Clinical decision making for caries management in children

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Abstract
The aim of this review of clinical decision making for caries management in children is to integrate current knowledge in the field of cariology into clinically usable concepts and procedures. Current evidence regarding the carious process and caries risk assessment allows the practitioner to go beyond traditional surgical management of dental caries. Therapy should focus on patient-specific approaches that include disease monitoring and preventive therapies supplemented when necessary by restorative care. The type and intensity of these therapies should be determined utilizing clinical data as well as knowledge of the caries process for that child. Changes in the management of dental caries will require health organizations and dental schools to educate students, practitioners, and patients in evidence- and risk-based care. (Pediatr Dent. 2002; 24:386-392)

KEYWORDS: DENTAL CARIES, EVIDENCE-BASED PRACTICE, PREVENTIVE DENTISTRY, RESTORATIVE DENTISTRY, PEDIATRIC DENTISTRY, RISK ASSESSMENT, DECISION MAKING

Historically, management of dental caries in primary and permanent teeth has involved clinical and radiographic identification of carious lesions followed by surgical intervention to remove and restore affected enamel and dentin. Only modest changes over the years have occurred in this surgical approach to dental caries treatment. Appropriate dental care in a child requires an understanding of the carious process that includes: (1) location and extent of the lesion, (2) patient’s age, (3) assessment and reassessment of disease activity, (4) prior therapy outcomes, (5) natural history of caries progression, and (6) preferences and expectations of guardians and practitioners (Fig 1). In this model, a child who has been identified as being at low risk for dental caries may need few diagnostic procedures and preventive therapies. Conversely, a child that is caries active may require frequent diagnostic procedures, intensive preventive therapies and restorative interventions.

Factors in decision making

Natural history of caries
A unique feature regarding caries management in children is the age of the child.

The earlier that a child becomes colonized with the cariogenic bacterial group, mutans streptococci, the greater is the child’s caries risk.1,2 Mutans streptococci are believed to be particularly caries conducive because of their ability to adhere to tooth surfaces, produce copious amounts of acid, survive and continue metabolism at low pH conditions.3 Permanent colonization of a child’s oral cavity with mutans streptococci can occur only after tooth eruption because mutans streptococci requires a non-shedding surface for attachment.4 Such colonization is generally the result of transmission of these organisms from the child’s primary caregiver, usually the mother.5 Those teeth that are first exposed to a cariogenic environment generally will be the first to show signs of disease. Consequently, children at high risk for early childhood caries may develop lesions on their maxillary anterior teeth soon after eruption.4 If these children continue to be at high risk, they may develop fissure caries of the primary molars and, later, molar proximal caries.7 Children with moderate caries risk will develop caries at a later age, normally molar fissure caries and possibly molar proximal caries.6,8 In general, caries on maxillary anterior primary teeth and on the molar proximal surfaces suggests high caries activity.

At the individual lesion level, caries progression is dependent on the site of the lesion and level of risk and disease activity, as well as age. Buccal-lingual smooth surface lesions, even if cavitated, may be readily amenable to preventive regimens, while cavitated fissure or cavitated proximal lesions may need restorative therapy to limit progression. Caries activity can be assessed by observing the speed of progression of existing lesions or the incidence of new lesions.
Four articles were located that examined caries progression of proximal lesions in primary teeth (Table 1). Even though 3 are confounded by the presence of preventive regimes, results are similar among studies with 72%-81% of lesions remaining in enamel after 1 year. In the fourth study, proximal lesion progression through primary tooth enamel in high-risk subjects not receiving fluoride took approximately 1.5 years. In low-risk children receiving regular topical fluoride therapy, progression took 3.5 years.10

Nine articles were identified that examined lesion progression of proximal caries in permanent teeth of children (Table 2). These studies showed that proximal lesion progression was even slower than in primary teeth. In the majority of these reports, radiographically evident enamel lesions remained in enamel for more than 1 year, with several studies showing that lesions remain in enamel after 3 years. Thus the accumulated evidence suggests that radiographic proximal lesions limited to the enamel may not require immediate surgical intervention, and sufficient time exists to implement and longitudinally evaluate preventive interventions.

Diagnosis

Decisions for therapy often are based on whether a tooth is diagnosed as cavitated by clinical or radiographic examination. The accuracy of correctly identifying fissure caries in permanent teeth by visual and tactile methods is in question. In general, the commonly used visual-tactile technique has low sensitivity (ability to correctly identify a tooth with caries), but high specificity (ability to correctly identify a tooth without caries).22 Only 1 article was located that addressed the validity of the diagnosis of fissure caries in primary teeth.23 Visual identification without the use of an explorer was reported to have a sensitivity of 0.45 and a specificity of 1.00. Interestingly, bitewing radiographs identified dentin caries originating in fissures with a sensitivity of 0.93 and a specificity of 0.89.

Three articles were located that examined the validity of radiographic proximal caries diagnosis in primary teeth. The majority of enamel lesions detected on radiographs are not cavitated and are not detectable clinically;24 and, in conflict with traditional understanding, many lesions that appear radiographically to be in the outer dentin also may not be cavitated (Table 3). Newer and more sensitive methods of clinical caries detection, such as laser fluorescence, fiber optic transillumination, and electrical conductivity, appear promising; yet, at this time, there is little evidence of the validity and reliability of these new approaches from human clinical trials.28 Contrary to new technologies, practicing dentists can obtain feedback on false positive and false negative visual-tactile diagnoses when they instrument a tooth. If a surgical intervention is justified on questionable lesions in a child, the tooth most likely to be carious may be opened and the diagnosis confirmed. This technique can determine whether interventions on other teeth in that child are needed.29 There also is evidence that a carious lesion sealed from the oral environment do not progress, and, therefore, inadvertent
covering of undetected caries with pit and fissure sealant will not cause harm provided the sealant remains intact.\textsuperscript{30,31}

In addition to determining whether a tooth is cavitated or not, caries diagnosis should attempt to estimate the more critical issue—whether a lesion is progressing or arrested. Currently, longitudinal evaluation of lesion progression at periodic recall visits is the best method to determine lesion activity and progression. Along with other information, such as the likelihood of a patient returning for recall visits and depth of a lesion, an active carious lesion may require preventive and restorative therapy, whereas non-active or arrested lesions may require no therapy. Such patient- and tooth-specific evaluations of caries diagnosis and progression will require changes from current practice since longitudinal information has been reported not to change dentists’ decision-making process.\textsuperscript{32}

Caries risk assessment

The goal of caries risk assessment in dentistry is to deliver preventive and restorative care specific to an individual patient’s needs. A current obstacle for clinical implementation of caries risk assessment is the lack of research studies investigating how the application of risk assessment methodologies affect future dental health outcomes. Furthermore, there is not one caries risk factor or combination of factors that have achieved high combinations of both positive and negative predictive values,\textsuperscript{33} although previous carious experience remains the best indicator of future caries development.

In young children, previous caries experience is not particularly useful since it is important to determine caries risk before

<table>
<thead>
<tr>
<th>Year</th>
<th>Author and country</th>
<th>N</th>
<th>Age at start</th>
<th>% of radiographically visible enamel lesions remaining in enamel</th>
<th>Lesion progression in months</th>
</tr>
</thead>
<tbody>
<tr>
<td>1981</td>
<td>Craig et al\textsuperscript{2} Australia</td>
<td>54</td>
<td>6-8 y</td>
<td>First primary molar: 81% after 1 y 81% after 2 y Second primary molar: 72% after 1 y 69% after 2 y</td>
<td>Sweden, bi-weekly fluoride, (hi/lo risk): 11/15 mo from outer to inner enamel 15/25 mo from inner enamel to dentine US: no fluoride, (hi/lo risk): 9/16 mo from outer to inner enamel 10/9 mo from inner enamel to dentine</td>
</tr>
<tr>
<td>1984</td>
<td>Shwartz et al\textsuperscript{3} Sweden and US</td>
<td>217</td>
<td>10-11 y (at end) US: 4-17 y (at end)</td>
<td>73% of outer lesions after 1 y 54% of inner lesions after 1 y 60% of any enamel lesion after 1 y</td>
<td></td>
</tr>
<tr>
<td>1992</td>
<td>Solanki and Sheiham\textsuperscript{4} England</td>
<td>50</td>
<td>5 y</td>
<td>78% of outer lesions after 1 y 55% of outer lesions after 2 y 29% of inner lesions after 1 y</td>
<td></td>
</tr>
<tr>
<td>1992</td>
<td>Peyron et al\textsuperscript{5} Sweden</td>
<td>468</td>
<td>3-4 y</td>
<td>78% of outer lesions after 1 y 55% of outer lesions after 2 y 29% of inner lesions after 1 y</td>
<td></td>
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<td>78% of outer lesions after 1 y 55% of outer lesions after 2 y 29% of inner lesions after 1 y</td>
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Table 1. Evidence of the Rate of Progression of Proximal Caries in Primary Teeth

<table>
<thead>
<tr>
<th>Year</th>
<th>Author and country</th>
<th>N</th>
<th>Age at start</th>
<th>% of radiographically visible enamel lesions remaining in enamel</th>
<th>Lesion progression in months</th>
</tr>
</thead>
<tbody>
<tr>
<td>1976</td>
<td>Zamir et al\textsuperscript{6} Israel</td>
<td>51</td>
<td>14-15 y</td>
<td>80% of outer lesions after 2 y 50% of inner lesions after 2 y</td>
<td>13.6 mo from outer to inner enamel 12.8 mo from inner enamel to dentine</td>
</tr>
<tr>
<td>1973</td>
<td>Berman and Slack\textsuperscript{7} England</td>
<td>353</td>
<td>11 y</td>
<td>52% of lesions after 3 y</td>
<td></td>
</tr>
<tr>
<td>1975</td>
<td>Haugejorden and Slack\textsuperscript{8} England</td>
<td>40</td>
<td>13-15 y</td>
<td>78% of outer lesions after 1 y 60% of inner lesions after 1 y</td>
<td></td>
</tr>
<tr>
<td>1981</td>
<td>Powell et al\textsuperscript{9} (1963 data) Australia</td>
<td>307</td>
<td>12-14 y</td>
<td>92% of inner lesions after 1 y</td>
<td></td>
</tr>
<tr>
<td>1984</td>
<td>Grondahl et al\textsuperscript{10} Sweden</td>
<td>135</td>
<td>13 y</td>
<td>82% of outer lesions after 3 y 45% of inner lesions after 3 y</td>
<td></td>
</tr>
<tr>
<td>1989</td>
<td>Bille and Carstens\textsuperscript{11} Denmark</td>
<td>278</td>
<td>13 y</td>
<td>90% of outer lesions after 2 y 79% of inner lesions after 2 y</td>
<td></td>
</tr>
<tr>
<td>1990</td>
<td>Lervik et al\textsuperscript{12} Norway</td>
<td>65</td>
<td>14-15 y</td>
<td>99% of outer lesions after 1 y 89% of inner lesions after 1 y</td>
<td>19 mo from outer to inner enamel 22 mo from inner enamel to dentine</td>
</tr>
<tr>
<td>1997</td>
<td>Hintze\textsuperscript{13} Denmark</td>
<td>219</td>
<td>14 y</td>
<td>69% of lesions after 3 y</td>
<td></td>
</tr>
<tr>
<td>1997</td>
<td>Lawrence and Sheiham\textsuperscript{14} Brazil</td>
<td>290</td>
<td>12-16 y</td>
<td>Fluoridated areas: 99% of outer lesions after 1 y 89% of inner lesions after 1 y Non-fluoridated areas: 98% of outer lesions after 1 y 87% of inner lesions after 1 y</td>
<td></td>
</tr>
</tbody>
</table>

Table 2. Evidence of the Rate of Progression of Proximal Caries in Permanent Teeth
have increased. creased frequency of sucrose consumption, risk status may appliance therapy, increase in mutans streptococci levels, in- detected or there are changes in the oral environment (eg, decreased. If there are increased numbers of new lesions are not detected, caries activity may be considered to have recall visits allows for better appraisal of caries activity and refinement of decisions for caries management. If, at a re- call visit, existing lesions have not progressed and new lesions of therapy, ongoing reassessment of a child’s caries risk at low risk for dental caries. Conversely, children at high risk require intense prevention to prevent caries initiation and to arrest caries progression.

Parent and practitioner preferences

The responsible parent(s), with the advice of the dental professional, is the one who must make decisions for dental therapy. In many cases, as a result of past experiences, the parent assumes that only surgical techniques can treat dental caries. The dental professional is obliged to inform the parent about alternative therapies based on scientific evidence, should change over time as a result of scientific progress and the practitioner’s continued learning and self-evaluation of outcomes.

Preventive therapy

Decisions for preventive therapy should be based on an understanding of risk indicators for the child. Very often, there is little discrimination on the intensity and type of preventive therapies that are prescribed to groups or individuals. Risk-based therapy assumes that there will be little benefit of preventive therapies for those children who are at low risk for dental caries. Conversely, children at high risk require intense prevention to prevent caries initiation and to arrest caries progression.

The most widely used method of applying fluoride topically is by means of dentifrice. Daily/twice-daily fluoride exposure through the controlled use of fluoridated dentifrice is now considered a major approach in the reduction of dental caries. Additionally, professional topical fluoride therapies, home fluoride mouth rinses and concentrated tray/brush-on therapies have had a long history of use to prevent dental caries. Except for recommending regular use results of risk assessment analysis, expected outcomes, and cost. Enabling the parent to be an active participant in choosing preventive and restorative therapies should produce better parent and patient compliance. Besides the obligation of thorough informed consent for therapy decisions, a dental professional may by training, capability, or preferences favor certain therapeutic approaches. Such preferences also need to be considered in therapy decisions because provider preferences will affect outcomes. These preferences Table 3. Evidence of the Validity of Bitewing Diagnosis of Proximal Caries in Primary Teeth

<table>
<thead>
<tr>
<th>Year</th>
<th>Author and country</th>
<th>N</th>
<th>Age</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1992</td>
<td>Pitts and Rimmer25 Scotland</td>
<td>756 proximal surfaces with 380 lesions</td>
<td>5-15 y</td>
<td>Lesions clinically cavitated: 2% of lesions in outer half of enamel 3% of lesions in inner half of enamel 28% of lesions in outer half of dentin 96% of lesions in inner half of dentin (50% of any dentin lesions)</td>
</tr>
<tr>
<td>1996</td>
<td>De Araujo et al26 Brazil</td>
<td>20 pts with 72 lesions</td>
<td>3-10 y</td>
<td>Lesions clinically cavitated: 6% of lesions in outer half of enamel (only 3 lesions were radiographically in inner enamel) 84% of lesions in dentin</td>
</tr>
<tr>
<td>1996</td>
<td>Nielsen et al27 Denmark</td>
<td>72 proximal surfaces with 43 lesions</td>
<td>NR</td>
<td>Lesions clinically cavitated: 11% of lesions in outer half of enamel 14% of lesions in inner half of enamel 63% of lesions in dentin</td>
</tr>
</tbody>
</table>
Restorative therapy

Currently, the practice of dentistry primarily utilizes a surgical model of care. Restoration of teeth due to caries still occupies substantial curriculum in dental schools and clinical time in dental practices. However, dental care should be based on preventive services and supplemented by restorative therapy only when indicated. Restorative therapy is a non-reversible procedure that makes a tooth susceptible to fracture and additional decay.53 This is particularly an issue in children, as longevity of restorations is less in the primary dentition than in the permanent dentition and reduced in younger than in older children.54 However, restorative therapy is necessary to eliminate cavitations when dental plaque removal from the tooth is difficult, when there is a high level of caries not reversed by preventive therapies, or when monitored white spots and small lesions show progression to cavitation. Additionally, restorations of teeth are essential where there is need to restore tooth integrity to prevent space loss or disease progression into the dental pulp.

Children at low risk may not need any restorative therapy. Children at moderate risk may require restoration of progressing and cavitated lesions, while white spot and enamel proximal lesions should be treated by preventive techniques and monitored for lesion progression. Children that remain at high risk, however, may require earlier restorative intervention of enamel proximal lesions as well as intervention of progressing and cavitated lesions to minimize continual caries development. In such high-risk cases, more aggressive treatment of primary teeth with stainless steel crown restorations may be better over time than multisurface intracoronal restorations.55,56

Summary

The scientific basis for caries diagnosis, risk assessment and preventive and restorative therapy for children requires further development and continued validation. Most needed are longitudinal studies examining the integration of risk assessment with preventive and restorative therapies. Nevertheless, sufficient evidence exists to allow practitioners to transcend traditional surgical management of dental caries. Current information on the dynamic nature of the carious process and risk assessment allows increased emphasis on patient-specific approaches that include disease monitoring and prevention as well as restorative therapies.

Recommendations

The dental literature supports:

1. The goal of caries risk assessment is to deliver patient-specific diagnostic, preventive and restorative services based on an individual patient’s needs.
2. The following caries risk factors need to be considered: present and past caries activity; socioeconomic status; sealant status; mutans streptococci levels; fluoride exposure; sugar consumption; special needs; and parent/sibling caries activity.
3. Dental caries management includes individualized prevention and restorative therapy.
Acknowledgments

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References

32. Thylstrup A, Bille J, Qvist V. Radiographic and observed tissue changes in approximal carious lesions at...


