Appendix 11

The corrosive effect of fluoride

As the water fluoridation trials began, concern mounted, and Arthur Rabinowich published a review in the Canadian Medical Association Journal April 1945, about the corrosive effect of fluorine. He wrote the following:

" ….in view of the increasing use of fluorine and fluorine containing compounds in industry….aside from death due to the corrosive action of the poison, death may result very rapidly from reduction of the calcium content of the blood".
Rabinowich added;
"In the 4th edition of Sollmann's Pharmacology it is stated that "the acute effects may be due partly to dis ionization of calcium, similar to oxalates and it is suggested that "intravenous injection of calcium chloride might be useful". In Webster's Legal Medicine and Toxicology there is the statement that fluorine "combines with the calcium of the tissues and becomes, therefore, a protoplasmic poison". In Scholes 's Clinical Toxicology it is stated that "sodium-fluoride produces disorders of the calcium metabolism similar to those which occur after removal of the para-thyroid glands". In the recent (8th) edition of Sydney Smith's Forensic Medicine it is pointed out that, in addition to their corrosive action, fluorides are " generally protoplasmic poisons closely allied to the oxalates " and, under 'oxalates" (p. 448) it is stated that there is every reason to believe that the action of oxalic acid is "partially or wholly due to the combination of the acid with the calcium salts of the blood and tissues, thus rendering the essential calcium ions inoperative". The resemblance to oxalates is also mentioned by Underhill and Koppanyi. The fact that acute fluoride poisoning may have a serious effect upon the calcium content of the blood is dealt with at some length in McNally's Toxicology."

Rabonwich continues; "OCCURRENCE
Fluorspar (CaF,), a naturally occurring fluorine compound, has been known since 1529. It was first used for etching of glass in 1670/17 and has been used extensively for this purpose since then. Clouding of electrical bulbs is an example. It is used extensively as a flux in smelting metals, during which volatile silicon tetrafluoride (SiF4) is a by-product. Enormous quantities of cryolite (Na,AlF,) are used for production of aluminium, and, during this process, highly volatile hydrofluoric acid (HF) is an unavoidable by-product. Phosphate rock, which contains about 3.5% fluorine, is used for manufacture of superphosphate used in animal and plant nutrition. Fluorides are required for manufacture of organic fluorides, and they are constituents of insecticide sprays and powders and of rat poison. Rubber, textile, ceramic, hide and skin, enamel, cement, glue, brick, wood, refrigeration and magnesium casting are examples of other industries in which fluorides are used."

"TOXICOLOGY
Types of poisoning. These may be considered under three headings, namely, (a) fluorine gas; (b) hydrofluoric acid, and (c) soluble salts of fluorine. Their importance as poisons is in the reverse order. Acute fluorine gas poisoning is so rare that it needs only mere mention; whereas, most of the deaths, accidental and suicidal, have been due to sodium fluoride and other soluble fluoride salts.
Fluorine. – This is one of the most active elements, combining very readily with metals in nature. It reacts very readily with the moisture in the air to form hydrofluoric acid. Industrially, therefore, the hazard, as in production of aluminium from cryolite, is not from the element but from the acid.
Fluorine is a light, greenish-yellow gas and produces intense irritation of the conjunctive and mucous membranes of the air passages, causing violent spasms of coughing, with pain in the chest, which may persist for some time after the victim has been removed to fresh air. The most serious effects are upon the lungs. The exact mechanism is not clear. Experiences with chlorine gas in the Great War and since then have shown that the most serious results, such as pulmonary oedema, are not explained entirely by production of a corrosive acid, and there is reason to believe that this also applies to fluorine.
Treatment. – Fresh air, warmth, rest, hot sweet drinks and oxygen immediately for any degree of cyanosis. Experiences with lung irritant gases in the last war showed that no person in whom it was possible to restore a pink colour by proper use of oxygen died from simple pulmonary oedema.18 Even mild cases should be kept under observation for the first 24 hours for the possibility of delayed onset of pulmonary oedema.19

Hydrofluoric acid. -This is a colourless liquid which fumes in air. It is a violent poison, both in itself and also because the commercial product may contain sulphuric acid. It irritates all tissues with which it comes into contact. The solutions used for etching of glass contain anywilere from 15 to 40% of hydrofluoric acid.

(a) Inhalation. – Inhalation of hydrofluoric acid fumes in very high concentrations may produce vomiting and rapid collapse. In lesser concentrations, they produce inflammation and ulceration of the conjunctivae and of the mucous membranes of the nose, mouth, larynx and bronchi. As little as 10 parts of the acid per 1 000,000 parts of air has produced such ulcerative lesions and broneho-pneumonia.20 For treatment, see fluorine.

In lower concentrations for short periods, except for temporary cough and lachrymation, which soon passes off in fresh air, the fumes are apparently harmless. The maximum allowable concentration for prolonged exposure is about 2.5 gmm. per cubic metre or, approximately, three parts of the gas per 1,000,000 parts of air.21,22 Prolonged and repeated exposure to mildly irritant concentrations seems to decrease the sensitivity to the fumes. A personal experience may be cited here as an example. While visiting an aluminium production plant, the writer found the fumes very uncomfortable; they produced marked lachrymation, a sensation of suffocation and cough which continued for about one-half hour after having left the contaminated atmosphere; whereas, no discomfort was noticed amongst the many workers, none coughed, and none showed any lachrymation.

(b) Ingestion. -Hydrofluoric acid, when taken by mouth, is a violent corrosive poison. The chief features are marked dysphagia, vomiting and collapse, and death may occur within a few minutes. The buccal mucosa may be bleached and there may be denudation of the epithelium of the tongue, pharynx and oesophagus. Even in contact with skin such solutions produce blisters and gangrene. The burns are painful and heal slowly.

The minimum lethal dose by mouth is not known. One tablespoonful of a 9% solution has caused death.6 When death does not occur within an hour, in addition to the signs and symptoms due to the corrosive action locally, there may be also those produced by reduction of the calcium content of the blood and by direct action of the poison on the heart muscle.

FLUORIDE SALTS

Most deaths, accidental and suicidal, have been due to these compounds. Sodium fluoride heads the list, and the description of it here applies, in general, to the other soluble salts.

Fluorine is widely distributed in nature. It is, therefore, a constituent of normal body tissues, particularly teeth and bone; but, like lead, it is present in minute traces and probably as a contamination rather than for physiological needs. Sodium fluoride is a corrosive and a general protoplasmic poison. It is poisonous to plants and bacteria and inhibits enzyme action (urease, lipase, etc.). It combines with calcium in an ionic state to form insoluble calcium fluoride. Calcium, as is well known, is indispensable for the functional integrity of the voluntary and autonomic nervous systems; but only calcium in ionic form is physiologically active, and it is precisely such calcium which is attacked by fluorides. Sodium fluoride thus lowers the available calcium of the blood and thus produces low-calcium tetany. For the same reason, sodium fluoride is an anticoagulant, but also probably bv an effect upon thrombin formation and also injury to the liver. Normal coagulation, for example, is not restored by replacing the calcium lost by precipitation but only if thrombin is also added.11

Sodium fluoride is not very soluble (a saturated solution contains, approximately, 4 grams per 100 c.c.) but, when in solution, it is rapidly absorbed from the stomach. Destruction of the mucous membrane lining of the stomach increases the absorption. Unlike the other halogens (chlorine, bromine and iodine), however, it is slowly excreted and thus tends to accumulate in the body tissues as insoluble calcium fluoride, which tends to deposit in the liver, kidneys and other tissues, as readily recognizable crystals. When deposited in bone, the calcium fluoride makes the latter white, harder and more brittle. Unlike oxalic acid, which also combines with, and thus lowers, the available calcium in the blood, sodium fluoride also exerts an alkaloid-like reaction. In lower organisms, for example, which do not require calcium, the fluorides still exert this action; whereas, oxalic acid does not.

Fatal dose. -The minimum lethal dose is not known. About 4 gm. have caused death in an adult. In general, the more soluble the fluoride the greater is its toxicity; but fluorine content is an equally important factor. Sodium fluosilicate (Na_2SiF_6) for example, used widely as a rat poison, is much less soluble than sodium fluoride, but, when adjusted to fluorine content, the toxicity is approximately the same. As little as 0.2 to 0.7 gm. of sodium fluosilicate have caused death in adults.23

Fatal period. -Though the salts tend to act more slowly than hydrofluoric acid, death has resulted within 5, 10 and 15 minutes, 24 25 but periods of 10 to 12 hours have been noted. In the case cited above, death occurred in three hours. The average is about eight hours.

(Source: - Review by Arthur Rabinowichin, the Canadian Medical Association Journal April 1945, concerning the corrosive effect of fluorine. Vol/page 52(4);345-90)