Great Starts HERE

Medical Management of Caries

Overview of the Literature

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Personalized Caries Management

If evidence is limited, what can be safely incorporated into practice? and how?

Preventive + Non-Surgical Intervention (Medical Model)

Detection
Diagnosis
Risk Assessment

Assessment

Restorative (Surgical) Intervention

Goal: Focus on health and preserve tooth structure

• Risk-Based
• Patient-Centered
• Focus on prevention and remineralization
• MI
• Best Evidence
• Targeted health care is paramount

• True with the increase in caries prevalence among 2-5 years of olds, and the marked disparity in dental caries distribution and access to care
  • 33% have caries experience (increase by age)
  • Millions $ for care in ER
  • Billions $ annually to treat dental caries

• Healthcare system has limited capacity-access to dental services for young children

• Pediatricians/family physicians have been called to assess oral health, help establish a dental home by age 1, and begin preventive interventions (Chou et al., Pediatrics 2013)
Child, Family, and Community Influences on Oral Health

Patient Education (or other means-policy, etc)

- Oral hygiene (fluoride) and diet are important components of caries management (common risk factors-diabetes, obesity, etc.)

- Evidence for patient-centered approaches (e.g., motivational interviewing) vs. traditional professional-directed counseling is building

- A recent systematic review of RCT showed varied success of MI in improving oral health. There is still a need for further studies with methodological rigor to better understand the roles of MI in dental practice (Gao et al., 2013)
Strategies with the strongest, consistent, highest quality evidence now-a-days are:

**Fluoride**  
**Sealants**

- Use other strategies to **supplement** well known interventions, rather than substituting them
Fluorides

Justifies the search for strategies to reduce the amount and virulence of microflora and could work synergistically with fluoride

<table>
<thead>
<tr>
<th>TFT Type</th>
<th>Prevented Fraction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Varnish (3)</td>
<td>40%</td>
</tr>
<tr>
<td>Gel (13)</td>
<td>21%</td>
</tr>
<tr>
<td>Rinse (30)</td>
<td>26%</td>
</tr>
<tr>
<td>Toothpaste (70)</td>
<td>24%</td>
</tr>
<tr>
<td>Overall (116)</td>
<td>24%</td>
</tr>
</tbody>
</table>

The Cochrane Database of Systematic Reviews, 2003, 2006, 2998

Marinho et al., 2013
Fluoride Varnishes
Sealants

Strong evidence for effectiveness for prevention (sound teeth) and arrest of caries lesions

- PF on sound teeth: 60-70%
- Caries reductions ranged from 86% at 12 months to 57% at 48-54 months
- Positive strong effect across included studies
  [Llodra, 1993; Rozier, 2001; Task Force on Community Preventive Services (2002); Ahovuo-Saloranta et al., 2004, 2008]

- PF on non-cavitated lesions: 71% (Griffin et al., 2008)
At 60 months for 91 patients with at least 48 months follow-up, major failures (irreversible pulpitis, loss of vitality, abscess or unrestorable tooth) were:

- 3 (3%) for the Hall Technique (treatment arm)
- 15 (16.5%) for the usual treatment (control) \((p < 0.01)\)

“…provides strong justification for additional RCTs...” (Fontana et al., 2012)
Strategies to Modify Oral Microbiota

- Active or passive immunization
- Replacement therapy
- Smart Bombs
- Ozone Treatment (Rickard et al., 2004)
- Povidine Iodine (Lopez et al., 2002; Zhan et al., 2006; Xu et al., 2009; Simratvir et al., 2010)
- Chlorhexidine (Rethman et al., 2011)
- Xylitol, Probiotics, Silver…
Consumption of sucrose-free chewing gum for 10–20 minutes after meals reduces the incidence of caries in high caries risk patients (6-8gr/day ; divide this in 2-3 times/day) (Rethman et al., 2011) CHOCKING HAZARD FOR YOUNG CHILDREN

There is also evidence for lozenges and hard candy (6-8g/day) (Antonio et al., 2011) CHOCKING HAZARD FOR YOUNG CHILDREN

Saliva or xylitol per se (Fontana and Gonzlaez-Cabezas,2012)

However, evidence for other forms of xylitol products (syrup, wipes, toothpastes, etc.) is still very limited
Influence of maternal xylitol consumption on mother-child transmission of mutans streptococci: 6-year follow-up (Soderling et al., 2001)

Xylitol pediatric topical oral syrup to prevent dental caries: a double-blind randomized clinical trial of efficacy (Milgrom et al., 2009)

Effects of xylitol wipes on cariogenic bacteria and caries in young children (Zhan et al., 2012)

Topical xylitol administration by parents for the promotion of oral health in infants: a caries prevention experiment at a Finnish Public Health Centre (Makinen et al., 2013)
Probiotics

Table 2. Controlled Clinical Trials with Caries as Endpoint

<table>
<thead>
<tr>
<th>First Author, yr</th>
<th>Design</th>
<th>n; Age (yrs) (dropout rate)</th>
<th>Vehicle, Time</th>
<th>Strain</th>
<th>Caries Test/Control</th>
<th>PF (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Näse, 2002</td>
<td>RCT</td>
<td>594; 1-6 (24%)</td>
<td>milk, 7m</td>
<td><em>L. rhamnosus</em> GG</td>
<td>15%/19%</td>
<td>21%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>164; 3-4</td>
<td>milk, 7m</td>
<td><em>L. rhamnosus</em> GG</td>
<td>10%/23%</td>
<td></td>
</tr>
<tr>
<td>Stecksen-Blicks, 2009</td>
<td>CRCT</td>
<td>174; 1-5 (25%)</td>
<td>milk, 21m</td>
<td><em>L. rhamnosus</em> LB21+2.5 ppm F</td>
<td>Δdmfs 0.4/1.6</td>
<td>75%</td>
</tr>
<tr>
<td>Petersson, 2011</td>
<td>RCT</td>
<td>160; 58-84 (38%)</td>
<td>milk, 15m</td>
<td><em>L. rhamnosus</em> LB21+5 ppm F</td>
<td>rev 68%/24%*</td>
<td>65%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td><em>L. rhamnosus</em> LB21</td>
<td>rev 54%/24%*</td>
<td>55%</td>
</tr>
</tbody>
</table>

*aRCT = randomized controlled trial; CRCT = cluster randomized controlled trial.

*b m = mos.

cPF = prevented fraction.

dCaries prevalence, %.

*eCaries reversals, %.

Twetman and Keller, 2012

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**Fig. 3.** Mean number of days with otitis media and on antibiotic therapy in children who participated for 21 months in the intervention and control units. *Statistically significantly less days with otitis media in the intervention units (p < 0.05).**

Caries Res 2009; 43:374–381
Silver Compounds

<table>
<thead>
<tr>
<th>Period</th>
<th>Advances</th>
</tr>
</thead>
<tbody>
<tr>
<td>Up to 1900</td>
<td>AgNO₃ used in caries management</td>
</tr>
<tr>
<td>1917</td>
<td>Howe’s solution (AgNH₃NO₃) invented and used up to 1950s</td>
</tr>
<tr>
<td>1970s–1990s</td>
<td>AgF used alone and combined with SnF₂ in clinical studies in Western Australia</td>
</tr>
<tr>
<td>1970s</td>
<td>Development of SDF in Japan supported by Central Pharmaceutical Council of the Ministry of Health and Welfare</td>
</tr>
<tr>
<td>1990s</td>
<td>SDF was recommended for young children in Brazil</td>
</tr>
<tr>
<td>2000s</td>
<td>Randomized controlled clinical trials on SDF and other preventive treatments</td>
</tr>
<tr>
<td>2000s</td>
<td>Addition of silver particle into restorative materials</td>
</tr>
</tbody>
</table>

Rosenblatt et al., 2009

Peng et al., 2012
Table 2 – Clinical evidence for silver compounds utilization (1905–2011).

<table>
<thead>
<tr>
<th>Author</th>
<th>Type of study, duration, and intended effect on caries</th>
<th>Treatment protocol and results</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prime J 1935 (^{13})</td>
<td>Case report Caries arresting Cohort (5 y)</td>
<td>AgNH$_3$NO$_3$ single application: 2 permanent upper incisors</td>
<td>AgNH$_3$NO$_3$ was useful for arresting caries</td>
</tr>
<tr>
<td>Klein H 1942 (^{14})</td>
<td>Caries prevention</td>
<td>Control (no treatment): 55 teeth 79% of treated and 77% of the untreated permanent molars became carious at recall</td>
<td>AgNH$_3$NO$_3$ was not effective in preventing caries</td>
</tr>
<tr>
<td>Seltzer S 1942 (^{8})</td>
<td>Cohort (1 y) Cavity sterilising and restoration</td>
<td>AgNH$_3$NO$_3$ single application: 9 teeth Control (no treatment): 12 teeth</td>
<td>AgNH$_3$NO$_3$ had limited sterilising effect</td>
</tr>
<tr>
<td>Schultz-Haudts S 1956 (^{15})</td>
<td>Cohort (1 y) Caries arresting</td>
<td>Excavation + AgNH$_3$NO$_3$ single application: 136 class II carious lesions The “caries arresting rate” was 82% and 17% respectively</td>
<td>“AgNO$_3$ has caries arresting effects in primary teeth.”</td>
</tr>
</tbody>
</table>

AgNH$_3$NO$_3$: Howe’s ammoniacal silver nitrate solution

Ca$_{10}$(PO$_4$)$_6$(OH)$_2$ + AgNO$_3$ → Ca(NO$_3$)$_2$ + Ag$_3$PO$_4$ + Ag$_2$O + H$_2$O

Peng et al., 2012
- 40% AgF (+ SnF₂ as a reducing agent; more soluble in water than other silver halides; pH=11)
- Arrest of deep caries lesions in primary teeth of young children in children difficult to manage

<table>
<thead>
<tr>
<th>Study</th>
<th>Cohort (y)</th>
<th>Treatment Details</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Craig GG 1981</td>
<td>2 y</td>
<td>40 wt% AgF followed by 10 wt% SnF₂ single application: 281 early lesions</td>
<td>“AgF followed by SnF₂ slowed the growth of existing lesions in primary teeth.”</td>
</tr>
<tr>
<td></td>
<td></td>
<td>74% of the proximal and 90% of the occlusal surface remained unchanged</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Cohort (6 y)</td>
<td>No effects on the permanent successors</td>
</tr>
<tr>
<td></td>
<td></td>
<td>40 wt% AgF followed by 10 wt% SnF₂ single application: 43 cavitated teeth</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Pulpotomy: 31 teeth with deep caries indicated for pulpotomy</td>
<td></td>
</tr>
<tr>
<td>Green E 1989</td>
<td>1.5 y</td>
<td>40 wt% AgF followed by 10 wt% SnF₂ single application: 1300 teeth</td>
<td>The combined treatment had a significantly lower incidence of caries for primary teeth compared with SnF₂ alone</td>
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<td></td>
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<td>10 wt% SnF₂ alone single application: 1563 teeth</td>
<td></td>
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<td></td>
<td></td>
<td>The proportion of teeth with caries after 18 month was 2.8% and 11.6% respectively, although no dmft was recorded at baseline</td>
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</tbody>
</table>

Peng et al., 2012
38% (44,800 ppm F) Silve Diammine Fluoride-SDF solution
(e.g., Fluoroplat, Safluoride, Saforide; ammonia and AgF combined to form a diammine silver ion complex Ag(NH$_3$)$_2^+$; claimed to be more stable than AgF, and can be kept at constant concentration for a longer time; pH=8-9)

- Antibacterial
- When in contact with dentin: Ag$_3$PO$_4$ (weakly soluble; turns black with sunlight or reducing agents) = Black, hard layer
- Metallic taste; transient gingival and mucosal irritation
  (Llodra et al., 2005)
- To counter stain: KI (no clinical trial; in vitro suggests same effect on biofilm, Knight et al., 2005)
- Low cost, easy to use
- Arrest caries in primary teeth of preschool children
- 8 clinical studies 1905-2011 (Peng et al., 2012)

\[
\text{Ca}_{10}(\text{PO}_4)_6(\text{OH})_2 + \text{Ag}(\text{NH}_3)_2\text{F} \rightarrow \text{CaF}_2 + \text{Ag}_3\text{PO}_4 \\
+ \quad \text{NH}_4\text{OH} \\
\text{Alkaline environment}
\]
Used on cav. lesions (Rosenblatt et al., 2009):

- No need for prior excavation (1x/year: PF ~ 70-84%; better than FV ~44-56%) (Chu et al., 2002)

- 2x/year better than nothing in prevention and arrest (primary teeth PF 79% and 64% in permanent teeth; Llodra et al., 2005)

- 38% more effective than 12% SDF (Yee et al., 2009)

- Equally effective to FV and sealant on sound/non-cavitated 1st perm molars (Liu et al., 2012)

- SDF-KI (washed) creates bond strengths no different than conditioned samples prior to GI application (In vitro; Knight et al., 2006) and no pulp pathology when used SDS close to pulp (Gotjamanos, 1996)
Conclusions

• Continue building strength of EBD approaches

• Continue using same outdated approach? Or change to use of best EBD approach for personalized risk-based care

• Best practices?

Questions:
• Use for prevention vs. arrest
• Stratified by risk?
• Hall crown/ART vs. SDF/AgNO₃?
• If we are going to restore, do we need anything prior (pulp capping)?
• Outcomes on variations in techniques (concentrations, need reducing agents, etc.)
• Will lesions remain arrested? (frequency)

Besinis et al., 2013