FLUORINE ALOPECIA

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(With Plate 6)

When the American investigators Smith, Lantz & Smith (1932) found that mottled enamel was the result of the protracted ingestion, during the period of calcification of the teeth, of toxic amounts of fluorine contained in the drinking water in a concentration higher than one part per million (1 p.p.m.), equivalent to gr. 1/120 of fluorine to a pint of water, they little realized that their important findings would become a starting-point for investigations extending far beyond their attempt at eliminating an unsightly dental appearance only. Since mottling of the teeth was thus demonstrated to be a lesion produced by a chemical noxon which found its way through the gastro-intestinal tract into the blood circulation, it became obvious that chronic fluorine poisoning (fluorosis) would manifest itself in other parts of the body as well.

In this country the widespread occurrence of mottled teeth enabled Spira (1942 *a*, *b*, *c*) to study the symptomatology of fluorosis, and to show, in particular, that mottled teeth were frequently, though not invariably, accompanied, amongst other signs and symptoms, by certain dermatoses, various dystrophies of the nails, and alopecia. He concluded that, as the organs of ectodermal origin so affected are regulated by the parathyroids, it is probably these endocrine glands themselves which are, in some as yet unexplained manner, first involved, and that their lowered function leads to a disturbance of the calcium metabolism of the body. The striking similarity of mottled teeth, as produced by fluorine in animal and man, to the dental lesions which resulted from experimental parathyroidectomy described by Erdheim (1906, 1911) has already been pointed out by earlier investigators (Bergara, 1927; Chaneles, 1929 *a*, *b*; Pavlovic & Tihomirov, 1932). It was, however, Spira (1942*d*, 1943*a*) who was, so far as can be ascertained, the first to show that, in fact, several pathological changes observed by him to accompany mottled teeth were likewise identical with the corresponding lesions demonstrated by Erdheim (1906) in his parathyroidectomized rats. As far as loss of hair, here to be discussed, is concerned, it was complained of by as many as 29% of 1099 men and women afflicted with mottled teeth (Spira, 1942*c*), and by 26% of 850 men of the average age of 29 years and by an equal percentage of 850 women of the average age of 21 years, examined at random (Spira, 1944*b*). In some cases the premature loss of hair was so striking a feature that the diagnosis of fluorosis could be made even before the anticipated possibility of the simultaneous presence of mottled teeth and mottled nails had been actually confirmed. Loss of hair following experimental parathyroidectomy, on the other hand, was also described by Erdheim (1906), and confirmed by Adler & Thaler, as quoted by him.

Yet, the widespread occurrence of alopecia in this country escaped the attention of the medical profession for a long time, and the victims themselves accepted the explanation that it is a natural process, hereditary in origin. Nothing has happened to justify the hope that the search for any concrete aetiological factor which might produce it in the great majority of cases would be successful. This failure was, in part, due to the fact that any such search was restricted, in the age of bacteriology and parasitology, to looking mainly for an infective agent in the causation of alopecia in seemingly healthy individuals. In those relatively rare cases in which an infection had been established, the timely removal of the offending factor was, as a rule, sufficient to restore the growth of hair. Since, however, in the absence of infection, it has not yet proved possible to determine the cause of the frequent occurrence of alopecia either by laboratory investigation or by clinical observation, every effort to eradicate it appears to have been given up as futile.

The fact cannot be denied that a high proportion of young men in this country suffer from an extensive loss of hair. It is sufficient to look round at one's neighbours in such places as the train, the park, at meetings, etc., for the frequent occurrence of alopecia in varying degrees to become obvious. Pl. 6 gives merely an idea of its extent in this country. The men here shown, 378 in number, represent a true cross-section of the bald portion of its population. They belonged to different spheres of life, and came from different parts of the country. Their average age was 31 years, and they were selected for no other reason than for their more or less pronounced degree of baldness at this relatively young age. Alopecia following any obvious disease of the scalp was excluded, and only cases accepted which appeared to have occurred in a manner similar

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to those occurring without any known cause at a later period of life. As seen from the appended table,

Ages of 378 *men* (= 100 %) *shown in Pl.* 6

Age	No.	%	Age	No.	%
19	3	0·8	29	27	7·2
20	0	0·0	30	33	8·7
21	6	1·6	31	30	7·9
22	0	0·0	32	43	11·4
23	2	0·5	33	39	10·3
24	11	2·9	34	34	9·0
25	16	4·2	35	35	9·3
26	6	1·6	36	29	7·7
27	23	6·1	37	10	2·6
28	28	7·4	38	3	0·8

Average age 31 years.

the oldest of them were 38 years old, at which age even in the past extensive loss of hair leading to permanent baldness would not yet have been regarded as a 'natural' process. Many of the men here presented, including the older ones, stated that they were becoming bald when they were only about 18 or 20 years old. The statement concerning the onset of baldness at this early age recurred so often in my investigation that a conclusion seemed justified that it was during this short span of life that toxic amounts of fluorine contained in the drinking water were ingested, which was, *ceteris paribus*, sufficient to produce it. It should, in this connexion, be remembered that loss of hair is, in most cases, a slow process which usually increases in its extent gradually, even before attention is drawn to it, and that it is, for this reason, as a rule impossible to determine the exact onset of alopecia. It is, therefore, reasonable to assume that, to become the cause of baldness, a period of less than 18–20 years would be required for toxic amounts of fluorine to be ingested in a certain concentration, e.g. such as has proved to be sufficient to produce mottled teeth, that is, 1 p.p.m. or higher. Kylin & Dicker (1939) found that in their series alopecia started before the age of 30 years, with a predilection for about 23–25 years, thus conforming to the figures given by Genner (1929), as quoted by them.

In any study of the problem of alopecia, the considerably lesser degree in which it is encountered in females warrants further investigation. The simplest explanation of this divergence might be that the degree of the toxicity of fluorine, like that of any other poison, depends on the quantity ingested and on the length of time during which it has been taken. It is submitted that, since it has been found, in the survey referred to above (Spira, 1944*b*), that loss of hair has been complained of by the sex groups in equal numbers, the difference in its degree may be due to ingestion of smaller quantities of fluids containing toxic amounts of fluorine in the case of females. Such an explanation cannot, however, apply generally, for a similar divergence in the degree of most of the other signs and symptoms of fluorosis would be expected, but was actually not always observed. The same objection would apply to any endeavour to explain the difference in the degree of alopecia encountered in the sexes by the interdependence of the functions exercised by the various glands which constitute the endocrine system, and by the possibility that the ovarian hormones act in an antagonistic manner upon the function of the parathyroids.

Some further light may be thrown upon the problem of loss of hair in adult life by the study of the so-called congenital alopecia (hypotrichosis), and of cases in which loss of hair began in early childhood. Though little has been written on the loss of hair in adults, considerable investigation has been carried out on alopecia occurring during the first few years of life. Reports on the subject concur in stating that alopecia which makes its appearance at this very early age is distributed amongst both sexes in equal numbers (Baer, 1907; Marcuse, 1907, as quoted by Baer, 1907; Petersen, 1915; Nobl, 1935). As in adult life so also in the juvenile form alopecia may occur as a solitary abnormality, without a corresponding involvement of the other organs of ectodermal origin (Ziegler, 1897; Bettmann, 1902; Kraus, 1903; Baer, 1907; Petersen, 1915). In other cases of congenital and juvenile alopecia, loss of hair has been found to be accompanied by dystrophies of the skin, teeth and nails, singly or in combination (Nicolle & Halipré, 1895; White, 1896; Pinkus, 1899; Waelsch, 1910; Buschke, 1911*a*, *b*; Eisenstaedt, 1913; Goeckermann, 1920; Friedmann, 1921; Oliver & Gilbert, 1926; Clouston, 1929; Weech, 1929; Brain, 1930; Broekema, 1933; Boothby & Woltman, 1935; Koch, 1939; Kylin & Dicker, 1939). Conversely, dystrophies of the skin, teeth and nails, respectively, may or may not be associated with alopecia.

The pathological changes in the various organs regulated by the parathyroid glands, when occurring singly as they sometimes do, or in several combinations as they are found often, though in a variable degree, have, however, in many cases been recorded in such a manner as to create the impression that each of them was a syndrome *sui generis*. Such is not the case. The effect of hypoparathyroidism has been shown by Erdheim (1906) not always to be uniform in its manifestations. Although anatomically his experimental rats were found to have been deprived of all the parathyroids in an equally effective manner, not all of them exhibited the same degree of response. In hypoparathyroidism, for some reason at present unknown, one organ regulated by the parathyroids is picked out more than another, with the result that the organs less affected exhibit changes not nearly commensurate with those most affected. One or other of them may even

escape altogether. It was this strange preponderance of pathological changes in one of the various organs rather than in others, which led to the common belief that the several disease pictures are separate clinical entities. Rather must it be assumed that hypoparathyroidism is a disease which exhibits a polymorphism in its manifestations, with one feature or one set of features appearing in the forefront on one occasion, and remaining in the background on another.

Moreover, alopecia making its appearance at birth in one case, and in infancy, in childhood or in adolescence in another; or occurring in several members of a family in one instance, and with a hereditary tendency in another: all these subdivisions in the classification of alopecia, and placing them in watertight compartments, led to a state of confusion which stood in the way of a correct interpretation and correlation of signs and symptoms belonging to the same disease picture, namely, that of hypoparathyroidism. Hence the various designations used in the past, such as infantile, juvenile, adolescent, adult, familial, hereditary, etc., indicate nothing but the time and the environmental circumstances in which the disease made its appearance in its various manifestations. In discussing the problem of congenital familial hereditary alopecia, Petersen (1915) states that the term 'congenital' is incorrect in so far as it is not the anomaly which is inborn *sensu strictiori*, but the pathological condition or the pathological predisposition which is the cause of the loss of hair. Spira (1943*c*), too, emphasizes that it is not heredity, in its strict meaning of the word, which plays an essential part in the causation of another effect of hypoparathyroidism, namely, of otosclerosis, but an inherited predisposition to its acquisition, should an opportunity of protracted exposure to toxic amounts of fluorine arise. It is thus the fluorine, ingested by the mother, which exerts its deleterious effect on the foetus in its intra-uterine life to an extent sufficient to produce 'congenital' or 'hereditary' alopecia; and it is the fluorine derived from drinking water and from other sources containing it in toxic amounts which produces signs and symptoms of fluorosis in several members of a family who ingested it for some length of time in quantities sufficient to cause, amongst others, 'familial' alopecia.

Alopecia having been found by several investigators to be not a uniform disease picture *per se*, but only one of the symptoms of a systemic condition, attention has been directed for a long time to finding its cause. Bettmann (1902), in discussing the congenital form of alopecia, considers a developmental defect in intra-uterine life to be the immediate cause. He states that the possibility cannot be ignored that the changes in the scalp are perhaps not at all to be traced to a primary defective anlage, but to secondary atrophic conditions which have been caused by some foetal morbid process. Kraus (1903) considers that in his cases of alopecia congenita familiaris the hair papillae which were present at one time in the foetus underwent extensive regressive changes in intra-uterine life. Petersen (1915) believes that the secretion of a noxious agent should be regarded as an aetiological factor. Several writers suspect some kind of endocrine disturbance as the cause of alopecia, and others specify one or other particular gland within the endocrine system or a combination of these glands as being at fault. Spira (1942 *c*, *d*, 1944 *b*) accuses fluorine as a causative agent on clinical grounds, although no improvement of alopecia followed the treatment directed against chronic fluorine poisoning. Petersen (1915) is right when he states that, whatever the treatment, it can be of no avail in most cases, since its success, even if follicles are at all present, depends on whether or not the papilla is still able to form a hair. A similar explanation is given by Spira (1943*c*, 1944*b*) for the failure to bring about a *restitutio ad integrum* in lesions caused by fluorine, such as otosclerotic foci, mottled teeth or mottled nails, any attempt at regeneration of a tissue which has been damaged beyond repair being unreasonable and doomed to failure.

Alopecia areata has so far always been kept out altogether from the study of the problem of generalized loss of hair, and considered as a separate entity. For this reason the few cases encountered in the present survey of the incidence of premature baldness amongst young men in this country have not been included. Special attention has, however, been paid to the recorded results of experimental and clinical observation made on this condition in the past, with a view to ascertaining whether some of the findings are not applicable to the larger problem of alopecia as well. Outstanding amongst the numerous experimental investigations on the action of thallium are those of Buschke (1900, 1901, 1903, 1911 *a*, *b*, 1913, 1922), Buschke & Peiser (1922 *a–e*, 1925) and Spitzer (1921, 1922 *a*, *b*). Buschke & Peiser found that loss of hair, either diffuse or even total, or appearing in large areas or small patches, which was its chief effect, was accompanied by cataract, disturbance of growth and development, and diminution or loss of sexual functions, as well as severe lesions of the skeleton in the sense of rachitic changes. According to them (1925), the cause of all these signs and symptoms was a disturbance of the endocrine apparatus (in particular that of the parathyroid glands), and a disturbance of the vegetative nervous system, which is intimately connected with the endocrine apparatus, thus giving a sufficient explanation of the occurrence of all the changes. They believe that this is a case of a central origin of thallium alopecia ('Diesen allen Symptomen ge-

meinsame Ursache sahen wir in einer Stoerung im endocrinen Apparat (vorzugsweise wohl in den Epithelkoerperchen) und des mit ihm in enger Verbindung stehenden vegetativen Nervensystems, womit saemtliche Veraenderungen eine ausreichende Erklaerung fanden. Es handelt sich hier um eine zentrale Genese der Thalliumalopezie'). Buschke (1913), in turn, quotes the ophthalmologist Ginsberg who was the first to point out that cataract following the protracted ingestion of minute doses of thallium may be due to changes in the parathyroids. Spitzer (1922 *a*, *b*), too, stresses that it is the parathyroid glands which are the damaged central organ causing alopecia, and recalls the well-known fact that loss of hair is one of the more frequent features in tetany.

On comparing these important findings on the action of thallium with those recorded by Erdheim (1906) on experimental parathyroidectomy it will be seen that they are identical. Alopecia, cataract and rachitic changes may occur in either of them. Thus administration of thallium produces a systemic condition which is analogous with that produced by the surgical removal of the parathyroid glands. Similarly, the action of fluorine has been shown on several occasions (Spira, 1942 *d*, 1943 *a*, *c*, 1944 *a*, *b*) closely to resemble the effects of radical parathyroidectomy. It would, therefore, appear that the actions of both thallium and fluorine are likewise identical; in both of them the involvement of the parathyroid glands resulting in hypoparathyroidism is stressed as a salient feature. Moreover, Buschke & Jacobsohn (1922) found that addition of calcium temporarily weakens to a considerable extent, or even temporarily abolishes, the effect of thallium, and Spira (1942 *c*, *d*, 1943 *a*, *c*, 1944 *a*, *b*) reported that the régime directed against fluorosis, with the administration of calcium forming an essential part of the régime, considerably alleviates such signs and symptoms caused by fluorine as are not yet beyond the stage of regeneration.

This analogy is remarkable for yet another reason. The second part of Buschke & Peiser's (1925) theory of the aetiology of alopecia areata, namely, the trophoneurotic theory, is based by them on the outstanding experiments of Joseph (1886, 1889) who produced areas of alopecia in cats by removing the second spinal ganglion. In Joseph's experiments alopecia followed only the extirpation of the ganglion itself, or it resulted from the section of the cervical nerve at a point peripheral to the ganglion, but it did not occur when the nerve had been cut centrally. According to Sklarz (1923), quoted by Buschke & Peiser (1925), in the first two events the sympathetic fibres which run from the ganglion cervicale supremum to the ganglion spinale II must have been cut as well, whereas the sympathetic bundle is spared if the nerve is cut at a point placed at a distance centrally away from the ganglion. Lévy-Franckel (1922) traces alopecia areata to an irritation of the vago-sympathicus, and Lévy-Franckel & Juster (1922, 1923, 1925, 1928) to a disturbance of the endocrino-sympathetic system. Jordan (1925) and Galewsky (1925) also state that alopecia areata is due to a central disturbance, probably sympathico-endocrine in character, and Roxburgh (1929) believes that loss of hair may be produced by a toxaemia acting by way of the sympathetic system, or by way of some endocrine glands.

Turning, for the sake of comparison, to the part played by the vegetative nervous system in the action of fluorine upon the organs regulated by the parathyroid glands, attention is directed to a report on a case of congenital ectodermal dysplasia with involvement of the skin and its appendages, the hair, teeth and nails (Spira, 1946), a condition which I attributed to the action of fluorine in intra-uterine life. The patient, a man 31 years old, has always had sparse hair, but even this sparse hair began noticeably to fall out, without any apparent reason, at the age of 19 years. This led to an advanced stage of baldness, most marked on the vertex, where only a few hairs were present. In view of the fact that the organs of ectodermal origin affected are controlled by the parathyroid glands, and also in view of the close association of the endocrine apparatus with the vegetative nervous system, the patient's response to the intravenous injections of adrenalin, atropine and pilocarpine was investigated. These pharmacodynamic tests established the presence of an increased irritability of the parasympathetic nervous system, a vagotonia. Similar results were obtained by Singer (1921) and Bloch (1926) in an endeavour to elucidate the relationship between dermatoses and allied conditions on the one hand, and an abnormal function of the endocrine system on the other.

This is not to infer that in cases of ectodermal dysplasias there is always an alteration in the tone of vagus only. Siebert (1922) states that, although the reaction to the various drugs experimentally administered in such cases is in some respects indicative of vagotonia, it indicates in other respects an increased irritability of the sympathetic nervous system. Sklarz (1921), too, believes that, because of the close relationship of the vagus and the sympathetic, both of which play an essential rôle in sustaining life and health, and also because of the vital dependence of both these systems upon the central nervous system, any disturbance in the sphere of action of the vagus may also bring about a disturbance in that of the sympathetic. It should also be remembered that, although the two great divisions of the vegetative nervous system act, to a certain extent, in a manner antagonistic to each other, it is on the harmoniously balanced co-ordination of their performance that the smooth working

of the body relies. A disturbance in any one of these divisions is bound to lead, in some degree, to a disturbance in the other as well. In fluorosis, the occurrence of such signs and symptoms as dermographia, urticaria, pruritus, chilblains, migraine, tachycardia, enuresis nocturna, hypersecretion of sweat in the hands and feet, as well as disturbance of the vasomotor mechanism (Raynaud's disease, acrocyanosis and acroasphyxia, angioneurotic oedema, etc.), indicates that, in many cases, the tonus of the sympathetic nervous system may also be increased.

Findings of this kind lead irresistibly to the conclusion that the parathyroids may represent not the point of direct attack in the action of both fluorine and thallium, but a junction, an intermediate vital link in a chain connecting the vegetative nervous system with the organs affected. It is thus the vegetative nervous system which is probably affected by the chemical noxa, fluorine and thallium respectively, before the parathyroids are secondarily involved; and it is the resulting disturbance in its equilibrium which, in turn, brings about a disturbance in the endocrine apparatus. The fact established by Chiari & Froehlich (1911), that reducing the calcium content causes a high degree of irritability of the vegetative nervous system, seems to lend support to this probability. The solution of the problem of whether it is more often the sympathetic or the parasympathetic nervous system in which fluorine brings about a predominant degree of hyperirritability by reducing the calcium content; and also the solution of the problem of whether, in any one case, sympathicotonia may occur at one point in time and vagotonia at another, thus giving rise to amphotonia, awaits further elucidation. More important still, the final question as to whether it is indeed the vegetative nervous system which is the primary or even perhaps the only target of attack by fluorine must also be left to further research. Lachmann (1941) suggests the possibility that in the process of hypoparathyroidism a calcium-regulating centre in the brain is involved.

Buschke (1900, 1901, 1903, 1911*a*, *b*, 1913, 1922), Buschke & Peiser (1922*a*, *b*, *c*, *d*, *e*, 1925), and Spitzer (1922 *a*, *b*), all succeeded in producing alopecia congenita in young rats, analogous with alopecia congenita (hypotrichosis) in man, by protracted administration of thallium to the mother during pregnancy. Furthermore, by producing alopecia in a litter of new-born rats when the mother was subjected to the protracted action of thallium only after their birth, they also demonstrated that thallium was excreted into the milk on which the young were reared. It is, on the other hand, of equally great interest to recall that Velu (1933), Smith & Smith (1935), Knouff, Edwards, Preston & Kitchin (1936), Murray (1936), Reid & Cheng (1937) and Day (1940) either experimentally produced or clinically observed the occurrence of deciduous mottled teeth in the young through the administration of fluorine to pregnant rats. Thus the placenta has been established to be permeable both to thallium and fluorine.

In view of these findings, the long-sought aetiological factor in the causation of congenital alopecia (hypotrichosis) is here submitted, on clinical grounds, to be traceable to the protracted ingestion of fluorine by the pregnant mother. This suggestion is strengthened by the observation that, in women, loss of hair may follow pregnancy (Poehlmann, 1913; Jordan, 1925; Nobl, 1935; Kylin & Dicker, 1939), and by the knowledge that, in its ultimate effect, the action of fluorine consists in its ability to precipitate calcium salts which are stored in the body as a material indispensable for sustaining the vitality of most of the organic functions, with subsequent lowering of the calcium content in blood and tissues. This deleterious action has been shown (Spira, 1943*c*) to apply also to the frequent onset of otosclerosis during pregnancy, the decalcified foci in the labyrinthine capsule being, just as much as the loss of hair is, the result of the mother sharing her calcium with the foetus at a time when calcium was already deficient through the action of fluorine.

The similarity between the actions of thallium and fluorine respectively is yet further strengthened by the observation of certain conditions which, considered by themselves, may appear to be a matter of coincidence and to be unimportant, but assume significance when attention is drawn to their co-existence by several writers, and are found to fit exactly into the picture of hypoparathyroidism. Buschke & Peiser (1925) observed the occurrence of pes cavus and claw toes in their cases of alopecia areata; and Spira (1944*b*) found that fluorosis is frequently accompanied by pes cavus, flat feet and hammer toes. A co-existence of flat feet with otosclerosis was noted by Frey & Orzechowski (1917, 1920), who were the first to draw attention to an association of otosclerosis with latent tetany due to parathyroid insufficiency. In addition, Jones & Atkins (1875), Buschke & Peiser (1925) and Spira (1944*b*) reported wide spacing of the teeth (diastema), and malalinement of the teeth in their respective investigations. A gross malalinement of the teeth associated with congenital alopecia has also been recorded by Bettmann (1902).

A tendency to low blood pressure and to constipation was noted by Bengtson (1933) and by Kylin & Dicker (1939) to accompany alopecia. Low blood pressure is one of the characteristic effects of the protracted action of fluorine, and Spira (1942*c*) found that as many as 49% of 1099 men and women with mottled teeth suffered from constipation. Tomasi (1940) reported a co-existence of alopecia

with gout and arthritic diathesis, and Spira (1944 *b*) drew the attention to so-called 'poor man's gout', with X-ray appearances of osteoarthritis in hallux valgus occurring frequently in chronic fluorine poisoning.

Bettmann (1904) quotes Jacquet who observed a frequent co-existence of coryza and enlargement of the tonsils with alopecia areata, and Barber (1921 *a*, *b*) found that, in alopecia areata, infective foci in the teeth and gums, the tonsils, nasopharynx or nasal sinuses, and severe colds play an all-important part. An association of alveolar pyorrhoea with 'mottled teeth', on the other hand, has been noted by several investigators, and Spira (1944*a*) pointed out that ulcerative and aphthous stomatitis, nasopharyngitis, atrophic rhinitis, sinusitis, enlargement of the tonsils and recurrent tonsillitis, and frequent attacks of 'common cold' belong to the symptomatology of chronic fluorine poisoning.

Some of the individual features of the various ectodermal dystrophies due to chronic fluorine poisoning are also similar. The hair falling out is comparable to onycholysis, a condition in which a loosening of a mottled nail occurs without the slightest warning, even to the extent of its being lifted out of its bed and falling off, and with mottled teeth becoming so loose as a result of defective calcification that they can be pulled out with the fingers. The brittleness and fragility affecting the hair in alopecia, too, mentioned also by Freshwater (1915), Petersen (1915) and Koch (1939), is one of the characteristics common to both mottled teeth and mottled nails, as well as to bones decalcified by the action of fluorine. In some cases the fragility of the hairs which still remain in alopecia is so pronounced that it can be noticed at a glance; they remain short and appear to lack the tendency to grow (Spira, 1942*d*, 1944*b*).

From the point of view of their toxic effect on the skin Spitzer (1922 *b*) lays considerable emphasis upon the similarity in the action of thallium to that of arsenic. He refers to the analogy of alopecia in adult life in its relationship to congenital alopecia as running parallel with the analogy, emphasized on several occasions by Jadassohn, concerning the relationship of arsenical palmar and plantar hyperkeratosis and the congenital keratoma palmae et plantae. On the other hand, the close relationship, evident in many respects, of the actions of fluorine and arsenic was pointed out on several occasions by Spira who, in particular, reported (1944*b*) that at least 17% of 1700 men and women examined by him at random in this country were afflicted with warts on the hands and fingers. It is an established fact that warts are one of the external manifestations of chronic arsenical poisoning.

It has also been shown by me on previous occasions that, like arsenic, fluorine is to be found as an impurity in many media. It has been ascertained (Spira, 1943 *b*) that the filter powder employed in this country in the process of purification of water by filtration through metal filters, often contains it, thus affecting the drinking water. It may be present in the soil, and will contaminate the water if the clay, lime or chalk which forms its bed contains fluorine anywhere in its course from its origin down to the well. The iron pipes sunk into the well, the storage tanks made of iron, cement or concrete, and the iron pipes leading from the tank to the tap from which the water is drawn for drinking and cooking purposes, may all contain fluorine as an impurity, and become a further source of contaminating the water supply. In the production of aluminium cooking utensils, the mineral cryolite, Na_3AlF_6 (or $3NaF.AlF_3$), which after purification contains roughly 50% fluorine, is an essential raw material. Since in the process of preparing food the aluminium is readily corroded by both acids and alkalies, the fluorine compounds contained therein are set free and contaminate the food.

It follows that toxic alopecia due to fluorine is a condition which can be prevented. Moreover, measures directed against further deterioration of an incipient alopecia can be reasonably expected to be successful in those favourable cases in which the hair papillae have not yet been damaged beyond the stage of recovery.

SUMMARY

A high proportion of young men in this country suffer from premature baldness. Alopecia is not a disease entity but one of the outstanding features of chronic fluorine poisoning (fluorosis). It is frequently associated with dystrophies of other organs of ectodermal origin, namely, those of the skin and its other appendages, the teeth and nails. Since all these organs are regulated by the parathyroid glands, it is concluded that alopecia, too, is produced by fluorine acting through the medium of these endocrine glands. By its ability to precipitate the calcium content of the body, fluorine may lay the foundation for alopecia already in intra-uterine life, or produce it at any time after birth.

The action of fluorine is closely similar to that of thallium. There is ample evidence to show that in both fluorosis and chronic thallium poisoning the vegetative nervous system is vitally involved.

Toxic alopecia due to fluorine is preventable.

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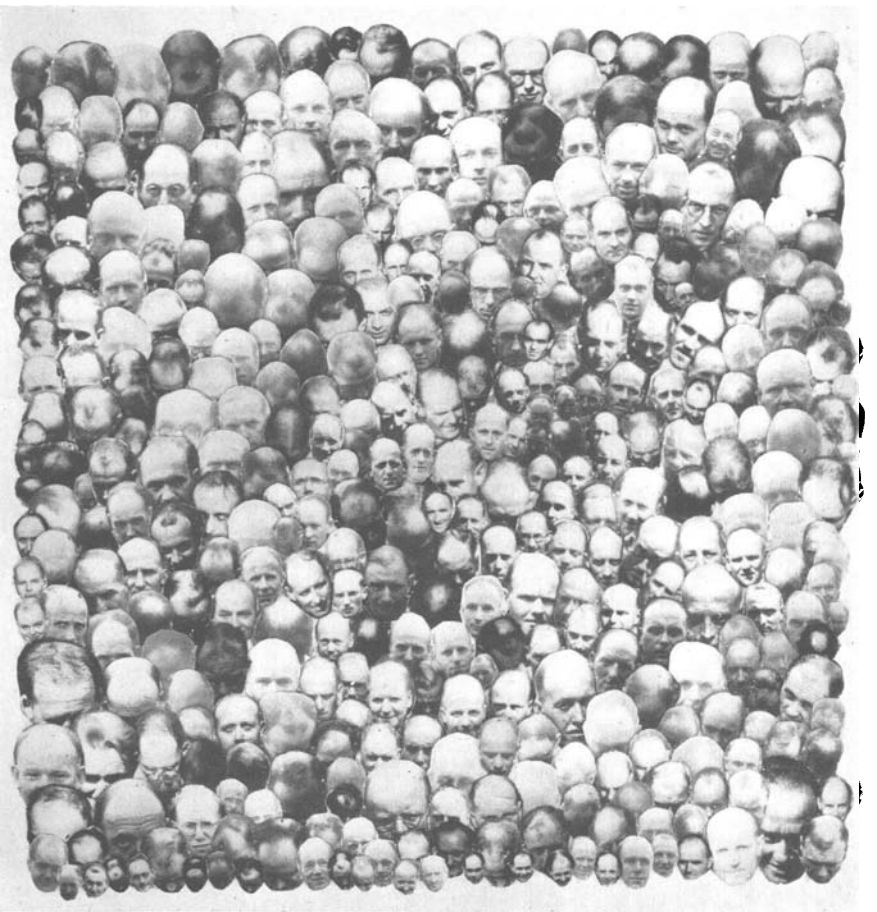

Showing baldness, not following any obvious disease of the scalp, in 378 men of the average age of 31 years.

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