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# Primary, secondary and tertiary treatment of dental caries

## A 20-year case report

Eugene J. Whitaker, DMD, PhD

**D**ental caries may be considered the outcome of three factors: agent, host and environment. Dentists use periodic dental visits as a method of screening to prevent dental caries and its consequences. Primary prevention targets asymptomatic patients to identify early risk factors for caries and to alter the pathological process before lesions develop. Secondary prevention targets patients early in the disease process to arrest or reverse the process and to improve the prognosis.

Both primary and secondary preventive measures incorporate the medical model of caries treatment and involve the use of dietary control, oral hygiene instruction, oral rinses, sealants, varnishes and systemic and topical fluoride therapy to prevent, arrest and remineralize initial lesions. On the other hand, tertiary prevention targets lesions that are resulting in complications, such as tooth cavitation and pain. To avert the sequelae of such complications, the dental practitioner follows the surgical model to treat advancing dental disease.

In this report, I describe the use of primary, secondary and tertiary preventive measures to treat dental caries in a high-risk patient during a 20-year period.

### CASE REPORT

The most reliable predictor of high caries risk to the permanent dentition is the caries experience in the primary dentition, with caries in primary molars having the highest predictive value.<sup>1</sup> In December 1983, the patient, an 8-year-old boy, came to my office for an initial examination and bitewing radiographs (Figure 1).

## ABSTRACT

**Background.** This case report follows the 20-year clinical treatment of a patient at high risk of developing dental caries. The author describes the treatment of the primary and permanent dentitions.

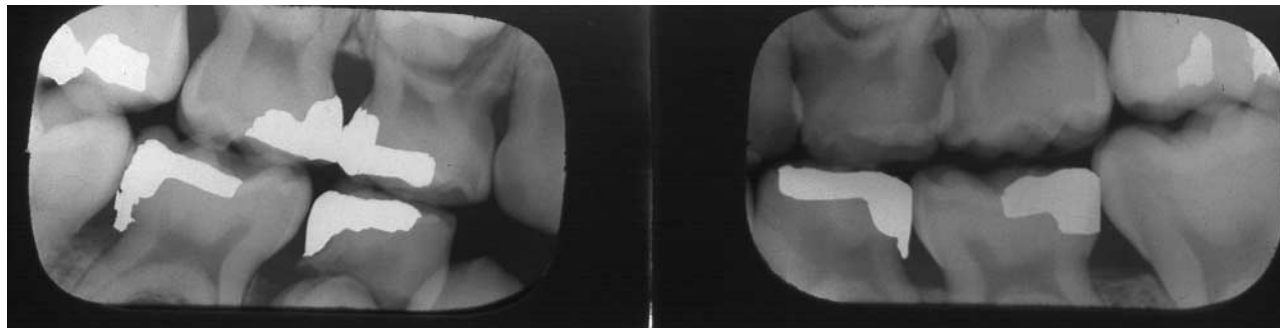
**Case Description.** The author describes the use of primary, secondary and tertiary measures to treat caries in a high-risk patient. He emphasizes the need for long-term care to monitor disease activity and describes the use of in-office tests for estimating *Streptococcus mutans* levels. This case illustrates the episodic nature of the caries process in which caries activity is related to risk factors, and the treatment regimen varies from tooth to tooth.

**Conclusion and Clinical Implications.** This case report documents the use of the medical paradigm in the treatment of dental caries, as well as the surgical paradigm as an important component of treatment.

**Key Words.** Dental caries; risk factors; medical paradigm.

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**Figure 1.** Initial radiographs obtained in December 1983, when the patient was aged 8 years.

The following teeth had been restored with amalgam: teeth nos. 3, A, B, 14, K, L, S and T. In addition, teeth nos. I and J had distal and mesial carious lesions, respectively. The clinical appearance of the patient's mouth placed him in a high-risk category.<sup>2</sup>

Routine questioning of the patient and his accompanying parent revealed no contributing medical history, and that two of the restorations (teeth nos. 3 and 14) had been placed within the last year. The parent reported that the boy drank fluoridated water and had received regular dental care beginning at age 3 years, including six-month recall appointments. The patient demonstrated adequate oral hygiene practices, and his parent confirmed that he brushed regularly at home and always at bedtime. However, the patient revealed that he ate sugary snacks after school and dinner without brushing his teeth immediately afterward.

**Prevention strategy.** I initiated the following caries preventive strategy: tertiary treatment of carious teeth nos. I and J, consisting of placement of amalgam restorations; primary treatment of teeth nos. 19 and 30, involving the placement of occlusal sealants; modification of the patient's diet to include fruit snacks after school and dinner; toothbrushing with fluoridated toothpaste immediately after breakfast and dinner; and rinsing with fluoridated mouthrinse after brushing before bedtime.

Three months after I placed the sealants, the patient had a recall appointment. Because his compliance was good and there was no clinical evidence of new or recurrent carious lesions, I instructed the patient to come back for six-month recall visits. I also referred him to an orthodontist because of an angle Class I malocclusion with

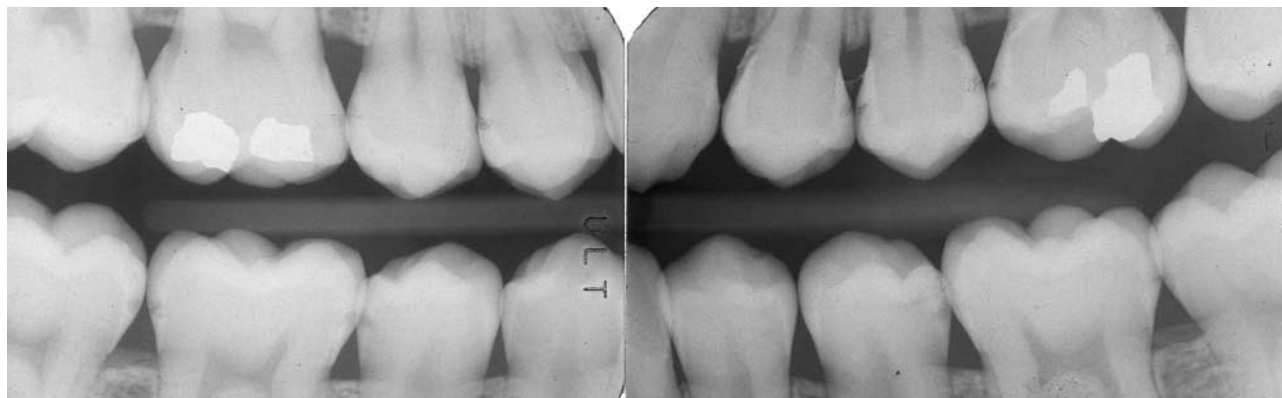
bilateral posterior crossbite, constricted palate and anterior open bite resulting from a thumb-sucking habit.

**Recall visits.** From 1984 through 1987, the patient had six-month recall appointments, and he received orthodontic therapy from 1985 through 1987. During the period in which he received orthodontic treatment, I prescribed topical fluoride brush-on gel for use after brushing in the morning and before bedtime. I obtained bitewing radiographs in February 1985, January 1986, August 1986 and August 1987. At this last visit, the patient was aged 12 years and his second molars were erupted fully. I placed occlusal sealants at this visit. Throughout this period, no new carious activity was evident.

However, the patient did not keep his recall appointments throughout 1988, and a clinical examination in March 1989 revealed a disappointing clinical picture. Bitewing radiographs (Figure 2) indicated demineralization of the following teeth: 2, 3, 4, 5, 12, 13, 14, 18, 19, 20, 29, 30 and 31. Because caries is a dynamic process, the progress of individual lesions is an important factor in the diagnosis and prognosis.<sup>3</sup> Clinical carious activity needs to be measured over time, and changes in disease activity indicate the need for strict periodic monitoring of high-risk patients.

**Caries risk assessment tests.** Because of the patient's clinical appearance and with the emergence in the mid-1980s of in-office, chairside tests to measure *Streptococcus mutans* levels, I recommended that the patient undergo caries risk assessment tests to help determine the factors that made him susceptible to dental caries.<sup>4</sup> These tests involve the use of saliva to measure

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**Figure 2.** Radiographs obtained in March 1989. The initial demineralization is seen interproximally on teeth nos. 2, 3, 4, 5, 12, 13, 14, 18, 19, 20, 29, 30 and 31. The lesions on the distal surface of teeth nos. 20 and 29 had advanced into the dentinoenamel junction.

flow rate and buffering capacity, which are nonetiologic factors but indicate caries risk, as well as measurement of *S. mutans*, which is an etiologic factor. Although the salivary flow rate and buffering capacity were normal, *S. mutans* levels were high ( $\geq 10^6$  colony-forming units/milliliter), as counted on mitis-salivarius agar containing sucrose and bacitracin.

In-office tests to measure *S. mutans* levels are not practical for every patient, because they measure only one component of the disease process, and a single microbiological examination is of little value on its own.<sup>5</sup> In addition, these tests are not sensitive (that is, a negative result is equally likely to be a false-negative or true-negative result), and the tests are not good at predicting the onset of disease activity. However, salivary determination of *S. mutans* levels is highly specific (that is, a positive test result is almost always a true positive result), and high counts likely correlate with caries activity.<sup>6</sup> Thus, a patient identified as being at high risk of developing caries on the basis of caries experience and whose radiographs reveal changes that reflect carious activity is most likely to test positive for *S. mutans*.

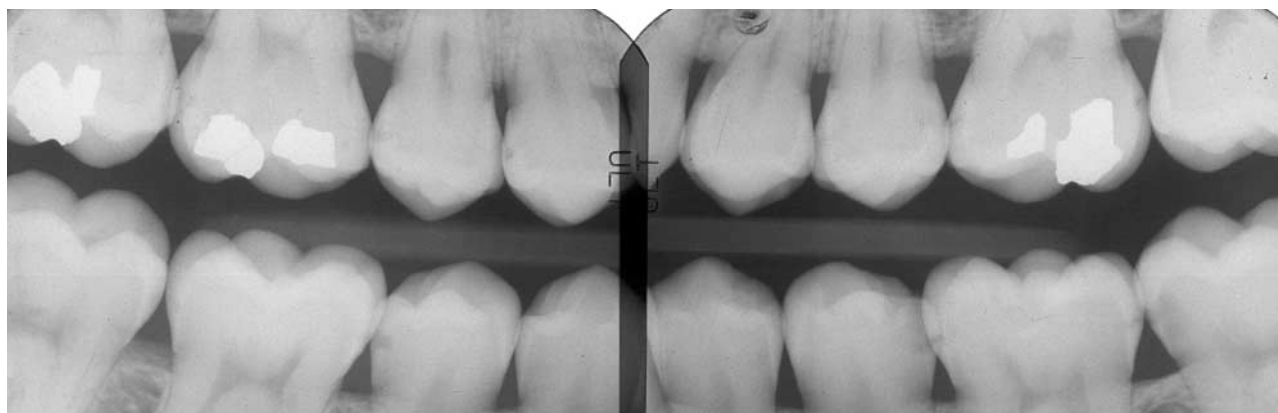
It may seem unnecessary to test patients bacteriologically while carious lesions, the nidi of infection, are left unrestored. However, a caries risk assessment can be used to motivate the patient (and, in this case, the parents) to intercept the disease process. For the moderate-to-high-risk patient, caries risk assessment allows for a custom-made prevention program.<sup>7</sup> In this case, the microbiological method was a valuable adjunct to taking a medical history and conducting a careful clinical examination, as well as an aid in diagnosing and explaining the disease activity.

After March 1989, the caries prevention strategy for this patient was as follows: because most of the lesions were noncavitated, the primary objective was to alter the oral environment to promote remineralization. Hence, I reinforced oral hygiene techniques and prescribed a fluoride gel (5,000 parts per million) to be applied in trays twice daily for 15 minutes each.

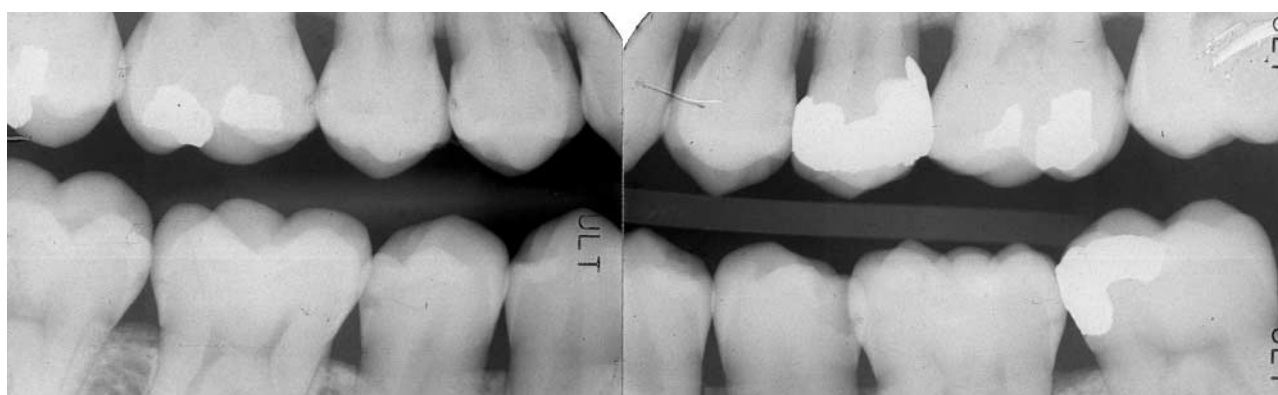
**Restorative treatment.** Dental practitioners generally recognize that surgical treatment is not indicated for lesions confined to the inner one-half of enamel and even for those that extend into dentin.<sup>8</sup> However, the presence of caries in the distal triangular fossa and in the distal surface of tooth no. 20 undermined the marginal ridge (Figure 2). Thus, in June 1989, I restored this tooth with resin-based composite using a minimally invasive tooth preparation,<sup>9</sup> such that buccal and lingual triangular ridges were not invaded by the restoration and distal contact was not broken (tunnel preparation). At this time, I also placed a preventive-resin restoration on the occlusal surface of tooth no. 19 and an occlusal amalgam restoration in tooth no. 2. I did not treat the other lesions surgically.

According to Anusavice,<sup>8</sup> radiographic evidence of carious activity is not sufficient to warrant placing a restoration; rather, each tooth must be monitored over time to determine if the lesions can be arrested. My patient had recall visits every three months for the remainder of 1989. In September 1989, I used an in-office test to again record *S. mutans* levels, which were low ( $\leq 10^5$  CFU/mL).

The patient continued the at-home oral hygiene regimen until December 1989. He then stopped using the fluoride trays and began using over-the-counter (OTC) mouthrinses (0.05 percent



**Figure 3.** Radiographs obtained in February 2000. The lesions in teeth nos. 13 and 18 were treated surgically, but the other interproximal lesions (for example, the distal surface of teeth nos. 19 and 29) were treated medically.



**Figure 4.** The patient's most recent radiographs were obtained in December 2004, when he was aged 29 years. Many lesions that had been observed in March 1989 (for example, tooth no. 29) were treated without operative intervention.

sodium fluoride) after brushing, because the clinical examination findings and bitewing radiographs revealed no advancement of the interproximal lesions. The patient's therapy from March until December 1989 was consistent with the recommendation of Anderson and colleagues,<sup>6</sup> who advised that caries be treated as a bacterial infection and, hence, be treated intensively on a short-term basis to a therapeutic endpoint.

I saw the patient at periodic six-month recall visits throughout the 1990s and found no change in caries activity. Every 18 months I obtained bitewing radiographs. Dental treatment was unremarkable throughout this period, with the exception of the extraction of the patient's third molars in March 1991. Bitewing radiographs obtained in February 2000 revealed a change in caries activity; lesions had advanced in teeth nos. 13 and 18 (Figure 3). Although a review of the patient's medical history revealed that he had been diagnosed with esophageal reflux in September 1999 (for which he was treated with omeprazole), his salivary buffering capacity was

normal in February 2000. The enlarged lesions in teeth nos. 13 and 18 illustrate the episodic and site-specific nature of the caries process, with rapid demineralization occurring as a result of bacterial proliferation in protected cavitated areas.

I placed amalgam restorations in teeth nos. 13 and 18. Through April 2000, the patient had three recall visits, at each of which he received fluoride varnish applications. I prescribed chlorhexidine (0.12 percent), to be used twice daily (0.5-ounce doses) for three months. In addition, I instructed the patient to chew xylitol gum for five minutes three times per day.

In September 2000, I found no progression in carious activity and again instructed the patient to have six-month recall appointments. To date, his long-term home care regimen includes brushing with a fluoridated toothpaste three times a day, flossing, rinsing with an OTC mouthrinse (0.05 percent sodium fluoride) and chewing xylitol gum. Figure 4 shows his most recent radiographs, obtained in December 2004.

Of interest is that of the 11 teeth with radiographic evidence of lesions in March 1989, four were treated operatively and seven were treated via nonsurgical modalities that recognize the potential of teeth to remineralize. Tooth no. 29 is of particular interest because the lesion had not advanced further into dentin since it was first observed in 1989.

## DISCUSSION

This case is based on the medical paradigm of treating dental caries by addressing the bacterial cause of the disease and emphasizing disease control. However, the case still involved the surgical paradigm, but it consisted of a minimally invasive approach in which surgical intervention was deferred until it could no longer be avoided. In addition, the patient was involved in treating the disease. Patient compliance is essential. Lesions confined to enamel and those extending slightly into dentin were arrested; only those with cavitation were treated surgically.

## CONCLUSION

This case documents the use of a treatment regimen for dental caries proposed by Anderson

and colleagues,<sup>6</sup> Anusavice<sup>8</sup> and others that integrates clinical experience, professional judgment and patient preference in the development of an individualized treatment plan. It is, however, only one case, and stringent, controlled, double-blind, longitudinal clinical trials are needed to validate the efficacy of the therapy for the population at large. ■

1. Li Y, Wang W. Predicting caries in permanent teeth from caries in primary teeth: an eight-year cohort study. *J Dent Res* 2002;81:561-6.

2. Caries diagnosis and risk assessment: a review of preventive strategies and management. *JADA* 1995;126(supplement):1S-24S.

3. Bader JD, Brown JP. Dilemmas in caries diagnosis. *JADA* 1993;124:48-50.

4. Krasse B. Caries risk: a practical guide for assessment and control. Chicago: Quintessence; 1985.

5. Bowen WH. Interpretation and use of microbiological findings in dental caries. *Oral Microbiol Immunol* 1986;1(1):82-6.

6. Anderson MH, Bales DJ, Omnell K. Modern management of dental caries: the cutting edge is not the dental bur. *JADA* 1993;124(6):37-44.

7. Powell LV. Caries risk assessment: relevance to the practitioner. *JADA* 1998;129:349-53.

8. Anusavice KJ. Treatment regimens in preventive and restorative dentistry. *JADA* 1995;126:727-43.

9. Murdoch-Kinch CA, McLean ME. Minimally invasive dentistry. *JADA* 2003;134(1):87-95.