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The effect of rinsing with water immediately after a professional fluoride gel application on fluoride uptake in demineralized enamel: an in vivo study

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Abstract

This study was designed to determine if allowing patients to rinse, eat, or drink immediately following topical application of an acidulated phosphate fluoride (APF) gel (1.23% F) had a significant effect on fluoride uptake into artificially induced incipient lesions. Enamel specimens were placed into specially prepared partial dentures of 14 subjects. All were given a professionally applied topical fluoride treatment with an APF thixotropic gel. One group of subjects was asked to rinse their mouths with tap water for two 30-sec periods immediately following the treatment and were permitted to eat or drink at will. The remaining subjects were not permitted to rinse, eat, or drink for 30 min. The fluoride content of the enamel specimens was determined at 30 min, 3, 7, 14, and 21 days following treatment using a microdrill technique. The study then was repeated in a crossover design. The results indicated that there was significantly less fluoride present in the enamel specimens when the subjects were allowed to rinse following the topical fluoride application. In both treatment groups a loss of fluoride from the enamel was observed as a function of time. For maximum fluoride efficacy, patients should not be permitted to rinse, eat, or drink for 30 min following the topical application of fluoride.

The practice of asking patients to refrain from rinsing, eating, or drinking for 30 min following a professionally applied topical fluoride treatment has been followed for many years.[1] The rationale for this procedure has been to give the fluoride additional time to "react" with the tooth surface. However, the need for this procedure has not been understood totally, identified, or even proven to exist. On the other hand, a number of recent reports have expressed concern regarding the amount of fluoride inadvertently ingested by children during a professionally applied topical fluoride treatment.[2-5] Because of the lack of scientific data indicating a benefit from not rinsing following a fluoride treatment and the concern regarding the inadvertent ingestion of fluoride during such treatments, a number of investigators[6-8] have recommended immediate removal of all residual fluoride in the oral cavity following topical application. The recommended procedure generally involves the use of a series of oral rinses and expectoration of all saliva produced over a 30-min period.

This study was designed to determine the effect of rinsing following a topical fluoride application on the cariostatic potential of the treatment. Cariostatic potential was determined in situ by the amount of fluoride deposition in, as well as the rate of fluoride release from, artificially induced incipient carious lesions following a professionally applied topical fluoride treatment.

Methods and Materials

The study utilized 14 subjects who had partial dentures which had been fabricated to accept enamel discs (3 mm diameter) in interproximal areas; the nature of these appliances has been described previously.[9] All participants read and signed informed consent statements prior to study initiation. The test design was a 2-way crossover using a randomized block distribution procedure. The subjects were divided randomly into 2 groups. One group received 1 treatment procedure during the first test period and the second

treatment procedure during the second period. The other group received treatments in the reverse order. Each crossover test period was three weeks.

The enamel specimens used in this study were prepared by removing 3 mm enamel cores from extracted sound human incisors. The dentin was removed leaving an enamel disc with a thickness of approximately 2 mm. During pretest preparation the specimens were mounted in the end of 1/4-in plastic rods for ease of handling. The outer surface of the enamel specimen was removed by grinding with 600 grit wet/dry sandpaper under a constant flow of water. The specimens then were polished using alumina[a] until a uniform surface was present. Each specimen then was immersed in a solution of 0.1 M lactic acid containing 0.2% polyacrylic acid[b] and 50% saturated hydroxyapatite (HAP) at a pH of 5.0 for 46 hr using essentially the procedure described by Chen et al.[10] The resulting artificially induced lesion was approximately 25μ in depth with a relatively sound outer layer having a thickness of 5-10 μ. All specimens were examined for pretest surface microhardness using a Vicker's® hardness indenter (3 indentations at 500 g load).

A topical fluoride treatment then was given to all subjects with their partial dentures and enamel chips in place using a 1.23% thixotropic acidulated phosphate fluoride (APF) topical gel.[c] The treatment technique utilized foam-lined trays.[d] The teeth were dried with air, the trays were approximately one-third filled with the gel and inserted into the subject's mouth. The subjects were asked to bite lightly to mold the trays around the teeth and the partial denture. The treatment time was 4 min with saliva and excess fluoride gel removed by aspiration. Following the topical treatment the trays were removed from the subject's mouth and, depending upon the group assignment, they were asked to either: (1) expectorate and refrain from rinsing their mouth or consume any liquids or solids for the next 30 min; or (2) expectorate and rinse their mouth with tap water (1.0 ppm F) twice for 30 sec each time, after which they were allowed to consume liquids or solids as desired.

The enamel specimens were removed from the partial dentures at posttreatment intervals of 30 min, 3, 7, 14, and 21 days and samples of enamel were removed for fluoride analysis using the microdrill technique described by Mobley.[11] This procedure involved drilling to a depth of 50μ into each specimen at each time interval. The enamel powder removed then was stored in a humid environment with all fluoride analyses performed following study completion. After each enamel sampling (drilling) procedure, the resulting hole was filled carefully with nail varnish and the enamel specimens were reinserted into the original partial denture and worn by the subject until the next time period.

At the end of the entire study the powdered enamel samples collected at each test period were analyzed for fluoride content by dissolving in 10μl 0.5 M $HClO_4$, buffering with 20μl citrate-EDTA and adding 20μl of distilled water. The fluoride content was determined using a calibrated fluoride ion-specific electrode[e] and a digital pH meter.[f] In addition, the surface microhardness of the specimens was measured as previously described.

The mean and standard error of the fluoride content of the enamel specimens for each group was calculated at each time interval. An analysis of variance was performed for the design. The *t*-test between treatment means at each sample period or between 2 sample periods of each treatment was done by using the design error mean square. Statistical differences were determined using a 1-tailed paired-*t* analysis ($p < 0.05$). Fluoride contents over the 21-day test period also were compared within groups to determine if the rate of loss was greater when a posttreatment rinse was employed. The analysis was performed using a least significant difference test ($p < 0.05$). The surface microhardness data were analyzed similarly.

Results

The results of the fluoride analyses of the induced incipient lesions are summarized in Table 1. No attempt was made to determine the fluoride content of the demineralized areas immediately following the topical fluoride applications, since it was felt that such assays would include a substantial amount of unreacted fluoride present in the lesions or on the enamel surface. At the conclusion of the 30-min period, the demineralized enamel from the patients who were not permitted to rinse, eat, or drink contained 13.85 μg F/mm^3. At this time, a significantly lower amount of fluoride (8.13 μg F/mm^3) was observed in patients who rinsed with tap water and were permitted to eat or drink during the 30-min postapplication period. This differential in the fluoride content of the demineralized enamel between the 2 treatment regimens persisted throughout the 21-day period and the differences were statistically significant at each sampling time.

The influence of time on the fluoride content of the demineralized enamel also is illustrated in Table 1.

[a] Gamma Alumina 3 — Buhler Co: Lake Bluff, IL.
[b] Carbopol #907 — BF Goodrich Co: Cleveland, OH.
[c] Gel II — Cooper Care Inc: Fairfield, NJ.
[d] Centrays — Cooper Care Inc: Fairfield, NJ.
[e] Model #9609 — Orion Research: Cambridge, MA.
[f] Accumet 620 — Fisher Scientific: Lexington, MA.

Table 1. Summary of Fluoride Uptake Data

Time (Following Treatment)	*No Posttreatment Rinse (μg F/mm³)*	*Posttreatment Rinse (μg F/mm³)*
30 min	13.85 ± 2.08*	8.13 ± 1.77
3 days	9.75 ± 2.08 **	4.72 ± 1.08 **
7 days	9.17 ± 1.90	3.05 ± 0.51
14 days	7.55 ± 1.62	4.28 ± 1.06
21 days	6.80 ± 1.66	3.51 ± 0.77

* Mean ± SEM.
** Values within brackets do not differ significantly.

With both of the posttreatment regimens a significant decrease in the enamel fluoride content occurred during the first 3 days following topical application of fluoride; the values of 9.75 and 4.72 μg F/mm³ for the no-rinse and rinse posttreatment regimens reflect decreases of 30 and 42%, respectively. The fluoride values during the remainder of the 21-day posttreatment period appeared to decrease as a function of time, particularly when the subjects were not permitted to rinse, eat, or drink during the 30-min period following the fluoride; however, the decrease in fluoride content between 3 and 21 days was not statistically significant in either treatment regimen.

Table 2 summarizes the results of the hardness measurements performed on the surface of the enamel specimens prior to the fluoride treatments and at the conclusion of the study. Both of the treatment regimens resulted in a significant increase in the hardness of the demineralized enamel with increases of 24.72 and 24.50 units in the 2 groups. However, no significant differences between the treatment regimens were observed.

Discussion

The results of this study indicate that rinsing at the conclusion of a professionally applied topical fluoride treatment and permitting the patients to eat or drink within 30 min following such treatments significantly reduced the fluoride content of artificially induced incipient lesions. It may be hypothesized that some of the fluoride present in the demineralized area may be either unreacted fluoride or the more soluble calcium fluoride rather than the desired, more permanently bound fluorhydroxyapatite. If so, one would suspect: (1) that fluoride in this form might be lost within a few days regardless of whether or not the patients were permitted to rinse, eat, or drink immediately following the topical fluoride treatment; and (2) that the enamel fluoride content ultimately would be comparable in the 2 treatment regimens. However, this hypothesis was not observed in the present study as there was nearly twice as much fluoride present in the enamel after 21 days following the no-rinse treatment regimen as compared to the alternative rinse procedure.

The finding of a continuous numerical decrease in enamel fluoride content during the interval between 3 and 21 days in the no-rinse treatment regimen, even though not statistically significant, suggests the possibility that fluoride dynamics following topical fluoride applications to incipient lesions may be different from that observed previously with sound enamel. Using sound enamel with both in vitro and in vivo studies, previous reports have shown appreciable deposition of fluoride in the outer few microns of enamel.[12-16] However, most of the fluoride was leached from the enamel during the first 24 hr following application and little change in fluoride content of the enamel was observed after 1 week.[12-16] In the present study there was a trend suggestive of a continuous loss of fluoride from the incipient lesions in the no-rinse regimen throughout the 21-day test period. This effect was unexpected and suggests the desirability of studying fluoride dynamics in demineralized areas during longer periods.

The microhardness data indicated that the topical fluoride treatment increased the hardness of the le-

Table 2. Summary of Enamel Surface Hardness Data

	Vicker's Hardness Values		
Group	*Pretest*	*Posttest*	*Δ*
No posttreatment rinse	56.20 ± 2.62* **	80.92 ± 4.15	24.72 ± 3.59
Posttreatment rinse	54.61 ± 2.89	79.11 ± 3.53	24.50 ± 3.98

* Mean ± SEM.
** Values within brackets do not differ significantly.

sions, but there were no significant differences between the 2 postapplication regimens at the end of the test period. Previous studies have shown that the remineralization of incipient lesions results in an increase in the surface hardness due to the increased mineral content.[17-19] Further, the ability of fluoride to enhance remineralization is well known.[20-24] Thus, the increased surface hardness following the topical fluoride applications was expected and was similar to that observed previously.[21] The fact that no difference in surface microhardness between the 2 postapplication regimens was detected in spite of significant differences in fluoride content is most likely due to the methods used to assess hardness and fluoride content in the present study. The fluoride was analyzed in a sample obtained by drilling to a depth of 50 μ, thereby including the entire lesion, while the hardness was measured in the outer 5-10 μ reflecting changes in only the surface of the lesions. If, in fact, fluoride was incorporated deeper into the lesion, its effect on remineralization may not be detectable by surface microhardness determinations and such measurements must be made on sections which traverse the depth of the lesion.[25]

The results obtained in this study indicate that permitting patients to rinse, eat, or drink during the first 30 min after a topical fluoride application results in less fluoride deposition in incipient lesions. The presence of greater levels of fluoride in incipient lesions which had not been rinsed following treatment must be considered as having a greater cariostatic potential. Until additional studies can be made, the recommendation not to permit patients to rinse, eat, or drink following professionally applied topical fluoride treatments should be continued. The amount of fluoride inadvertently ingested during a topical fluoride application should be minimized through the use of proper application procedures with aspiration plus thorough expectoration of residual treatment solution as described by LeCompte and Doyle.[26]

Conclusion

Significantly greater amounts of fluoride deposition in demineralized enamel were observed when patients were not permitted to rinse, eat, or drink for 30 min following the fluoride treatment. This difference in fluoride deposition was apparent throughout the 3-week test period. An increase in the hardness of the lesions, indicative of remineralization, was observed with both fluoride application procedures.

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1. Knutson JW: Sodium fluoride solution: technique for applications to the teeth. J Am Dent Assoc 36:37–39, 1948.
2. Ekstrand J, Kock G, Lindgren LE, Peterson LG: Pharmacokinetics of fluoride gels in children and adults. Caries Res 15:213–20, 1981.
3. LeCompte EJ, Doyle TE: Oral fluoride retention following various topical application techniques in children. J Dent Res 61:1397–1400, 1982.
4. LeCompte EJ, Whitford GM: Pharmacokinetics of fluoride from APF gel and fluoride tablets in children. J Dent Res 61:469–72, 1982.
5. LeCompte EJ, Rubenstein LK : Oral fluoride retention with thixotropic and APF gels and foam-lined and unlined trays. J Dent Res 63:69–70, 1984.
6. Ekstrand J: Pharmacokinetics of fluoride following topical fluoride application. IADR Program and Abstracts, no S46, 1985.
7. Allmann DW: Effect of fluoride on tissue cAMP and enzyme activity. IADR Program and Abstracts, no S48, 1985.
8. LeCompte EJ: Clinical application of topical fluoride products, risks, benefits, and recommendations. IADR Program and Abstracts, no S49, 1985.
9. Stookey GK, Schemehorn BR, Cheetham BL, Wood GD, Walton GV: In situ fluoride uptake from fluoride dentifrices by carious enamel. J Dent Res 64:900–903, 1985.
10. Chen WC, Zawacki SJ, Nancollas GH, White DJ: Constant composition remineralization of early carious lesions in bovine enamel. IADR Program and Abstracts, no 1135, 1985.
11. Mobley MJ: Fluoride uptake from in situ brushing with an SnF_2 and an NaF dentifrice. J Dent Res 60:1943–48, 1981.
12. Mellberg TR, Laakso PV, Nicholson CR: The acquisition of loss of fluoride by topically fluoridated human tooth enamel. Arch Oral Biol 11:1213–20, 1966.
13. Brudevold F, McCann HG, Nilsson R, Richardson B, Coklica V: The chemistry of caries inhibition. Problems and challenges in topical treatments. J Dent Res 46:37–45, 1967.
14. Aasenden R, Brudevold F, McCann HG: The response of intact and experimentally altered human enamel to topical fluoride. Arch Oral Biol 13:543–52, 1968.
15. Lovelock DJ: The loss of topically applied fluoride from the surface of human enamel in vitro using ^{18}F. Arch Oral Biol 18:27–29, 1973.
16. Bruun C: Uptake and retention of fluoride by intact enamel in vivo after application of neutral sodium fluoride. Scand J Dent Res 81:92–100, 1973.
17. Feagin F, Koulourides T, Pigman W: The characterization of enamel surface demineralization, remineralization, and associated hardness changes in human and bovine enamel. Arch Oral Biol 14:1407–17, 1969.
18. Koulourides T, Phantumvanit P, Munksgaard EC, Housch T: An intra-oral model used for studies of fluoride incorporation in enamel. J Oral Path 3:185–96, 1974.
19. Pearce EIF, Moore AJ: Remineralization of softened bovine enamel following treatment of overlying plaque with a mineral-enriching solution. J Dent Res 64:416–21, 1985.
20. Gelhard TBFM, ten Cate JM, Arends J: Rehardening of artificial enamel lesions in vivo. Caries Res 13:80–83, 1979.
21. Koulourides T, Cameron B: Enamel remineralization as a factor in the pathogenesis of dental caries. J Oral Path 9:255–69, 1980.
22. Silverstone LM: The effect of fluoride in the remineralization of enamel caries and caries-like lesions in vitro. J Public Health Dent 42:42–53, 1982.
23. Featherstone JDB, Cutress TW, Rodgers BE, Dennison PJ:

Remineralization of artificial caries-like lesions in vivo by a self-administered mouthrinse or paste. Caries Res 16:235–42, 1982.
24. ten Cate JM, Shariati M, Featherstone JDB: Enhancement of (salivary) remineralization by 'dipping' solutions. Caries Res 19:335–41, 1985.
25. Featherstone JDB, ten Cate JM, Shariati M, Arends J: Comparison of artificial caries-like lesions by quantitative microradiography and microhardness profiles. Caries Res 17:385–91, 1983.
26. LeCompte EJ, Doyle TE: Effects of suctioning devices on oral fluoride retention. J Am Dent Assoc 110:357–60, 1985.

Quotable quote: Reye syndrome update

The FDA is continuing its public education campaign about the possible relationship between the use of aspirin and other salicylates in treating influenza and chicken pox and the development of Reye syndrome (RS) in children and teenagers.

In a Public Health Service (PHS) pilot study completed last winter and published in the medical literature in October, 1985,[1] significantly more cases than controls had received salicylates during matched antecedent illnesses. Researchers found that this association held even after they analyzed the data for such potentially confounding factors as severity of antecedent illness; possible differential recall of illness, events, and medications by case and control parents; possible misclassification of medication; possible misclassification of noncases as cases or failure of participating centers to enroll patients with the syndrome; and possibility of exposure to salicylates after onset of RS and not during matched antecedent illness. In the pilot study of 30 patients with RS and 145 controls, 93% of the children who developed RS received salicylates in the antecedent illness, while only 46% of the controls had received salicylates.

During the last 5 years, 4 case-control state studies reported a statistically significant association between salicylate ingestion in an antecedent chicken pox or respiratory illness with the development of RS in children and teenagers (see August and December, 1982 *Drug Bulletins*). Figures from these 4 studies showed between 96 and 100% of the cases and between 44 and 71% of controls received salicylates.

In a national telephone survey recently conducted by FDA to assess public awareness of the possible association between RS and salicylates, only 12% of the 1155 parents surveyed said they would give their children aspirin for flu or chicken pox, and most of these were parents of older children. In addition, 84% of the parents said they were aware of RS and 53% were aware that aspirin products should be avoided when treating children for flu or chicken pox.

A previous survey conducted by the Centers for Disease Control in 1981 showed 69% of parents were likely to give aspirin to their children for these conditions. This compares favorably to the 12% figure 4 years later.

The FDA believes that this increased knowledge about RS, particularly among parents of young children, is due in part to education campaigns (including media coverage and the use of public service announcements) it has been conducting since 1982. These have been aimed primarily at parents of young children. The survey showed that physicians were also a significant source of information about RS.

In accordance with the findings of the 1985 survey that the parents of older children and teenagers were less likely to know about the possible association of aspirin and RS (and to findings in the pilot study that teenagers are more susceptible to RS than previously thought), current education efforts are being directed at these parents and at teenagers themselves, who may self-medicate but be unaware that they are susceptible to RS.

The FDA public education campaign continues to be conducted in conjunction with similar efforts by industry, pharmacists, and other health professionals. Included are posters for physicians' offices, store posters, public service radio and television announcements, notices in newspapers and magazines, a new brochure, and letters to health professionals.

FDA Drug Bulletin, DH & HS, PHS,
FDA, Rockville, MD. 15(4):40.
December, 1985.

[1] Hurwitz ES, et al.: Public Health Service study on Reye syndrome and medications: report of the pilot phase. N Engl J Med 313:849–57, 1985.