changes being due to endocrine deficiency was mentioned, but the suspicion that the parathyroid glands might be at fault has hardly ever arisen. It should, however, be remembered that fluorine as an aetiological factor producing mottled teeth,

through the parathyroid glands being primarily affected in a manner analogous with that in which Erdheim produced dental lesions by means of parathyroidectomy (1906), has been recognized by Bergara (1927), Chaneles (1929b) and Pavlovic & Tihomirov (1932).

In addition, it has been clinically demonstrated (Spira, 1942 c, d) that, like mottled teeth, mottled nails and some dermatoses are also produced by fluorine, since treatment directed against fluorosis brought about the disappearance of both the lesions of the nails and those of the skin. The dermatoses concerned are 'dhobi-itch', furunculosis, urticaria and facial eczema. 'Dhobi-itch' is a skin disease occurring between and underneath the toes, and more especially in the third and fourth interdigital spaces. Excoriations, rhagadae and deep fissures develop which produce a sensation of burning and itching. Often the epidermis is raised, and a vesicle sometimes of considerable size is formed which, as a result of scratching, breaks and exudes a slightly turbid fluid occasionally tinged with blood. When secondary infection is superadded, a cellulitis of the foot may develop. The condition which has for many years been considered as being due to a fungus infection, although very often no fungus could be detected, has been observed to take its course with or

and 8 show cases of cheiropompholyx associated with lesions of the nails.

It has been shown by me on former occasions (1928, 1933) that the disease picture produced by the irritant contained in the drinking water and aluminium cooking utensils is very similar to that of chronic arsenical poisoning. It was for this reason that the latter has in the past been suspected by me in cases of gastro-intestinal disturbances, peripheral neuritis and various dermatoses of obscure origin. A search for arsenic, however, revealed in these cases negative results. It is also known that one of the external manifestations of chronic arsenical poisoning is the occurrence of multiple warts on any part of the body, but more especially on the hands and fingers. In the present investigation as many as 17 % of those examined have on inspection been found to be afflicted with warts on the hands and fingers. People who stated that they have had verrucae in the past, but who did not exhibit them at present, were not included. These verrucae often appear in crops, in place of those which may have been removed by means of excision or treatment by radium and deep X-rays. A few such cases yielded in a dramatic manner in the past to treatment directed against fluorosis. Such cases of verrucae would thus appear to suggest that these multiple neoplastic structures are capable of being produced by a chemical noxon, such as arsenic, fluorine or substances belonging, from the point of view of their mode of action, to the same group of poisons.

In a discussion on mottled teeth (Dean, 1936) it has been suggested that freckling of the skin is a cutaneous dystrophy which corresponds to mottled teeth, and should, therefore, be designated as 'mottled skin', by which name it is popularly known. An association of freckled skin with dystrophies of the nails, hair and teeth has been described by Gilford (1904) and by MacKee & Andrews (1924), and with mottled teeth by Black & McKay (1916). In the present investigation 11 % of the people examined exhibited a mottled skin on parts of the body not exposed to the action of light and air. Freckles found on parts exposed to the sun were not recorded.

THE BONES

The tissue which has been longest known to be affected by fluorine is bone. It was also through the changes affecting the skeleton that the parathyroid glands were recognized as organs which regulated the calcium metabolism of the body. Such changes as osteosclerosis, osteoporosis, exostoses, osteomalacia and fragility of the bones have been described by numerous writers as being due to a disturbed calcium metabolism resulting from the protracted action of small amounts of fluorine. Day (1940) described stiffness of the back and other skeletal disturbances resembling rheumatoid or arthritic conditions observed in India in an area with endemic fluorosis, and Kemp, Murray & Wilson (1942) attributed some cases of spondylosis deformans (spondylitis osteoarthritica) in this country to chronic fluorine poisoning. So far as I know, however, a dystrophy affecting the metatarsal bones and the phalanges of the toes, characterized by their tendency to grow out of alignment in a medial, lateral, dorsal or plantar direction have never before been described as frequently co-existent with

Table 4. Incidence of dystrophies of the skin

	Numbers examined					
	Total 1700		Men 850		Women 850	
	Inc.	%	Inc.	%	Inc.	%
'Dhobi-itch'	1000	59	529	62	471	55
Furunculosis	533	31	368	43	165	19
Urticaria and other dermatoses	532	31	252	30	280	33
Warts	297	17	125	15	172	20
Freckles ('mottled skin')	183	11	64	8	119	14

without treatment, and the attack to end by the epidermis peeling in large flakes, but to disappear promptly, without local treatment, when the régime directed against fluorosis has been carried out. The widespread occurrence of the affection in this country is a well-known fact, and it was shown in my first survey (1942c) that an inquiry into its incidence revealed that 19 % of people found to have mottled teeth were affected. In the present series the survey was made not by interrogation, but by personal inspection of the feet. The inspection revealed the presence of 'dhobi-itch' in not fewer than 59 % of all the 1700 people examined. This result is not surprising, since every case of peeling between the toes has been duly recorded as one of 'dhobi-itch'. Thus 'dhobi-itch' should be regarded as another visible sign of chronic fluorine poisoning. The incidence of furunculosis and that of urticaria and other dermatoses has been investigated in the present series in exactly the same manner as in the first survey (Spira, 1942c), namely by means of interrogation. It was found to be practically identical in both surveys. Amongst the 'other dermatoses' seborrhoic dermatitis and eczema intertrigo seemed to predominate. Cheiropompholyx and dysidrosis, known to be refractory to every kind of local treatment, have in several instances in the past yielded promptly to one directed against fluorosis. Pl. 4, figs. 7

signs and symptoms of fluorosis. The effect of such dystrophy is the production of deformities known as 'hammer-toes' and 'hallux valgus' (Pl. 4, figs. 5, 6). The latter is produced by thickening of the epiphyses forming the metatarso-phalangeal joint, which consequently becomes distorted to such an extent as to lead to a lateral deviation of the toe. A similar tendency of the other toes may lead to overlapping. Instead of meeting, the big toes form an angle of up to as much as 80°, with

the big toe became sporadically inflamed, and a condition developed which was so closely similar to gout that it is suggested that these are the cases which are often referred to as 'the poor man's gout'. In long-standing cases of this type X-ray examination reveals the presence of osteo-arthritis. Further clinical observation should be capable of clearing up the question as to what extent fluorine plays a part in the production of diseases thought to be due to metabolic disturbances.

Table 5. *Incidence of bone dystrophies*

	Numbers examined					
	Total 1700		Men 850		Women 850	
	Inc.	%	Inc.	%	Inc.	%
Hallux valgus	1099	65	511	60	588	69

the apex of the angle formed by the metatarso-phalangeal joints. As seen from Table 5, 65 % of the 1700 people examined were found to be afflicted with some degree of hallux valgus. The malalignment of the metatarsal bones leads to formation of flat feet or of pes cavus. Such crippling of the feet in the population of this country deserves further careful clinical investigation in place of the time-honoured explanation that the several dystrophies of the feet are due to wearing tight shoes. Moreover, in many cases the metatarso-phalangeal joint of

SUMMARY

Dystrophies due to chronic fluorine poisoning in organs regulated by the parathyroid glands, namely the skin and its appendages, the nails, teeth and hair, and also in bones, are here shown to be unusually common in this country. They should be regarded as easily detectable signs of a serious disturbance of the calcium metabolism of the body. Fluorine is a poison of which the action, in many respects, is similar to that of arsenic. It is the task of the authorities responsible for the health of the nation to search for ways and means capable of minimizing its effect.

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REFERENCES

- AINSWORTH, N. J. (1933). *Brit. Dent. J.* **55**, 233.
 BEČKER, S. W. (1930). *Arch. Derm. Syph., Chicago*, **21**, 957.
 BECKS, H. & RYDER, W. D. (1931). *Arch. Path.* **12**, 358. Cited by Thoma (1941).
 BERGARA, C. (1927). *C.R. Soc. Biol., Paris*, **97**, 600.
 BERGER, W. (1880). *Schmidts Jb.* **187**, 114.
 BLACK, G. V. & MCKAY, F. S. (1916). *Dent. Cosmos*, **58**, 129, 477, 627, 781, 894.
 BLOCH, B. (1926). *Schweiz. med. Wschr.* **56**, 753.
 BLOCH, B. & STAUFFER, W. (1929). *Arch. Derm. Syph., Chicago*, **19**, 22.
 BOOTHBY, W. M. (1931). *Proc. Mayo Clinic*, **6**, 535.
 BOOTHBY, W. M. (1932). *Proc. Mayo Clinic*, **7**, 361.
 BOOTHBY, W. M., HAINES, S. F. & PEMBERTON, J. J. (1930). *Proc. Mayo Clinic*, **5**, 180.
 BOOTHBY, W. M., HAINES, S. F. & PEMBERTON, J. J. (1931). *Amer. J. Med. Sci.* **181**, 81.
 BOOTHBY, W. M. & WOLTMAN, H. W. (1935). *Proc. Mayo Clinic*, **10**, 87.
 BRAIN, R. T. (1930). *Proc. Roy. Soc. Med.* **23**, 163, 1482.
 BROEKEMA, J. H. (1933). *Acta derm.-venereol., Stockh.*, **14**, 113.
 BROUGH, J. C. (1928). *Amer. J. Physiol.* **86**, 538. Cited by Thoma (1941).
 CHANELES, J. (1929a). *Rev. Soc. Argent. Biol.* **5**, 317, 336, 340, 352, 376, 386.
 CHANELES, J. (1929b). *C.R. Soc. Biol., Paris*, **102**, 860, 863.
 CHENG, R. G. & REID, E. (1937). *Chin. J. Physiol.* **12**, 223.
 CLOUSTON, H. R. (1929). *Canad. Med. Ass. J.* **21**, 18.
 COMEL, M. (1930). *Arch. fisiol.* **29**, 123. Cited by Thoma (1941).
 COOPER, Z. K. (1930). *Arch. Derm. Syph., Chicago*, **21**, 1007.
 DAY, C. D. M. (1940). *Brit. Dent. J.* **68**, 409.
 DEAN, H. T. (1936). *J. Amer. Med. Ass.* **107**, 1269.
 DEAN, H. T. & ELVOVE, E. (1936). *Amer. J. Publ. Hlth*, **26**, 567.
 EISENSTAEDT, J. S. (1913). *J. Amer. Med. Ass.* **60**, 27.
 ELLER, J. J. & ANDERSON, N. P. (1928). *Med. J. Rec.* **127**, 318.
 ERDHEIM, J. (1906). *Mitt. Grenzgeb. Med. Chir.* **16**, 632.
 FOERSTER, O. H. (1916). *J. Cutan. Dis.* **34**, 1.
 FOSSUM, C. (1928). *J. Amer. Dent. Ass.* **15**, 1701.
 FRASER (1927). *Arch. Derm. Syph., Chicago*, **16**, 359.
 FRIEDMANN, M. (1921). *Arch. Derm. Syph., Berl.*, **135**, 161.
 GAUD, CHARNOT & LANGLAIS (1935). *Odontologie*, **73**, 373.
 GILFORD, H. (1904). *Practitioner*, **73**, 188.
 GOECKERMANN, W. H. (1920). *Arch. Derm. Syph., Chicago*, **1**, 396.
 HELLER, J. (1926). *Dtsch. med. Wschr.* **52**, 786.
 HERTZ, A. F. (1915). *Practitioner*, **94**, 107.
 HILL, A. M. (1933). *Arch. Derm. Syph., Chicago*, **28**, 66.
 HOFFMANN, R. (1908). *Arch. Derm. Syph., Berl.*, **89**, 381.
 HÖJER, J. A. (1924). *Acta paediatr.* **3**, 8. Cited by Thoma (1941).
 HOLTZ, F. (1933). *Arch. klin. Chir.* **177**, 32.
 HOLTZ, F., GISSEL, H. & ROSSMANN, E. (1934). *Dtsch. Z. Chir.* **242**, 521.

- HOWE, P. R. (1920). *Dent. Cosmos*, **62**, 586. Cited by Thoma (1941).
- HOWE, P. R. (1922). *J. Amer. Dent. Ass.* **9**, 55. Cited by Thoma (1941).
- JACOBSEN, A. W. (1928). *J. Amer. Med. Ass.* **90**, 686.
- JONES, J. H. (1926). *J. Biol. Chem.* **70**, 647. Cited by Thoma (1941).
- KAZNELSON, P., REIMANN, F. & WEINER, W. (1929). *Klin. Wschr.* **81**, 1071.
- KEMP, F. H., MURRAY, M. M. & WILSON, D. C. (1942). *Lancet*, **2**, 93.
- KNOUFF, R. A., EDWARDS, L. F., PRESTON, D. W. & KITCHIN, P. C. (1936). *J. Dent. Res.* **15**, 291.
- MC EWEN E. L. (1916). *J. Cutan. Dis.* **34**, 15.
- MACKEE, G. M. & ANDREWS, G. C. (1924). *Arch. Derm. Syph., Chicago*, **10**, 673.
- MAIRE, M. & WÖRINGER, F. (1930). *Bull. Soc. Fr. Derm. Syph.* **37**, 1214.
- MELLANBY, M. (1929). *Spec. Rep. Ser. Med. Res. Coun., Lond.*, no. 140. Cited by Thoma (1941).
- MELLANBY, M. (1930). *Spec. Rep. Ser. Med. Res. Coun., Lond.*, no. 153. Cited by Thoma (1941).
- MENDES DA COSTA, S. & VAN DER VALK, J. W. (1919). *Urol. Cutan. Rev.* **23**, 159.
- MILLAN, G. (1937). *Bull. Méd.* **51**, 137.
- MONTGOMERY, D. W. (1916). *J. Cutan. Dis.* **34**, 285.
- MURRAY, F. A. (1921). *Brit. J. Derm.* **33**, 409.
- NICOLLE, G. & HALIPRÉ, A. (1895). *Ann. Derm. Syph., Paris*, **6**, 675, 804.
- PAPASTRATIGAKIS, C. (1922). *Paris Méd.* **2**, 475.
- PARDO-CASTELLO, V. (1941). *Diseases of the Nails*. Springfield, Illinois.
- PAVLOVIC, R. A. & TIHOMIROV, D. M. (1932). *C.R. Soc. Biol., Paris*, **110**, 497.
- REID, E. (1936). *Chin. J. Physiol.* **10**, 259.
- REID, E. & CHENG, R. G. (1937). *Chin. J. Physiol.* **12**, 233.
- ROSEBURY, T. & FOLEY, F. (1934). *J. Dent. Res.* **14**, 359. Cited by Thoma (1941).
- SAMUEL, H. C. (1919). *Brit. J. Child. Dis.* **16**, 217.
- SHELLING, D. H. (1930). *Proc. Soc. Exp. Biol., N.Y.*, **28**, 303. Cited by Thoma (1941).
- SIMONS, R. (1939). *Arch. Derm.* **178**, 400.
- SINGER, P. L. (1931). *Arch. Derm. Syph., Chicago*, **24**, 112.
- SMITH, M. C. (1936). *J. Dent. Res.* **15**, 281.
- SMITH, M. C., LANTZ, E. M. & SMITH, H. V. (1932). *J. Dent. Res.* **12**, 149.
- SMITH, M. C., LANTZ, E. M. & SMITH, H. V. (1935). *J. Amer. Dent. Ass.* **35**, 817.
- SPIRA, L. (1928). *Franco-Brit. Med. Rev.* **5**, 1, 61.
- SPIRA, L. (1933). *The Clinical Aspect of Chronic Poisoning by Aluminium and its Alloys*. London.
- SPIRA, L. (1941). *Lancet*, **2**, 23.
- SPIRA, L. (1942a). *Lancet*, **1**, 649.
- SPIRA, L. (1942b). *Brit. Dent. J.* **73**, 149.
- SPIRA, L. (1942c). *Edinb. Med. J.* **49**, 707.
- SPIRA, L. (1942d). *J. Hyg., Camb.*, **42**, 500.
- SPIRA, L. (1943). *J. Hyg., Camb.*, **43**, 69.
- SPIRA, L. & GRIMBLEBY, F. H. (1943). *J. Hyg., Camb.*, **43**, 142.
- SPIRA, L. & KOLESAR, P. D. (1942). *Brit. Dent. J.* **73**, 233.
- SPRETER VON KREUDENSTEIN (1937). *Dtsch. Zahn-, Mund- u. Kieferhkl.* **4**, 208. Cited by Thoma (1941).
- SPRETER VON KREUDENSTEIN (1938). *Dtsch. Zahn-, Mund- u. Kieferhkl.* **5**, 777. Cited by Thoma (1941).
- STUBENBORD, J. G. & STUBENBORD, W. D. (1935). *Arch. Derm. Syph., Chicago*, **32**, 761.
- TAYLOR, N. B., WELD, C. B., BRANION, H. D. & KAY, H. D. (1931). *Canad. Med. Ass. J.* **25**, 20. Cited by Thoma (1941).
- THOMA, K. H. (1941). *Oral Pathology*. London.
- THOMPSON, H. B. (1928). *J. Amer. Med. Ass.* **91**, 1547.
- THURNAM, J. (1848). *Proc. Roy. Med. Chir. Soc.* **31**, 71.
- TOBIAS, N. (1925). *J. Amer. Med. Ass.* **84**, 1568.
- TOURNAINE, A. & SOULIGNAC (1937). *Bull. Soc. Fr. Derm. Syph.* **2**, 305.
- URECHIA, C. I. & POPOVICIU, G. (1928). *C.R. Soc. Biol., Paris*, **98**, 405. Cited by Thoma (1941).
- VELU, H. (1933). *Bull. Acad. Méd.* **110**, 799.
- WEECH, A. A. (1929). *Amer. J. Dis. Child.* **37**, 766.
- WHITE, C. (1934). *J. Amer. Med. Ass.* **102**, 2178.
- WHITE, C. J. (1896). *J. Cutan. Genit. Urin. Dis.* **14**, 220.
- WOLBACH, S. B. & HOWE, P. R. (1925). *J. Exp. Med.* **42**, 753. Cited by Thoma (1941).
- WOLBACH, S. B. & HOWE, P. R. (1926). *Arch. Path.* **1**, 1. Cited by Thoma (1941).
- WOLBACH, S. B. & HOWE, P. R. (1928). *Arch. Path.* **5**, 239. Cited by Thoma (1941).
- WOLBACH, S. B. & HOWE, P. R. (1933a). *J. Exp. Med.* **57**, 511. Cited by Thoma (1941).
- WOLBACH, S. B. & HOWE, P. R. (1933b). *Amer. J. Path.* **9**, 275. Cited by Thoma (1941).

EXPLANATION OF PLATE 4

Fig. 1. Finger-nails of a woman showing leukonychia, which affects nearly their whole surfaces, together with those of her 17 months old child showing longitudinal striation (onychorrhexis) and chalky white specks (leukonychia punctata).

Figs. 2, 3. Mottled teeth.

Fig. 4. Mottled teeth spaced widely apart.

Fig. 5. Hallux valgus, with metatarsal bones and phalanges out of alignment.

Fig. 6. Side view of foot illustrated in Fig. 5, showing hammer-toes.

Figs. 7, 8. Cheilopompholyx associated with nails showing pits.

Fig. 9. Association of alopecia with mottled teeth.

Figs. 1-9.