Threshold for Surgical Intervention

We can justify a delay in restorative treatment of enamel lesions in the inner half of enamel and even slightly into dentin on the basis that caries progression in moderate-risk and high-risk patients through enamel is slow.[9] Caries progression has been decreasing over recent decades and is slower in patients who have received regular fluoride treatment or who consume fluoridated water.[10,11] Progression times through enamel may take from six to eight years.[12-16] Since many enamel lesions remain unchanged or progress very slowly over long periods and because progression rates through dentin also may be comparably slow, there is adequate time to apply infection control and monitoring procedures to assess caries risk and lesion activity status over extended periods of time.[17]

To minimize variability in decision-making and to optimize cost-effectiveness and the cost-benefit parameters of care, the strongest evidence on treatment regimens must be used. Summarized in Table 1 is a comparative assessment of the strength of evidence of various treatment options for caries management in coronal areas of permanent teeth for adolescent and adult patients. As can be clearly seen, the strength and quality of data-supporting management options for adult patients are very poor. This deficiency is associated in part with the failure to monitor treatment efficacy as a function of individual risk over time.

For teeth with cavitated surfaces, a restoration should be placed after initial efforts to reduce caries risk have been taken. As shown in Table 2, all tooth surfaces with cavitated lesions should be restored since they cannot be reliably remineralized and maintained free of plaque. For teeth with approximal lesions, the surface integrity cannot readily be determined unless the teeth are separated or the lesion severity is sufficiently great (middle third of dentin, D2, or inner third of dentin, D3) that the probability of cavitation is very high.[18] The results of these studies indicate that approximately 60 percent of approximal tooth surfaces with radiolucencies extending into the outer half of dentin are not cavitated.[18] However, these results do not agree well with those of Akpata et al. who reported that only 20.9 percent of the surfaces were not cavitated when the lesions were found in the outer half of dentin.[19] This difference could have been explained if the individual subjects had been assessed for risk at baseline and over the course of the study prior to cavitation assessment.

Foster investigated the proportion of approximal carious lesions extending up to 1 mm into dentin that progressed over a three-year period.[20] After thirty-six months, lesions that extended over 0.5 mm and up to 1 mm into the dentin were significantly more likely to have progressed (92 percent) than shallower lesions that extended up to only 0.5 mm into dentin (50 percent). These results sug-

Table 1. Strength and quality of evidence on efficacy of caries management treatment options for high-risk patients (0 = none, 1 = minimal, 2 = fair; 3 = good)

Treatment Option	Adolescents	Adults
Fluoride toothpaste	2	2
Fluoride tablets, mouthrinses, or combined fluoride sources	2	2
Fluoride varnish only	2	0
Sealant only	2	1
Chlorhexidine only	1	1
Chlorhexidine plus fluoride	1	1
Sealant plus chlorhexidine	0	0
Sealant plus fluoride	1	0
Sealant plus chlorhexidine and fluoride	0	0

Table 2. Treatment options based on caries risk, lesion severity, and surface integrity

	Low Risk	Moderate Risk	High Risk	High Risk
Lesion Severity	E1, E2	E2, D1	D1, D2, D3	D1, D2, D3
Surface Integrity	Noncavitated or Questionable	Questionable	Cavitated	Cavitated
Caries Activity	Inactive or questionable	Questionable	Active, progressing slowly	Active, progressing rapidly
Treatment Option	Diet and oral hygiene control; monitor for new lesions at 6- to 12-mo recall periods	Diet and oral hygiene control; professional and home flossing with 1% CHX; periodic F; monitor at 6-mo recall periods until shifted to low risk (or < 2.5 x 10^5 CFU/mL)	Diet and oral hygiene control; professional and home flossing with 1% CHX; seal pits and fissures; restore all cavitated surfaces; daily F; monitor at 3- to 6-mo recall periods until shifted to low risk (< 2.5 x 10^5 CFU S. mutans/mL)	Diet and oral hygiene control; professional and home flossing with 1% CHX; seal pits and fissures; restore all cavitated surfaces; daily F; monitor at 1- to 3-mo recall periods until risk is reduced (< 2.5 x 10^5 CFU S. mutans/mL)

gest that operative intervention be considered for approximal lesions that extend deeper than 0.5 mm into the dentin, while preventive treatment and re-assessment may be considered for shallower lesions.

Professional flossing with chlorhexidine gel should be performed at least four times per year to ensure adequate reduction of *S. mutans* levels. Since chlorhexidine is not effective against lactobacilli, plaque removal and diet modification may reduce the level of lactobacilli and the probability for lesion progression. In addition, fluoride therapy should be closely linked with the use of chlorhexidine use for high-risk patients. Little additional benefit is likely to be realized by treating moderate-risk or low-risk patients with chlorhexidine, although fluoride therapy is still advised for moderate-risk patients until they are shifted to a low-risk level.

Clearly, monitoring for positive and negative changes in the activity status of the disease is the most important aspect of caries management. If the caries process is active, early interventions to arrest the process will reduce the probability of cavitation and potential restorations. Monitoring at intervals determined by risk also will ensure that the prescribed treatment benefits will be sustained and that low-risk patients will not be overtreated. Since the strength and quality of evidence for most treatment options are relatively poor overall (Table 1), doses and frequencies of therapeutic agents must be adjusted periodically, depending on whether the targeted outcomes are achieved or not.

REFERENCES

1. Selwitz RH, Nowjack-Raymer R, Driscoll WS, Li S-H. Evaluation after 4 years of the combined use of fluoride and dental sealants. Community Dent Oral Epidemiol 1995;23:30-5.
2. Llodra JC, Bravo M, Delgrado-Rodriguez M, Baca P, Galvez R. Factors influencing the effectiveness of sealants—a meta analysis. Community Dent Oral Epidemiol 1993;21:261-8.
3. Mertz-Fairhurst EJ, Curtis JW, Ergle JW, Rueggeberg FA, Adair SM. Ultraconservative and cariostatic sealed restorations: results at year 10. J Am Dent Assoc 1998;129:55-66.
4. van Rijkom HM, Truin GJ, van 't Hof MA. A meta-analysis of clinical studies on the caries-inhibiting effect of chlorhexidine treatment. J Dent Res 1996;75(2):790-5.
5. Gisselsson H, Birkhead D, Björn AL. Effect of professional flossing with chlorhexidine gel on approximal caries in 12- to 15-year-old schoolchildren. Caries Res 1988;22:187-92.
6. Zickert I, Emilson CG, Krasse B. Effect of caries preventive measures in children highly infected with the bacterium Streptococcus mutans. Arch Oral Biol 1982;27:861-8.
7. Luoma H. A simultaneous reduction of caries and gingivitis in a group of school children receiving chlorhexidine-fluoride applications. Caries Res 1978;12:290-8.
8. Axelsson P, Buischi YA, Barbosa MF, Karlsson R, Prado MC. The effect of a new oral hygiene training program on approximal caries in 12-15-year-old Brazilian children: results after three years. Adv Dent Res 1994;8:278-84.
9. Shwartz M, Grondahl HG, Pliskin JS, Boffa J. A longitudinal analysis from bite-wing radiographs of the rate of progression of approximal carious lesions through human dental enamel. Arch Oral Biol 1984;29(7):529-36.
10. Ekanayake LS, Sheiham A. Reducing rates of progression of dental caries in British schoolchildren: a study using bitewing radiographs. Br Dent J 1987;163(8):265-9.
11. Pitts NB. Monitoring of caries progression in permanent and primary posterior approximal enamel by bitewing radiography. Community Dent Oral Epidemiol 1983;11:228-35.
12. Zamir T, Fisher D, Fishel D, Sharav Y. A longitudinal radiographic study of the rate of spread of human approximal dental caries. Arch Oral Biol 1976;21:523-6.
13. Gröndahl H-G. Dental caries and restorations in teenagers: II. A longitudinal study of caries increment in proximal surfaces among urban teenagers in Sweden. Swed Dent J 1977;1:51-7.
14. Berkey CS, Douglass CW, Valachovic RW, Chauncey HH. Longitudinal radiographic analysis of carious lesion progression. Community Dent Oral Epidemiol 1988;16:83-90.
15. Hugoson A, Goran K, Hallonsten AL. Caries prevalence and distribution in individuals aged 20-80 years in Jonkoping, Sweden, 1973 and 1983. Swed Dent J 1988b;12(4):133-40.
16. Hugoson A, Goran K, Bergendal T, Laurell L, Lundgren D. Caries prevalence and distribution in individuals aged 3-20 years in Jonkoping, Sweden, 1973, 1978, and 1983. Swed Dent J 1988a;12:125-32.
17. Emslie RD. Radiographic assessment of approximal caries. J Dent Res 1959;38:1225-6.
18. Pitts NB, Rimmer PA. An in vivo comparison of radiographic and directly assessed clinical caries status of posterior approximal surfaces in primary and permanent teeth. Caries Res 1992;26:146-52.
19. Akpata ES, Farid MR, al-Saif K, Roberts EA. Cavitation at radiolucent areas on proximal surfaces of posterior teeth. Caries Res 1996;30(5):313-6.
20. Foster LV. Three year in vivo investigation to determine the progression of approximal primary carious lesions extending into dentine. Br Dent J 1998;185(7):353-7.