The Conclusion of a Ten-Year Study of Water Fluoridation


In areas where the potable water supplies contain the fluoride ion at optimum concentration at the source, the dental caries experience of children who ingest these water fluorides during the years of tooth development is about 60 per cent less than among children in areas with fluoride-deficient water supplies. Adults who have used such water supplies continuously enjoy the dental benefits obtained during childhood.

Controlled water fluoridation for the prevention of dental caries, i.e., the addition of fluoride compounds in optimum concentration to fluoride-deficient supplies, has been studied since 1945 in three different areas. These studies have demonstrated that dental caries can be effectively reduced through controlled water fluoridation to the same extent as observed in areas where water contains the fluoride at the source. A recent review presented the DMF ( decayed, missing, or filled teeth ) rates for six- to 10-year-old children after nine years of fluoride experience in Grand Rapids, Mich., Newburgh, N. Y., and Brantford, Ontario, and compared these data with those in Aurora, Ill., which uses a water supply with naturally occurring fluoride at 1.2 ppm F. At ages six to nine the rates in all four communities were found to be quite comparable and at age 10 the rates for the three communities fluoridating their water supplies approached the expectancy level noted in Aurora.

One of the most comprehensive of the studies, the Newburgh-Kingston Caries-Fluorine Study has recently issued its final report based on 10 years of fluoridation experience. The report, consisting of three definitive papers

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dealing with the history of the study and its pediatric and dental aspects, and a fourth paper dealing with fluoride metabolism, was presented before the New York Institute of Clinical Oral Pathology on December 12, 1955. These papers appear in the March, 1956, issue of the Journal of the American Dental Association.⁴⁻⁷

Prior to the initiation of controlled water fluoridation programs in 1945 extensive epidemiological investigations⁸ᵃ,ᵇ had demonstrated (1) the occurrence of a defect of tooth enamel which discolored and, in extreme cases, caused pitting of the enamel; (2) the discovery that the stain or mottled enamel was caused by the ingestion of water-borne fluorides during the years of enamel calcification; (3) the direct relationship of the degree of mottling to the fluoride content of the water; (4) an inverse relationship of dental caries to fluorosed or mottled teeth; and (5) that where the water supply contained approximately 1.0 ppm F, the residents enjoyed considerable protection against dental caries without the hazard of disfiguring mottled enamel.

Cox and his co-workers⁹ in 1939 suggested that the addition of fluorides to food and water to bring the fluoride content up to the optimum level could prevent dental caries if ingested during the years of tooth development. Ast¹⁰ in 1942 outlined a plan to test the caries-fluorine hypothesis. He suggested a study of two comparable communities with fluoride-deficient water supplies, one of which should have its water supply supplemented with sodium fluoride to bring its fluoride content up to 1.0 ppm and the second to serve as a control.

This plan was considered by the New York State Department of Health. In 1944 a Technical Advisory Committee on the Fluoridation of Water Supplies was appointed to study the proposal. The committee was also asked to recommend the types of medical and dental examinations which should be made to determine the efficacy and safety of water fluoridation. After a careful review of the literature and the objectives of the study, the committee recommended that a long-range study be undertaken. The cities of Newburgh and Kingston, each with a population of approximately 30,000, situated about 35 miles apart on the west bank of the Hudson River and using fluoride-deficient water supplies, were asked to participate in a 10-year study. Newburgh agreed to serve as the study area and to have its water supply supplemented with sodium fluoride to bring its fluoride content up to 1.0–1.2 ppm. Kingston agreed to serve as the control and continue to use its water supply with approximately 0.1 ppm F.

In June, 1944, base line pediatric and dental examinations were begun and on May 2, 1945, Newburgh's water supply was fluoridated. This process has been in continuous operation since that date. The base line data showed that the children aged six to 12 in both cities had a similar dental caries experience. The Kingston rate was 20.2 DMF teeth per 100 permanent teeth and the Newburgh rate was 20.6. Periodic progress reports have demonstrated a downward trend in the dental caries experience among the children in Newburgh. In Kingston the caries rates have remained relatively unchanged.

In June, 1955, clinical and intraoral dental roentgenographic examinations were completed after 10 years of fluoride experience. In Newburgh 1,519 children aged six to 14 and 109 aged 16 who had had continuous residence there throughout the period of fluoridation were examined. The Kingston children examined included 2,021 aged six to 14 and 119 aged 16. The clinical examinations were made in both cities by the staff senior dentist and the roentgenograms were taken by the staff
senior dentist and dental hygienist. The films were developed and sent to the Dental Bureau office in Albany. There statisticians randomized the film series so that the interpreters did not know whether they were reading Newburgh or Kingston films.

The children aged six through nine years in Newburgh had used fluoridated water throughout their lives. The 10- to 12-year-old children, who were under two years of age in 1945, had used fluoridated water during the partial calcification of the crowns of the first permanent molars and throughout the calcification of the second permanent molar crowns. The 13- to 14-year-old children were three to four years old in 1945. These children started drinking fluoridated water after the calcification of the crowns of the first molar teeth but prior to the eruption of these teeth, and throughout the period of calcification of the crowns of the second molars. The 16-year-old children were six years of age when fluoridation was started. At that time their first permanent molars were beginning to erupt into the mouth and the crowns of their second molars were almost fully calcified.

The DMF rate for the six- to nine-year-old children in Newburgh was 58 per cent lower than that for the Kingston children. The 10- to 12-year-old children in Newburgh had a DMF rate 53 per cent lower. At ages 13 to 14 the DMF rate was 48 per cent lower, and at age 16 it was 41 per cent lower, than the rates in Kingston (Table 1).

The first permanent molar is frequently referred to as the keystone of the dental arch and warrants special consideration because of its strategic position in the mouth. This tooth, because of its morphology and the early age at which it erupts into the mouth, frequently succumbs to dental caries early in life. It is therefore significant to note that among the six- to nine-year-old children in Newburgh the DMF rate for first permanent molars was 53 per cent lower than that for the Kingston children in the same age group. The DMF rate in Newburgh at ages 10 to 12 was 30 per cent lower, at ages 13 to 14 it was 14 per cent lower, and at age 16 it was 4 per cent lower, than in Kingston (Figure 1).

Of even greater significance is the observation that the children in Newburgh at age six to nine had 68 per cent fewer untreated carious first molars and 88 per cent fewer first molars lost than did
the Kingston children of the same ages. The 10- to 12-year-old children in Newburgh had a rate 45 per cent lower for untreated caries and 78 per cent lower for missing first molars. At ages 13 to 14 the differences were 26 per cent for untreated caries and 42 per cent for missing first molars, and at age 16 the differences were 41 per cent for untreated caries and 32 per cent for missing first molars (Table 2).

Another significant observation was that ingested water fluorides afford selective protection to the proximal (adjacent) surfaces of the teeth in comparison with the occlusal (biting) surfaces. This is highly important because the proximal surfaces present difficulties in both caries detection and correction. Frequently caries on the proximal surface of a tooth requires the cutting of much sound tooth structure in order to place an adequate filling in the tooth. At each of the age levels studied the per cent of differentiable carious proximal surfaces among the Kingston children was about three times greater than that noted in the Newburgh children.

At ages six through nine all of the deciduous cuspids and deciduous molars are normally present in the mouth. If any of these teeth are missing it may reasonably be presumed that they were lost because of caries. Among the six- to nine-year-old children in Newburgh 25.5 per cent had all these teeth present and caries free, as compared with 4.7 per cent of the Kingston children (Table 3).

Dean's 11 epidemiological studies of endemic dental fluorosis demonstrated that there was no disfiguring dental fluorosis at the level of about 1.0 ppm F. Unfortunately, the term mottled enamel or dental fluorosis is applied to all degrees of this condition. In its more severe forms it does produce discoloring stains and possibly pitting of the enamel. However, in the milder forms of fluorosis the enamel of the tooth has a high luster which enhances

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**Table 2—Status of Erupted First Permanent Molars in Children Ages 6–16, Based on Clinical and Roentgenographic Examinations, Newburgh * and Kingston, N. Y., 1954–1955**

<table>
<thead>
<tr>
<th>Age</th>
<th>Caries-Free</th>
<th>DMF **</th>
<th>Filled</th>
<th>Untreated Caries</th>
<th>Missing</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Newburgh</td>
<td>Kingston</td>
<td>Newburgh</td>
<td>Kingston</td>
<td>Newburgh</td>
</tr>
<tr>
<td>6–9</td>
<td>74.9</td>
<td>46.7</td>
<td>25.1</td>
<td>53.3</td>
<td>14.2</td>
</tr>
<tr>
<td>10–12</td>
<td>36.8</td>
<td>10.9</td>
<td>63.2</td>
<td>90.0</td>
<td>40.2</td>
</tr>
<tr>
<td>13–14</td>
<td>19.3</td>
<td>5.9</td>
<td>80.7</td>
<td>94.1</td>
<td>43.9</td>
</tr>
<tr>
<td>16</td>
<td>8.5</td>
<td>4.8</td>
<td>91.5</td>
<td>95.2</td>
<td>55.0</td>
</tr>
</tbody>
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* Sodium fluoride added to Newburgh's water supply beginning May 2, 1945.
† Adjusted to the first permanent molar population in the Kingston 1954–1955 examination.
‡ Age at last birthday at time of examination.
** DMF includes permanent teeth decayed, missing (lost subsequent to eruption), or filled.
§ Newburgh children of this age group exposed to fluoridated water from time of birth.
the beauty of the tooth rather than disfigures it. The detection of the early signs of dental fluorosis requires an examiner who has had extensive experience in areas of endemic fluorosis. The average dental practitioner would in all probability not detect the earliest signs of mottling.

In order to determine whether the children in Newburgh showed any signs of dental fluorosis a specially trained officer of the Public Health Service with long experience in the detection of the mildest of such lesions was requested to make the examinations. He examined 621 children aged seven to 14 in Newburgh, of whom 438 had resided there continuously since the start of fluoridation. In Kingston 612 children of the same ages were examined. In addition to dental fluorosis, examinations were made for enamel opacities due to causes other than ingested fluorides. These other enamel opacities are generally developmental hypoplasias. They usually appear as circular white or colored patches and most of them are obvious even to the untrained eye.

Among the 438 children with continuous residence in Newburgh, 46 had questionable fluorosis, 26 had very mild, and six had mild fluorosis. There were no cases of moderate or severe mottling and in no instance was there any disfiguring discoloration. Thirty-six of the Newburgh children examined had nonfluoride opacities. Of the 612 children examined in Kingston, 115 had nonfluoride opacities. The relatively infrequent occurrence of nonfluoride enamel opacities in Newburgh compared with Kingston tends to confirm a previous report that ingested water fluorides at the recommended concentration appear to reduce the occurrence of hypoplastic spots on the teeth.

The same groups of children examined for enamel opacities were also examined for evidence of gingivitis. A positive score was recorded only for flagrant gingivitis, thus making it possible to place greater emphasis on advanced disease and minimize examiner bias. There was slightly, but significantly, more gingivitis observed among the Kingston children than among those in Newburgh.

The final report on the pediatric findings of the Newburgh-Kingston study pointed out that all the scientific evi-
dence available at the time the study was first proposed indicated the safety of drinking water containing about 1.0 ppm F at the source. There was no reason at that time to believe that fluorides, when added to the drinking water as part of the water treatment process, would act in any way differently from fluorides already present. Nevertheless, it was considered desirable to test this remote possibility under the carefully controlled conditions established for the long-term Newburgh-Kingston study.

Closely similar groups of children were studied in Newburgh and Kingston. In the final year of the study 500 of the children enrolled in Newburgh and 405 in Kingston were examined in the study clinic. The points of concentration in the examination were those related to possible systemic effects of fluoride ingestion as manifested by changes in growth and development or in abnormalities on the physical, laboratory, and roentgenographic examinations. Each child was given a general medical examination by a qualified pediatrician. Height and weight were measured. Roentgenograms were taken of the right hand, both knees, and the lumbar spine. Bone density and bone age (maturation of the skeleton) were estimated by independent observers who were not aware of the city of origin of the individual roentgenograms. Laboratory examinations, including hemoglobin level, total leucocyte count, and routine urinalysis were also made. No differences of medical significance could be found between the groups of children in the two cities. This indicated the absence of any findings suggestive of systemic effects from the drinking of fluoridated water during the period of most rapid growth. In addition, special detailed studies of the eyes and ears were performed on a smaller group of children; these included determination of visual acuity, visual fields, and hearing levels. The results of these special examinations were well within the range of expected prevalence of the conditions studied.

Reference was made to another recently published paper which presented further evidence for the absence of systemic effects from fluoridated water. The purpose of this study was to determine whether any irritative effects on the kidneys follow prolonged use of fluoridated water. The quantitative excretion of albumin, red blood cells, and casts in 12-hour urine specimens in 12-year-old boys, using a modified Addis technic, was determined in the two cities. The differences in the results between the groups in the two cities tended to favor the Newburgh children, but no medical significance could be attributed to any of the differences.

The review of current knowledge of the metabolism of fluorides, particularly in the human body, applied this information in estimating the factors of safety in water fluoridation. Knowledge of blood fluoride levels, of the rate and mechanism of urinary excretion of fluoride, and of the magnitude and mechanism of bone deposition increases our understanding of some important biological effects of toxic doses of fluorides, such as acute fluoride poisoning, crippling fluorosis, osteosclerosis, and mottled enamel.

The blood fluoride level in experimental animals given lethal doses of fluoride rises to a peak in a half hour to an hour, falls rapidly within two to three hours, and returns to its normal level within 24 hours. The blood does not tend to accumulate fluoride, although the blood fluoride level in persons drinking fluoridated water is somewhat higher than in persons drinking fluoride-deficient water.

When human beings ingest small amounts of fluoride a significant fraction is promptly excreted in the urine. It is probable that when human beings
Ingest small amounts of fluoride equivalent to that of fluoridated water over a period of years, the daily urinary excretion is greater than half of the amount absorbed each day. The extraordinarily rapid and efficient urinary excretion of fluoride is attributable to a somewhat lower resorption of fluoride in the kidney tubules than is characteristic of chloride.

The other mechanism for removal of fluoride from the blood is by deposition in the bones, the amount of fluoride present in the hard tissues probably being directly dependent on the amount of fluoride taken into the body day after day. The mechanism of fluoride deposition is simple, the fluoride ion replacing the hydroxyl groups of the surface of the bone crystals. There is no indication that any notable biological disadvantage results from this. Fluoride deposition in bone is a reversible process.

With regard to acute fluoride poisoning there is at least a 2,500-fold factor of safety in water fluoridation. The mechanics of water fluoridation are such that it is impossible to produce acute fluoride poisoning either by accident or intent.

Crippling fluorosis, characterized by a stiffening in the back due to calcification of the broad ligaments of the back, occurs with a daily intake of 20 to 80 milligrams of fluoride or more for 10 to 20 years. Since five gallons of fluoridated water at 1 ppm F contain 20 milligrams, it is obvious that crippling fluorosis can never be produced by drinking fluoridated water. The earliest evidence of osteosclerosis, a hypercalcification detectable by roentgen examination, does not occur with an intake of fluoride below eight to 10 times the level of fluoridated water.

The evidence with respect to heart disease, kidney disease, cancer, and possible influence of fluoride on the thyroid is also reviewed. Ample statistics are available to indicate no influence of fluoride intake on any of these at the levels found in any water supplies in the United States. Studies on experimental animals with the use of radioactive fluoride show that the thyroid gland does not concentrate fluoride as it does iodide. The presence of renal impairment in experimental animals and in human beings with long-standing kidney disease appears not to affect excretion of fluoride by the kidneys.

The comprehensive analysis of the Newburgh-Kingston Caries-Fluorine Study after 10 years of experience, added to the wealth of evidence previously reported, demonstrates conclusively two important facts—fluoridation is effective in reducing dental caries and it is a safe public health practice.

REFERENCES