We are currently studying a group of patients with metastatic bone pain whose thyroid glands have not been previously irradiated to see if some evidence can be gained to support the last-mentioned hypothesis. Prior to receiving doses of NaF for treatment of their bone pain and two weeks after initiation of treatment numerous thyroid tests are being performed including: 1. Total Murphy Pattee by competitive protein-binding analysis, 2. "Free" T-4 either by the dialysis method (Bio-Science) or by a modification of the effective thyroxin ratio (Mallinckrodt), 3. Total T-3 by radioimmunoassay, 4. T-3 resin uptake, 5. Two hour radioactive iodine uptake will be performed, the patient will then be given 500 mg of perchlorate (which is normally used in brain scanning); for the next hour serial radioiodine uptakes will be determined at 10 minute intervals; for the next 1/2 hour, at 15 minute intervals.

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THE SPINAL CORD

by

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SUMMARY: The author presents evidence of damage to the spinal cord in skeletal fluorosis. Since pressure on the cord by osteophytes was ruled out direct action of fluoride on cord tissue is likely to be responsible for this condition.

Neurological complications of skeletal fluorosis have been described in endemic areas, especially in India. They consist of radiculomyelopathy which may lead to spastic tetraplegia. We wish to present evidence of neurological complications in a 60 year-old male who had been working for 10 years in an aluminum smelter. Following a fall on his back in November 1970, the patient complained of increasing weakness in the right leg, pains in the lumber spine and vertigo.

The author observed on examination, limited mobility of the vertebral column, bilateral protrusion of bulbi, ataxic gait, absence of reflexes on arms and legs and tetraparesis which was most noticeable on the legs, especially on the right side. Also, the patient manifested loss of mental acuity, mental depression and a limited ability of orientation. X-ray examination revealed evidence of fluorosis, degree one to two according to Roholm's classification. The patient expired on May 30th, 1971.

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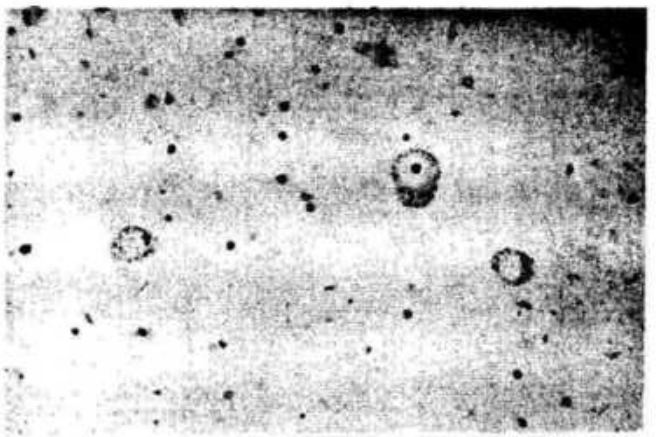
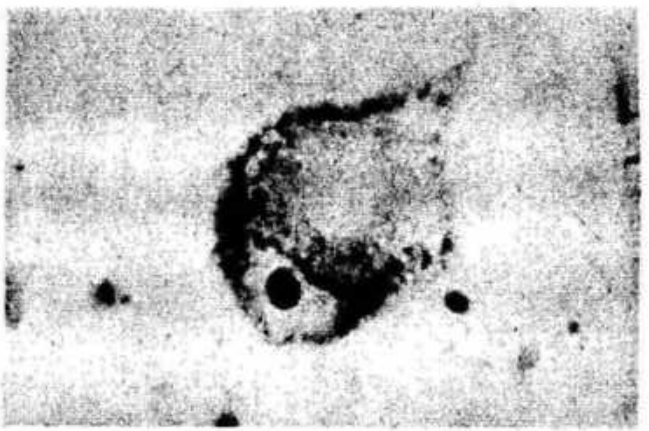
Fig. 1
Microscopic Specimen of Tissue of Anterior Horn



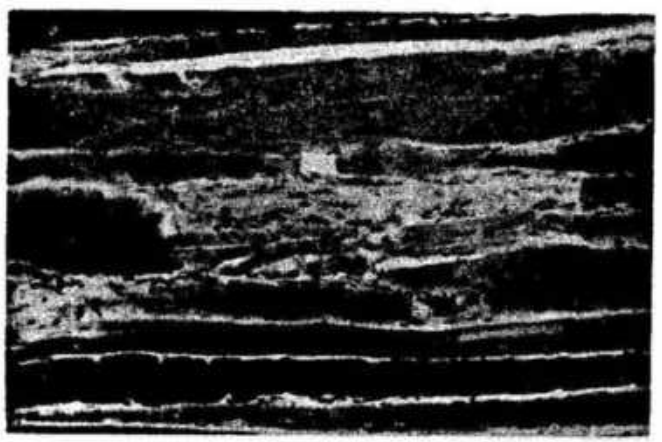
Increased translucency of cytoplasm; reduction of tissue bodies.

Fig. 2
Typical "Fish cell"



Vacuolated nucleus.

Fig. 3
Skeletal Musculature in Fluorosis



Focal necrosis of muscle fibers with proliferation of fibroblasts and sarcoblasts.

At autopsy, a 4 x 4 cm large glioblastoma was found in the left parietal portion of the cerebrum; in the spinal cord, a distinct decrease of the number of ganglion cells of the anterior horn (fig.1) and pathobiosis of the ganglion cells with contracted cells, increased translucency of the cytoplasm, reduction and partial clotting of the Nissl bodies which are located near the cell membrane. Furthermore, we noted cells whose nucleus was lying at their periphery with a vacuolated nucleus (so-called "fish cells") (fig. 2). Moreover, we observed a reduction of number of ganglion cells on the medical posterior funiculus and, to a lesser degree, at the lateral horns. The skeletal musculature revealed focal necrosis of muscle fibers with fibrosis and proliferation of fibroblasts and sarcoblasts (fig. 3). Additionally, plaque-like calcifications in the musculature were seen near the periosteal attachments. The bones showed periosteal apposition of bone substance with widened and spongiosed compacta. Additional findings included a scar of an old cardiac infarct at the left ventricle and evidence of a generalized, moderately severe arteriosclerosis. No fluoride-related damage was found in the parenchymatous organs, particularly on liver and kidneys. The fluoride content of the iliac crest was 0.74% (7.400 ppm) ashed, compared to normal values which range from 0.05 to 0.1%.

The pareses of the extremities were attributable to the lesions of the cells of the motor anterior horns (myelopathy) and the changes in the cells of the muscles (myopathies). Whereas the simultaneous occurrences of the cerebral tumor and fluorosis must be considered coincidental, the structural changes of the anterior motor cells and the skeletal musculature are etiologically related to the existing fluorosis. Significantly, we found no evidence of a narrowing of the spinal canals nor of the foramina intervertebralia to which damage to the cells of the pyramidal column is usually attributed in endemic skeletal fluorosis. Because other causes were excluded we believe that these spinal cord lesions and the associated muscular damage are due to direct action of the fluoride ion on the ganglion and muscle cells.