

INDUSTRIAL FLUOROSIS

by

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SUMMARY: This is a review of findings on workers in an aluminum plant with industrial fluorosis. Early signs of the disease are nocturnal back pains and restriction of the rotation of the trunk. Stage I of the disease usually occurs after 10 years, stage II after 15 years and stage III after 20 years. The diagnosis was established at an early stage through biopsies of the iliac crest by histological and micro-analytical determinations of fluoride. A fluoride level exceeding 4000 ppm in the iliac crest ash was found to be associated with typical signs of fluorosis. The early histological changes including the microradiographic findings and typical foci of calcification in the corticalis are demonstrated.

Three necropsies in patients with fluorosis at different stages are reviewed. One of them showed a lesion in the cells of the anterior horn of the spinal cord which was believed to be related to fluorine. Cristallographic studies revealed that crystals of fluorotic bone mineral had become more slender. Increase in gastric acidity was associated with greater sensitivity toward fluoride. Prophylaxis and treatment of industrial fluorosis are discussed.

Industrial fluorosis is a rare occupational disease in workers of fluorine processing or manufacturing industries (factories for the production of aluminum, cryolite, hydrofluoric acid, fertilizers, insecticides, and glass. Industrial fluorosis was recognized for the first time in 1932 by Møller and Gudjonsson (1) in Danish cryolite workers. Roholm

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thoroughly described this disease in several monographs (2-4).

In 1944 Peperkorn and Kähling (5) described the first cases of industrial fluorosis in Germany. They found signs of fluorosis in 34 out of 47 workers in a hydrofluoric acid factory in Dohna (Saxony). In 1958, Fritz (6) extended the investigations in this factory to 156 persons and he found typical skeletal fluorosis in 67. Since then additional cases of industrial fluorosis were recorded from the U.S.A. (7-11) Great Britain (12-15), Soveit Union (16-18), Norway (18), France (20, 21), Switzerland (22, 23), and Germany (24-29).

We have also had the opportunity to investigate some patients who were suffering from industrial fluorosis, and we have reported about it in several communications (30-37). These patients are workers in smelters of an aluminum factory. Producing aluminum electrolytically, cryolite (Na_3AlF_6), aluminum fluoride (AlF_3) and soda (Na_2CO_3) are added to alumina (Al_2O_3 , aluminum oxide) as fluxing agents. As a result the melting temperature is reduced from 2000° to 950° C. The anode-effect, the steam pressure of the salts, and the mechanic manipulations (breaking off the crusts, changing the anodes) cause the development of gas and dust which vapors contain hydrofluoric acid (HF), carbontetrafluoride (CF_4), cryolite (Na_3AlF_6), and aluminum fluoride (AlF_3). The fluorides are inhaled and swallowed. Because of the high temperatures the workers drink as much as 2 to 5 liters of liquid during one shift (29).

Symptomatology

In the initial stages, the complaints of the patients are not remarkable. At first they experience vague rheumatic pains, then the pains become localized in the spine, especially in the lumbosacral region. Later, a sensation of stiffness in the lumbar and cervical spine develop.

However, we also found patients with slight radiological changes (subtle signs or stage O-I). who complained of intense pains in the spine and in the large joints. On the other hand, some patients whose fluorosis was radiologically distinct were almost without complaints.

A study of Domesle (29) showed that, of 400 aluminum workers, 44% had stiffness and pains in the back beginning in the lumbar and extending up to the cervical spine; 25% had pains in the large joints; 63% stinging and hypesthesia in the arms and legs after working hours. Many workers complained of pains at night and while resting, but movement caused them to disappear. In addition, attacks of vertigo, ringing and rushing in the ears, anorexia, constipation, nausea and, in 60%, cough and dyspnea appeared frequently.

Clinical Findings

Clinically one finds at first a restriction of movement of the spine, affecting eventually the cervical spine. As an early clinical sign of fluorosis we observed the restriction of rotation of the trunk (Fig. 1). This finding occurred when bone changes became demonstrable roentgenologically in the early stage.

Fig. 1

Restriction of Rotation of Trunk, An Early Sign of Fluorosis



Left: Normal Right: Restricted rotation

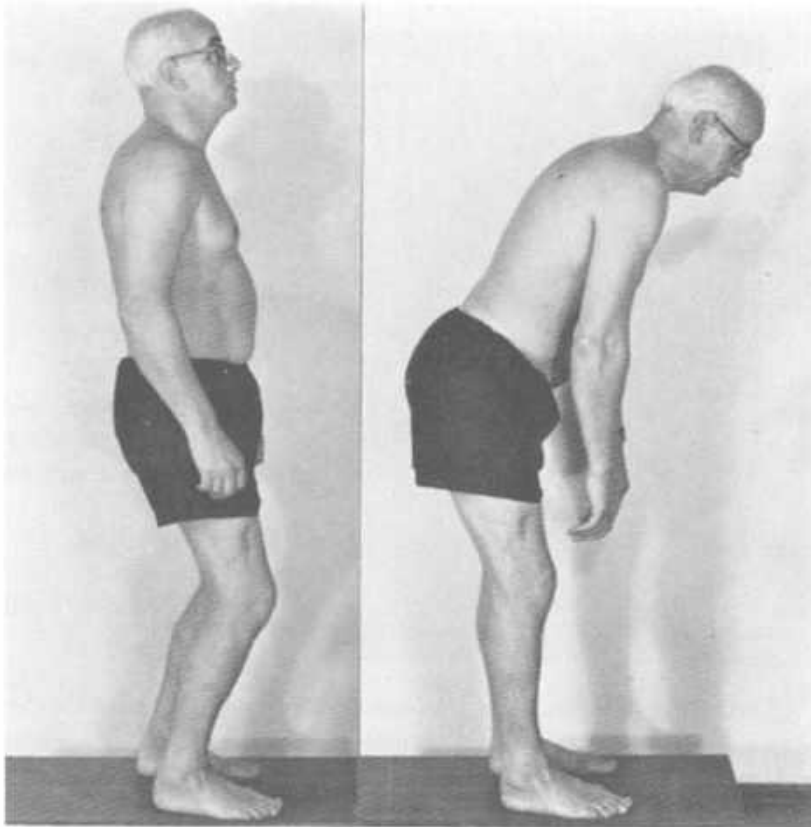
Later on, complete stiffening of the spine (Fig. 2) and a marked restriction of the vital capacity and respiratory movement ensue. In the advanced stage, a more or less extensive restriction of movements of the large joints, especially of the hip-joints, supervenes. Therefore the clinical features resemble closely those of Bechterev's disease.

Laboratory Findings

Biochemically, calcium, phosphorus, and the acid phosphatase levels of the serum did not show any distinct changes; the erythrocyte sedimentation rate was normal, there was no evidence of anemia. The plasma alkaline phosphatase is sometimes increased. Domesle (29), however, did not find any correlation between the behavior of the alkaline phosphatase and the extent of the skeletal fluorosis.

Radiological Findings

In addition to the well-known radiological stage classification,

Fig. 2Stiffening of the Spine in Fluorosis Stage II

stage I to III according to Roholm (3), two prestages according to Fritz (6) have proved to be important in our investigations: the so-called Schwachzeichen (subtle signs) and the stage O-I. Concerning the subtle signs, which are only important in connection with a history of exposure to fluoride, a condensation of the bone-structure and an enlargement of the bone trabeculae in the lumbar spine are evident. In addition, there are subtle accompanying shadows along the tibia, fibula, radius, and ulna.

At the stage O-I (Fig. 3a, b) the structure of the thoracic spine has already increased in density, whereas in the lumbar region the normal structure of the bones begins to disappear. The periosteal appositions of new bone at the bones of the forearms and lower legs are more distinct.

For better proof of the periosteal appositions on these bones, we used slightly underexposed photographs similar to the kind of radiography employed for soft parts.

Fig. 3W. K., Fluorosis 0 to I

3a. Slight sclerosis of lumbar spine



3b. Periosteal new bone formation on radius

In distinction to Roholm's findings, we noted that a longer duration of exposure is required for the development of fluorosis. Subtle signs develop after an average period of 10.7 years (2 to 25 years), stage 0-I after 12.2 years (5 to 33 years), stage I after 15.7 years (8 to 38 years), stage II after 17.6 years (11 to 21 years), stage III after 19.5 years (19 to 20 years) of duration of employment (Table 1).

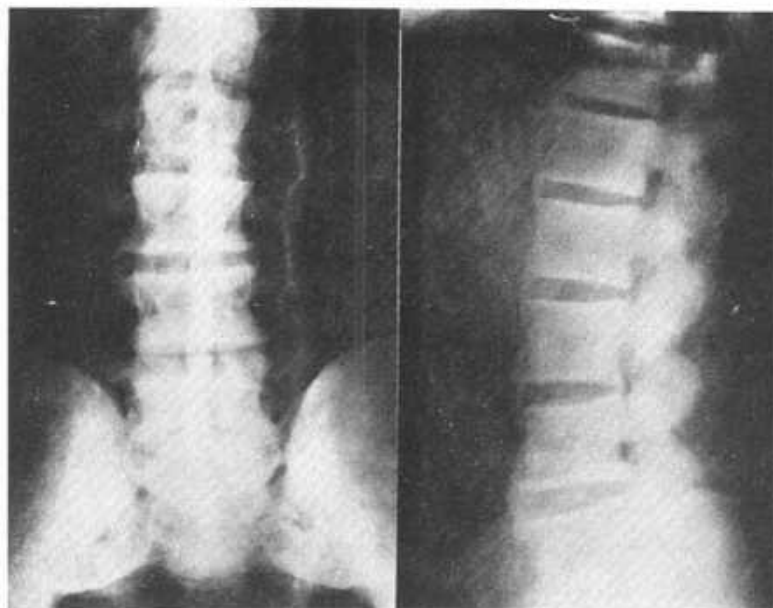
TABLE 1

Average Duration of Fluoride Exposure for the
Single Stages of Fluorosis (in years)

Vague symptoms (Schwachzeichen)	10.71 years	(2 - 25 years)
stage 0-I	12.15 years	(5 - 33 years)
stage I	15.70 years	(8 - 38 years)
stage II	17.6 years	(11 - 21 years)
stage III	19.5 years	(19 - 20 years)

The differences are due to different conditions of production: Roholm's data was concerned with a cryolite factory, whereas our experience was in connection with an aluminum factory where the fluoride concentration in the factory hall was lower.

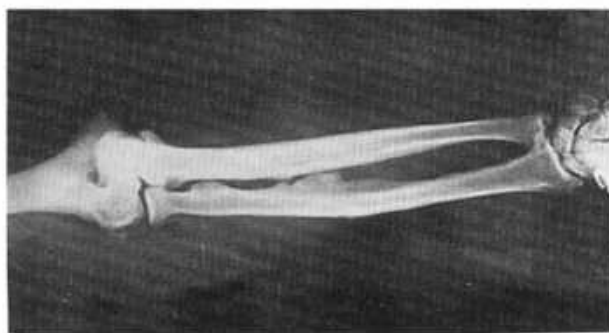
Figures 4a and b present bones of a 56 year-old patient with a fluorosis stage III. An extreme marble-like sclerosis of the bones with ossification of the longitudinal ligaments of the spine are noted. The

Fig. 4E. W., Fluorosis Stage III

4a. Marked sclerosis of lumbar spine; ossification of longitudinal ligaments.



4b. Marked sclerosis of the pelvis; distinct prominence of the muscular attachments; ossification of ligaments and sacroiliac joints.



4c. Calcified interosseous ligament between radius and ulna.

bone structure is no longer discernible, the sacroiliac joints are ossified. The formation of outgrowths and spurs at the muscular insertions and the ossification of the ligaments of the pelvic floor complete this picture. Outgrowths like a saw blade develop also between radius and ulna (Fig. 4c).

Pathologic-Anatomical Findings

We obtained necropsy findings of three patients with industrial fluorosis, a fluorosis III, a I-II, and an old fluorosis (3 years without any fluorine contact). The patients died from a traffic accident, a brain tumor, and a decompensated mitralinsufficiency respectively.

The pictures show the changes of the skeletal system in fluorosis stage III. Here we can see in the final stage a complete ossification of the longitudinal ligaments of the spine which has practically stiffened to a stick (Fig. 5a).

The small vertebral joints and the costovertebral joints are ankylosed by the ossification of the capsules, the muscular attachments protrude as exostoses. The spongy bone is condensed massively,

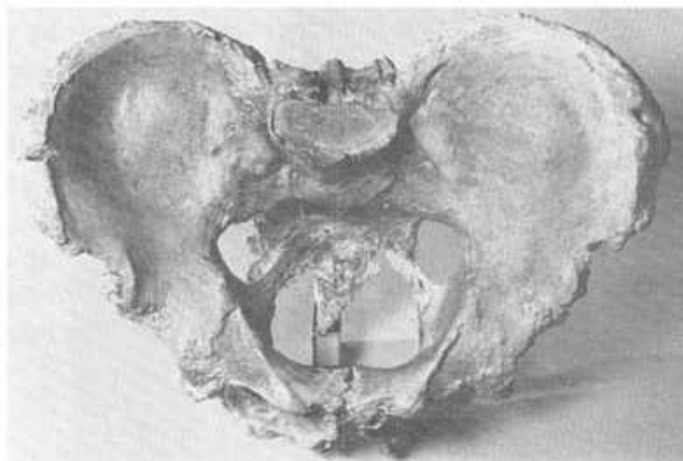
allowing hardly any space for bone marrow. The above-described changes are seen clearly on the pelvis (Fig. 5b): a projection of the muscular attachments on the iliac crest and pubic bone, ossification of the sacroiliac joints, of the pubic symphysis and of the ligaments of the pelvic floor. Peripherally, ossifications occur at muscular attachments. Saw-blade-like appositions are found at the inferior surface of the ribs between ulna and radius (Fig. 5c) and between the tibia and fibula. The long bones exhibit an increase in bone substance at the cross section with distinct thickening of the whole corticalis (Fig. 6).

Fig. 5

E. W., Fluorosis Stage III

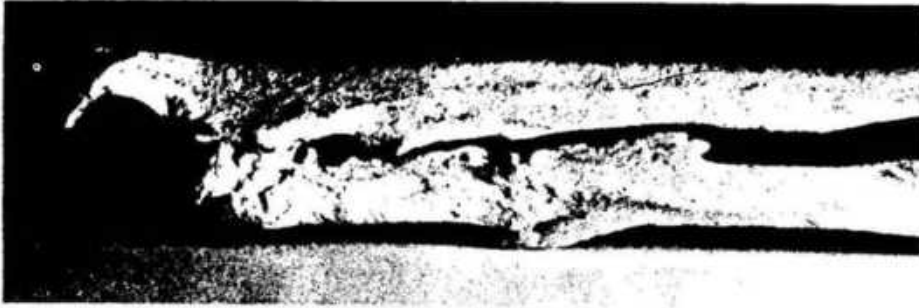


5a. Macerated preparation of spine



5b. Macerated preparation of pelvis

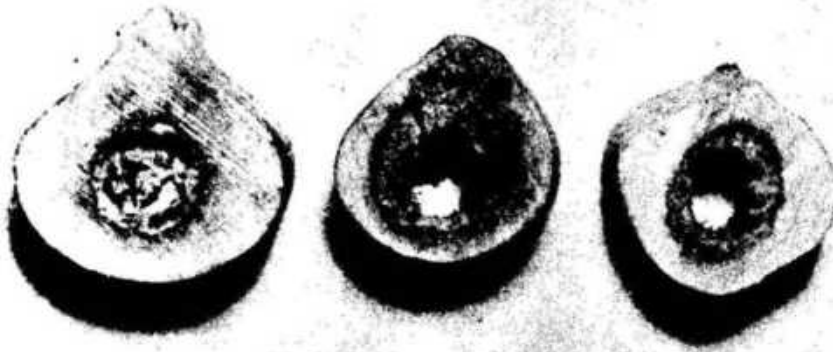
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5c. Macerated preparation of forearm

Fig. 6

Extensive Thickening of Corticalis of Femur



Fluorosis stage III (left) compared with two normal femora (center and right)

No pronounced narrowing of the intervertebral foramina and of the spinal canal in the cases with radiculomyelopathies such as was described by our Indian colleagues in endemic fluorosis (38-47) were encountered. We agree with Jolly (48) that in endemic fluorosis exostoses form in the canal of the cervical spine due to the custom in India of carrying heavy loads on the head. Changes in other organs which are not age-related such as damage to brain, liver, kidneys, and the vascular system were not found with one exception namely, the patient who experienced pathological changes in the nervous system which will be recorded later in another paper.

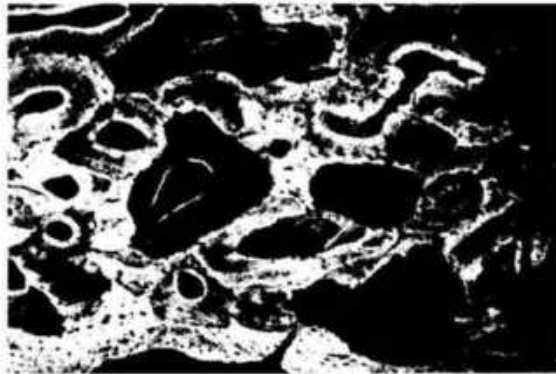
Histological Findings

Our histological findings are based on the analysis of 25 iliac crest needle biopsies and on 3 autopsied cases at different stages of

fluorosis, especially on the beginning stage of fluorosis. Since the details about the histological changes of industrial fluorosis were reported previously (32) the present discussion will be confined to a summary of what we observed: 1.) coarsening and condensation of the spongy bone, 2.) subperiosteal formation of fibrous bone with transformation into lamellar bone leading to the formation of exophytes, 3.) thickening and spongiosation of the corticalis, 4.) enlarged osteocyte cavities, 5.) irregular matrix formation with a high turnover rate (Fig. 7), mosaic structure of the cement-lines, formation of chalky granulae, and foci with a coarse fibrous structure, a high content of mucopolysaccharides, and with a trend to calcification (Fig. 8) which were described by us for the first time in 1972 (32,34).

Fig. 7

Microradiogram of Rib Showing a High Bone Turnover Rate



Fluorosis stage III (1:75)

Fig. 8

Typical Subperiosteal Focus with Coarsened Fibrous Structure

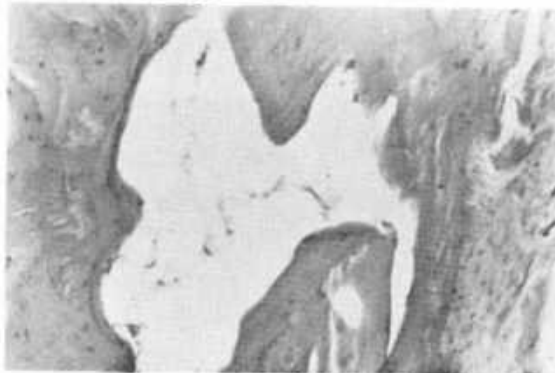


(H. E., 1:170)

These changes—subperiosteal formation of new bone, spongiosation of the corticalis as a symptom of endosteal bone resorption as well as disturbances of the matrix formation and of the mineralization—appear to be typical of fluorosis. Our most recent studies showed that the above-mentioned foci are situated, in most cases, in the corticalis near the periosteum and that the direction of their fibers does not correspond to the course of the surrounding osteons, but to that of the inserting tendinous fibers at the periosteum (Fig. 9). We consider them

Fig. 9

Small Subperiosteal Foci



Direction of fibers corresponding to those of periosteum (H. E., 1:120)

residues of connective tissue of former periosteum which were included in the bone formation and incrustated with calcium. The microradiogram, showing a high calcium content of these foci, supports this interpretation (Fig. 10)*. The bone structure did not reveal evidence of a secondary hyperparathyroidism.

The above-described findings become more distinct as the degree of fluoride intoxication advances with, however, wide individual variations.

Histochemically we observed distinct activities of the alkaline and acid phosphatase in some cases as well of the β -glucuronidase in the endosteal cells and osteocytes which constitutes additional evidence of increased bone turnover during chronic fluoride intoxication.

Fluorine Determination in the Bone Ash

We (34) have developed a special method in order to confirm the diagnosis of "fluorosis" in doubtful cases in the early stage or in sporadic

* I am grateful to Prof. Kuhlencordt, Hamburg, for the preparation of the microradiograms

Fig. 10Microradiogram of Iliac Crest, Fluorosis III

Typical focus of calcium accumulation (1:75)

dically occurring fluoroses: During the iliac crest biopsy a second bone cylinder is removed which is dried at a temperature of 105° C up to weight constancy and ashed for 2 hours at 450° C until the bone is white and porous. The fluorine determination is made according to a modified action of the method of Megregian (49) (determination of fluorine by distillation with perchloric acid and vapor and formation of a complex of circoneriochromcyanine). Here the fluorine contents could be related to the single radiological stages. The normal value is 0.08% fluoride (800 ppm). We found the first distinct histological and radiological changes in bones containing approximately 0.35% (3500 ppm) to 0.40% fluorine in the ash of the iliac crest (Table 2). Nearly the same limits were recorded by Sankaram and Gadekar (44) in human beings and by Schmidt and Rand (50) in cattle. Jackson and Weidmann (51) found higher values, whereas Freitag et al. (52) reported lower ones.

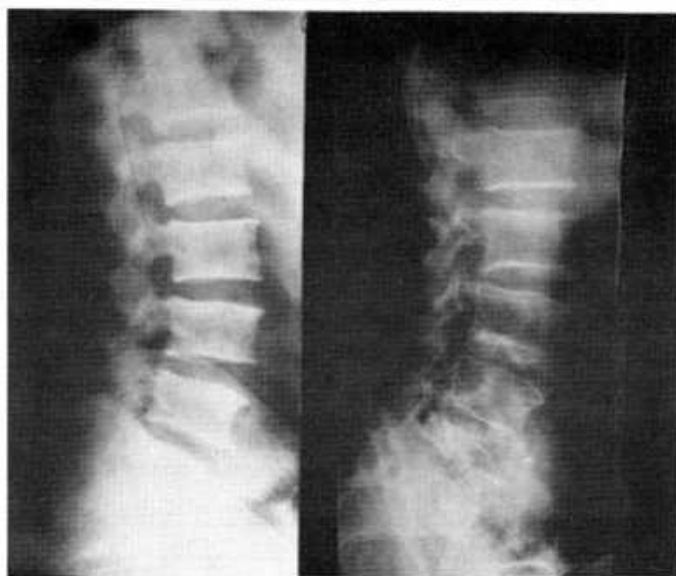
TABLE 2Fluoride Content of the Iliac Crest Ashed at Various Stages of Fluorosis

	<u>Percentage</u>	<u>PPM</u>
normal individuals	0.05 - 0.11	500 - 1100
Vague symptoms (Schwachzeichen)	0.35 - 0.45	3500 - 4500
stage 0 - I	0.50 - 0.55	5000 - 5500
stage I	0.60 - 0.70	6000 - 7000
stage II	0.75 - 0.90	7500 - 9000
stage III	> 1.0	> 10,000

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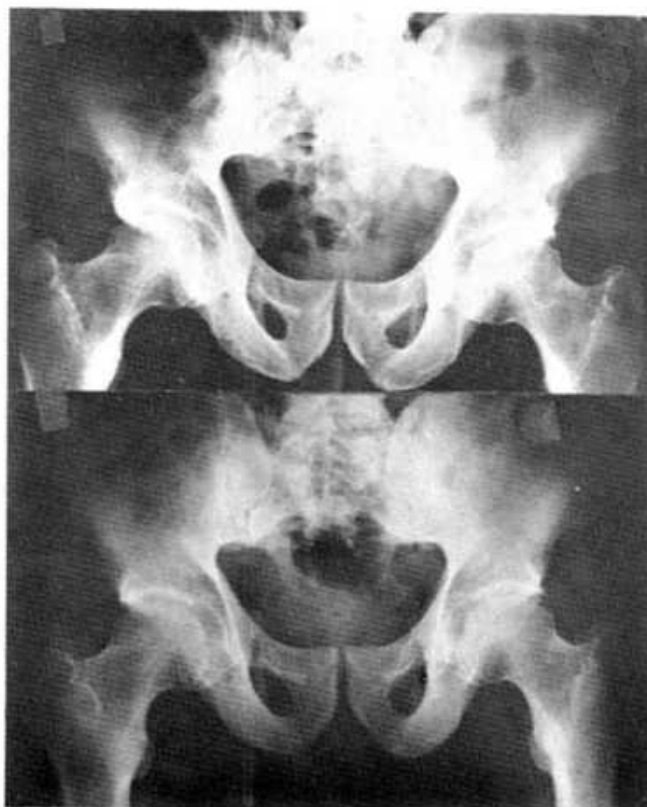
Special Observations

1. In 1939, Roholm (4) assumed that in fluorosis the degree of skeletal sclerosis declined after cessation of fluoride intake. Fritz (6, 27) made the same observation in 7 cases and Herbert and Francon in one (21). We confirmed this finding in 2 such cases. Their sclerosis decreased markedly within 2 years and 5 years respectively, whereas the ossifications in the ligaments persisted. In 1968, 63 year-old patient M. G. who had been working in an aluminum factory for 26 years, developed typical fluorosis, stage II. Following a change in occupation, the radiological check-up in 1973 showed a distinct decrease of the sclerosis (Fig. 11a, b). In 9 additional patients, the bone density remained unchanged for 5 years in spite of a change of employment.

Fig. 11aFluorosis Stage II Within Five Years

Note decrease of sclerosis in spine.

2. Whereas in general no serious neurological complications are reported in industrial fluorosis, as mentioned above, we nevertheless observed the following case. Patient E. N., 64 years-old, with fluorosis stage I-II died of a cerebral glioblastoma. In addition, the histological examination yielded distinct damage to the cells of the anterior horn of the spinal cord and localized destruction of muscle fibers. Whereas it appears that the brain tumor and fluorosis were coincidental, the damage to the spine and muscles could be related to the existing chronic fluoride intoxication.

Fig. 11bSame Patient as in Fig. 11a

Note decrease of sclerosis in pelvis.

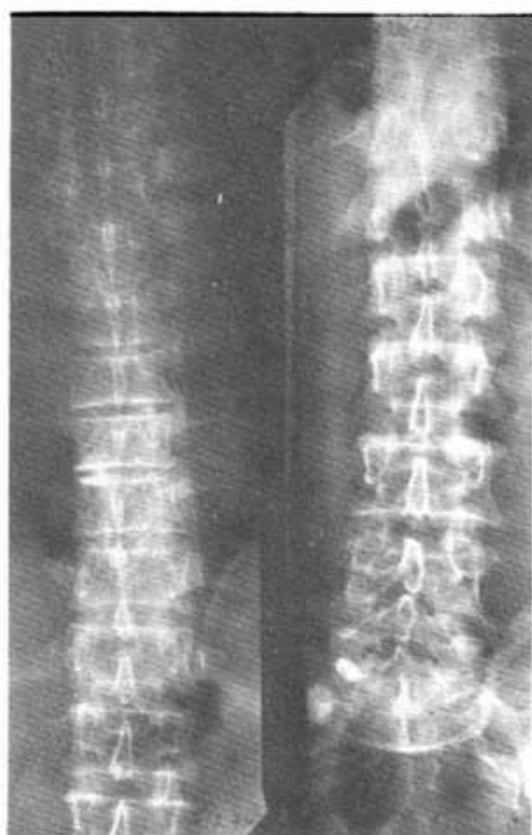
3. In several patients we failed to notice evidence of typical sclerosis in the radiogram. Instead, the picture of so-called "hypertrophic atrophy" was found as in the following case:

Patient G. M., 62 years-old, had been working for 20 years in an aluminum smelter. X-rays showed typical hypertrophic atrophy with slight periosteal appositions on the forearms and lower legs (Fig. 12 a-c). The fluoride content in the ash of the iliac crest was 0.63%. Histologically a thin spongy compacta and narrow bone trabeculae as well as coarsening spongy and hyperostotic cortical structures were found. We observed similar findings following therapy with sodium fluoride for osteoporosis. It is likely that a previously existing osteoporosis is superimposed upon fluorosis or the predominance of the fluoride-induced bone resorption in conjunction with thickening of the statically loaded bone structure may be responsible.

Additional Special Studies

1. Gastric acid and the stage of fluorosis: As reported pre-

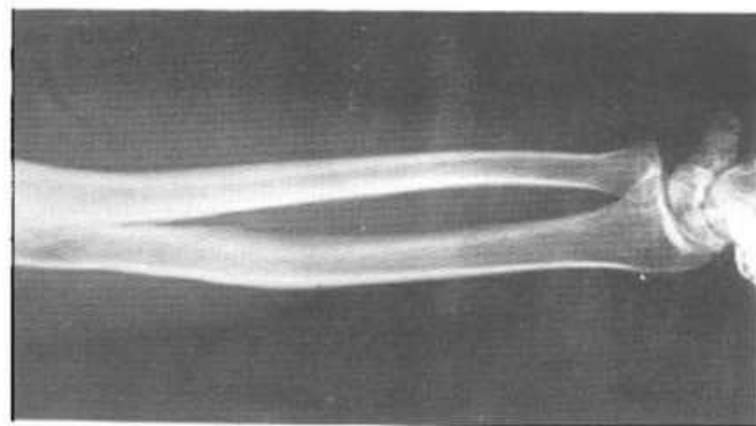
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Fig. 12"Hypertrophic Atrophy" of Bone Structure

12a. Lumbar spine



12b. Pelvis

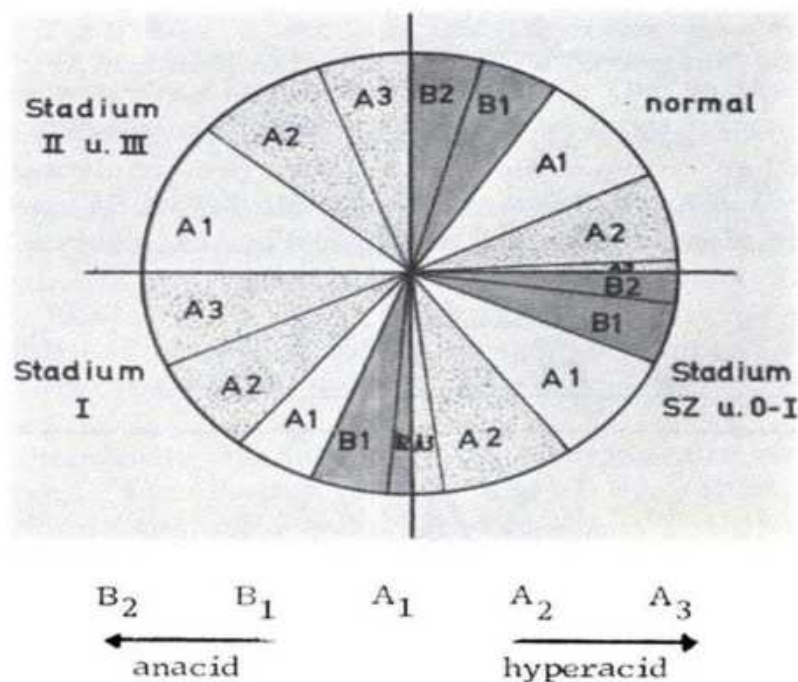


12c. Lower legs and forearms only slight periosteal appositions

viously (32, 53-57) individual susceptibilities, particularly nutritional habits, the capacity of absorption of the gastrointestinal tract and the renal threshold for fluorides play an important role in the development and severity of fluorosis. We therefore studied the relationship of gastric acid in 150 aluminum workers to the degree of severity of fluorosis. The gastric acid was determined by the Acido-test. Figure 13 shows a distinct correlation namely, with increasing severity of fluorosis the percentage of hyperacid persons ($A_2 + A_3$) increases and the proportion of hypo- or an- acid persons ($B_1 + B_2$) decreases. The differences were statistically significant. These findings prove that fluoride resorption is obviously diminished by a deficiency of gastric acid and that it is enhanced by hyperacidity.

Fig. 13

Correlation Between Gastric Acidity and Development of Fluorosis



2. Physical examinations: In the iliac crest biopsies and in the necropsies we noticed repeatedly an extreme hardness of the fluorotic bone. The studies of the physical properties of the fluorotic bone show the following (33, 58): a) An increase in the microhardness at the cut surface of a piece of the femur is more pronounced in fluorosis I-II than in fluorosis III; b) A threefold higher compressive strength per area of a fluorosis III vertebra; c) An increase of the module of elasticity of the femur slices, 2 cm long, and of the defined cylinders of the corticalis of the femur in moderate fluorosis. In severe fluorosis a decrease was observed; d) An increase of the breaking strength area of slices of femur in both cases of fluorosis. In flu-

orosis III we found the breaking strength per area was impaired at the exactly defined cylinders of corticalis of the femur whereas there was a slight increase in strength in fluorosis I-II.

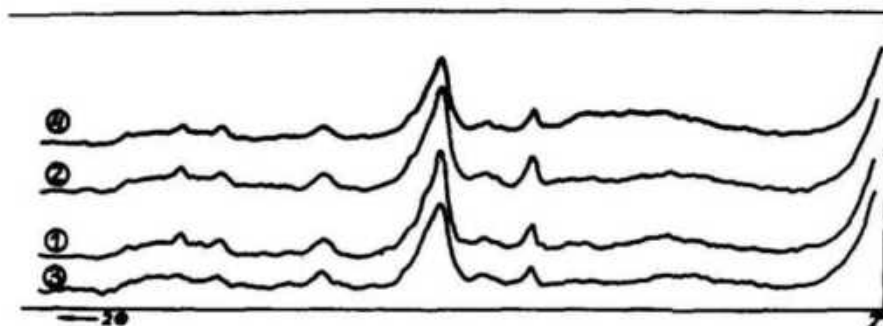
A moderate degree of fluorosis causes a real increase in bone strength, but severe fluorosis shows partly a static inferiority (cortex cylinder). This inferiority, explicable by the serious histological changes, might perhaps be compensated, at least partially, by the enormous quantity of bone.

3. Crystallographic studies: Material and Method - Fluorotic bones from a patient who had changed his job as an aluminum worker one year previously, fluorotic bones of two patients who had changed their job several years previously as well as bones of rats fed 20 mg NaF/day/kg for 12 months were studied. Diffractometric examinations were carried out on a diffractometer of the type Miller-Mikro 111 (radiation $\text{CuK}\alpha$, 26 kV, 36 mA with nickel filter, goniometer speed: $1/2^\circ/\text{min}$. The intensities were integrated with a Dupa polar planimeter).

Results - The hydroxyapatite crystals were changed in the bone of the patient who had changed his job one year previously and also in the bones of rats fed with NaF inasmuch as their "a" -axis was shorter, i. e. they were more slender than the average (Fig. 14, curves 1 and 3).

Fig. 14

Curves of X-Ray-Diffraction Analysis



- curve 1 - advanced fluorosis after one year without fluorine contact
- curve 2 - moderate fluorosis, 4 years without fluorine contact
- curve 3 - bones of rats fed 20 mg NaF/day/kg for 12 months
- curve 4 - slight fluorosis, 3 years without exposure to fluoride

The bones of the patients who had changed their job several years previously showed no crystal changes in spite of the sclerosis which is to be seen on the x-ray picture and of the higher than average fluoride level

(curves 2 and 4). Our findings are similar to those of Baud and Bang (59, 60) and demonstrate that it is possible for the normal crystal forms to be restored after the patient has not been exposed to fluorides for several years.

Therapy and Prophylaxis

In the G. D. R., since 1950, industrial fluorosis has been recognized as an occupational disease which is reportable and for which compensation is required (Law Code of the G. D. R. 1950, 50, p. 389). By further decrees and laws of the G. D. R. government, industrial medical care of all workers has been markedly improved. Prophylactically, it is important to reduce the fluoride concentration of dust at the place of work, especially by improved ventilation of enclosed factory areas.

At the examination for employment, persons with the following diseases are considered totally unsuitable: liver and kidney changes, blood and thyroid gland diseases, post-traumatic or congenital skeletal damage, infectious and para-infectious diseases of the apparatus of locomotion (rheumatism, Bechterev's disease); also, workers with distinct degenerative changes of the spine and of the large joints are unsuitable.

Following the initial examination, the workers are examined once a year; at this examination, special attention is paid to the following points: pains in the spine and great joints, nausea, lack of appetite, constipation, headache, dyspnea, restriction of the movability of the spine (especially of rotation), of the respiratory chest expansion and of the vital capacity.

Biochemically, the following examinations are carried out: calcium, phosphorus and alkaline phosphatase of the serum, 24-hour-excretion of fluoride in the urine and a blood assay.

Every third year, at least, the following parts of the skeleton are x-rayed: thorax, pelvis, lumbar spine in two directions, one lower leg and one forearm in the anterior-posterior direction with the short exposure technique.

In doubtful cases, such as patients with distinct complaints and clinical findings but with slight roentgenological changes, we carry out an iliac crest needle biopsy. If there are distinct histological changes and if the fluoride values in the iliac crest ash are above 0.35% to 0.40%, such cases must also be recognized as an occupational disease in spite of the minimal degree of roentgenological changes.

Therapeutically, it is necessary to remove the worker from the fluoride-endangered place of employment as soon as fluorosis has been diagnosed. Physiotherapeutical measures like massage, short-

wave and ultrasonic therapy, balneotherapy, and a medicamentous treatment with indometacin or phenylbutazon brings about an improvement of the condition but the patients will not become symptom-free.

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Discussion

Dr. Sinclair: I note that you find very little osteoporosis and no evidence of secondary hyperparathyroidism, such as Dr. Teotia reported here. Could this be because your patients have a higher calcium intake than hers and thus do not require mobilization of calcium from their bones? Also, I am not clear why you see no anemia. You showed great diminution of the bone marrow due to osteosclerosis, but you observe no anemia.

Dr. Franke: My cases agree with those of Professor Jolly; he too encountered no osteoporosis. All exposed workers in my studies received a well-balanced diet and a normal intake of calcium

(ca. 1 g/day). As to your second question, we have studied 400 aluminum workers afflicted with different stages of osteofluorosis and found no anemia. The cases I presented here showed a normal blood picture.

Dr. M. Teotia: To add to what Dr. Sinclair has said, in our cases osteoporosis could be correlated with lower calcium intake. In addition, because our patients live in the endemic areas from early childhood, their bones contain a larger amount of the less-soluble, more stable fluoroapatite from which ionized calcium is less available than from hydroxyapatite. This leads to a greater stimulation of the parathyroid glands and, therefore, we observe more evidence of secondary hyperparathyroidism. We have measured parathyroid hormone levels in the serum and have demonstrated hypertrophy of the parathyroid glands in a 63-year-old patient who developed tertiary hyperparathyroidism. This was reported in the British Medical Journal (17:637-40, 1973). We find that osteoporosis and secondary hyperparathyroidism are definitely related to deficiency in calcium intake and to the duration of exposure to fluoride.

Dr. Franke: With respect to the measurement of bone strength in industrial skeletal fluorosis, we repeatedly noticed extreme hardness of fluorosed bones in iliac crest biopsies and necropsies. Our studies revealed

- a. an increase in the microhardness at the cut-surface of a piece of femur which is more pronounced in fluorosis stages I and II than in stage III,
- b. a three-fold higher compression strength per unit area in stage III vertebrae,
- c. an increase in the module of elasticity in sections of the femur 2 cm deep and 5 mm thick in moderate fluorosis (stages I and II) but a decrease in elasticity in severe fluorosis (stage III),
- d. an increase in breaking strength per unit area in segments of femur in stages I and II but not in stage III. Moderate osteofluorosis gives rise to a real increase in bone strength but severe fluorosis causes a decrease for which, however, the enormous increase in the quantity of bone partially compensates.

Dr. Hanhijärvi: Did you measure urinary fluoride concentrations?

Dr. Franke: Yes, but we could not relate urinary fluoride levels with the degree of osteofluorosis. We often found severe fluorosis

associated with low urinary fluoride levels and incipient fluorosis with very high levels.

Dr. Hanhijarvi: You stated that bone strength declines in stage III fluorosis. Was this bone substance from the surface or from the interior?

Dr. Franke: It was cortical bone.

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INFLUENCE OF PROPAGATION MEDIA AND AMENDMENTS ON
FLUORIDE TOXICITY OF CORDYLINE TERMINALIS
'BABY DOLL'

by

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SUMMARY: Cuttings of *Cordyline terminalis* 'Baby Doll' a popular foliage plant used extensively in dish gardens because of its attractive red coloration often exhibits tip necrosis in the propagation bed. This necrosis is caused by fluoride found in the soil-water solution. Proper selection of propagating medium and elevation of pH can alleviate the fluoride-induced necrosis.

Demand for plant materials in ornamental plant combinations (groups of different indoor foliage plants in a single container) has increased considerably in recent years. Foliage plants with good keeping quality and bright colors are highly prized in combinations with other plants because they provide a visual focal point. One foliage plant with excellent characteristics for combinations is *Cordyline terminalis* Kunth 'Baby Doll'. This plant grows upright, roots readily and possesses foliage of a maroon color with light rose margins.

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