changed many people or simply from one patient who heard that message, was cared for well, and told another to create an ensuing snowball of individual doctor-patient relationships. Lobbying carries with it that same investment of time and treasure with no guarantee of return nor easily parsed cause and effect.

I hope my resident got the message that we judge the differences lobbying makes not by the exuberance of the day on the Hill, but by our Academy's body of work. Title VII, Head Start, CMS Medicaid Manual, Congressional testimony, representation on government panels, access to legislative offices, and the use of Academy guidelines in policy matters are just some examples of the fruits of advocacy's labor.

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Letters to the Editor

An Alternative Explanation for the Prevalence and Distribution of Enamel Defects

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I read with great interest the recently published article, "Enamel Defects in the Complete Primary Dentition of Children Born at Term and Preterm."[1] I would like to comment on the authors explanation of why defects of dental enamel (DDE) appeared more frequently on the facial surfaces of the anterior teeth, even in the term children who were not subjected to tracheal intubation. They propose that this location suggests "the local effects of local traumas....". However, they might have considered the following explanation proposed in the discussion section of an article authored by myself and others, "Macroscopic enamel defects of primary anterior teeth—types, prevalence, and distribution."[2]

"The thickness of enamel might explain our results that developmental defects were seen most commonly on maxillary teeth, facial surfaces, and the middle third of the crown. The primary maxillary anterior teeth have thicker enamel then their mandibular counterparts. Primary anterior teeth generally have thicker enamel on their facial surfaces and in the middle third of the crown. In addition, the incisal/cuspal third of exfoliated primary anterior teeth have the thinnest enamel and usually are worn significantly, precluding observation of enamel defects in many instances. Kraus and Jordan (1965) explain that the varying thicknesses of enamel in the same tooth may be due to "different rates of enamel apposition in different parts of the same tooth.., regardless of whether or not the ameloblastic life spans differ, or whether or not calcification ceases simultaneously throughout the crown..." This postulate is supported by our observations that the thickest surfaces and locations exhibited the highest prevalence of [developmental enamel defects] DED. If the secretion and maturation of enamel occurs most rapidly on these thicker teeth, surfaces, and locations, then the greater metabolic demand of the ameloblasts in these areas might make them especially vulnerable to any insult. A severe metabolic disturbance might affect all teeth and surfaces, while a milder perturbation might preferentially affect the most metabolically active ameloblasts or the most rapidly maturing enamel."

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