Panel Discussion

Drs. Axelsson, Bawden, Hassell, Lainson, Poulsen, Ranney, Steinberg, and Wei

Questions:

The presentations suggested that mechanical plaque removal in conjunction with frequent professional prophylaxis can reduce or eliminate caries and periodontal disease. Unfortunately, this program requires a very dedicated, fanatical type of person and wealth, or a state-supported dental program:

1. How realistic is your program?
2. In the U.S., without government programs, how can we justify frequent professional prophylaxis and be reimbursed?
3. In the pediatric population with poor refined motor control, how effective have you been in teaching them interproximal cleaning?

Answer: Dr. Axelsson.

Based on the many studies done in Sweden, it can be concluded that clean teeth will not decay. An effective plaque control program is carried out in the schools by dental hygienists and nurses. Such programs begin prenatally with pregnant women so that their children will be free of dental disease. Such programs extend to the infants and toddlers. The statistics clearly show that it is a cost-effective program in Sweden and the total time and effort spent in such a plaque program is much less than the time spent in restoring teeth.

Question:

There is a significant difference in the type of microflora and periodontitis. If one follows a population from 10 to 15 years of age, what specific changes in microflora would indicate signs of irreversible changes of periodontal attachment loss?

Answer: Dr. Ranney

I don’t believe that the microflora have been studied sufficiently to overcome the problems of the variability among samples so that we can decide which are the critical or key organisms to look for as a sign of lesion progression. Considerably more work needs to be done before that question can be answered.

Question:

Animal studies have shown that hormonal changes interfere with the host response to dental plaque. What do you suggest may be the best way to examine such hormonal changes in puberty?

Answers: Dr. Hassell

There is very little doubt that hormonal changes are a problem primarily of young people and young adults but tend to disappear after the ages of 29 to 30. There has been a good deal of work in experimental animals indicating that phenytoin has inhibitory effects on various hormonal actions. What the cause and affect might be in relation to gingival response to plaque in development of gingival overgrowth is simply not known.

Dr. Steinberg

I concur with Dr. Hassell, because I don’t think there is any single direct etiological cause as far as the dilantin hyperplasia is concerned. It has a multifactorial etiology and it would be dangerous to pin it down to just hormones, which seem to alter almost any type of inflammatory response.

Question:

What is the best clinical management of pregnancy gingivitis?

Answer: Dr. Lainson

I think that has been well established by several studies, and in particular the studies of Dr. W. Cohen. He and his coworkers showed that if you meticulously control the local factors you either prevent, or can reverse, pregnancy gingivitis. We manage it primarily through meticulous oral hygiene, through preventive maintenance procedures, and multiple professional visits prior to the delivery of the child.

Question:

Dr. Vanarsdall, is orthodontic treatment contraindicated in patients with dilantin hyperplasia?

Answers: Dr. Vanarsdall

Absolutely not. Most of the patients that I see with dilantin hyperplasia need it because the tissue overgrowth tends to move the teeth around. The manage-
ment technique in subjects with dilantin hyperplasia is to “get in” and “get out” of the case orthodontically as quickly as possible. I prefer to have the tissue removed and have the appliances on the patient within 10 days. By orthodontically moving the teeth during the healing period, they will level in line very rapidly. It is important to have the patients on plaque control with a rigid program of weekly or bi-weekly appointments with a dental hygienist to keep the inflammation down. If I’m going to treat them, I take the responsibility of keeping plaque down to a level I can control.

Dr. Hassell

I just want to comment about whether tissue moves teeth around or whether the tissue simply fills up the spaces in between. To my knowledge there hasn’t been any longitudinal work done in this area at all. There is a good deal of clinical impression, and I concur with the clinical impression that when a child comes in and he’s got teeth that look like a broken picket fence, and all of those spaces between the teeth are filled up with fibrous tissue, it is easy to say that the tissue pushed them around. But there is no substantive data to document that statement and back it up.

Dr. Vanarsdall

I think that in a dilantin hyperplasia patient, if orthodontic treatment is required they should have it done. It is important that they should be able to look more like a normal individual, so anything that will enhance their appearance should be done. You can really minimize the amount of overgrowth by meticulous oral hygiene.

Question:

What type of bristles on an electric toothbrush do you recommend? Please comment on the utilization of a hard toothbrush with vigorous brushing for the control of gingival overgrowth.

Answer: Dr. Steinberg

We like to use a softer brush so that we can get into the gingival sulcus and pseudopockets as much as possible. I do prescribe an electric toothbrush for many of the dilantin patients because I feel that it is important to give gingival massage and insure meticulous plaque removal to aid these patients.

Question:

Dr. Bawden has suggested that there is an increase in the prevalence of periodontal disease in his population, especially in the younger patient, and that the pedodontist and the educational programs should take a more aggressive role in preventing the disease and treating it. What measure would you suggest be taken?

Answer: Dr. Bawden

I’m not sure. I think the problem in our school is that in spite of the fact that we talk about plaque control and we talk about preventive dentistry and so on, those are not the attitudes that really dominate the curriculum. Crown and bridge and operative dentistry are departments which dominate the curriculum. I think that students, by and large, have the background they need to deal with the problems, but they just don’t think that is the most important part of dentistry. I think you need to manipulate your curriculum somehow to try to re-orient the students to the problem.

Now, as I said in my presentation, not withstanding Dr. Axelsson’s excellent work, it is very difficult to get people to keep their mouths clean. I spent a year in his country and I can tell you that his country and my state are two different worlds. Sweden is a very homogeneous population, genetically and culturally, and is highly literate. Their lifestyle is highly disciplined and they are a health-oriented population. They have expensive public health programs and those kind of things work very well for them. I’m not so sure that they’ll work as well for us. I would like to have him visit my state and observe the heterogeneous population, their literacy, health knowledge, self-discipline, and the number of other problems that confront us there.

There is no question that meticulous oral hygiene will accomplish many things, and I think in his society the approach is extremely realistic. I’m not so sure it is very realistic where I live.

Question:

Would the panel discuss the use of the sulcus bleeding index for the selection of a population to study the initiation of gingivitis? Is the use of the periodontal probe in the gingival sulcus for gingival bleeding wise, or would it be better to use extra-gingival sulcus stimulation?

Answers: Dr. Poulsen

This method was developed by Löe, who put his patients on a no-oral-hygiene regime and observed the time it took for them to reach a state of inflammation, including whether there was gingival bleeding, and he used different selected gingival papillary areas. As far as using the periodontal probe, we do not try to put the probe too deeply into the gingival sulcus because we are so frequently working with children, so we tend to have the probe at an angle to the tooth surface and then lightly place the probe under the gingival margin.

Dr. Hassell

We recently performed a large epidemiological study of the dilantin lesion in an institutionalized population in the Boston area. In attempting to quantify the degree of gingival inflammation, we found
that none of the standard indices work on people that have enlarged gums. We wanted to use a bleeding index, which was also not particularly good, because it takes a lot more time for the blood to go from the bottom of the sulcus to the margin in a 10 mm pseudopocket, than in a 2 mm sulcus. So instead of using a periodontal probe, we used large endodontic paper points and found that it was very sensitive. By inserting the dry sterile paper points into the depth of the pseudopocket and simply wiping it back and forth, very often we had blood on the tip of the point which didn’t appear at the gingival margin for as long as three or four minutes. It turned out to be a very sensitive index of early inflammation in these patients.

Question:
Papillon-Lefevre syndrome has been mentioned several times in the conference — have there been any advances in the treatment of this disease, which usually results in the loss of all teeth by age 16?

Answers: Dr. Tinanoff

We have recently treated a difficult case of Papillon-Lefevre syndrome which I would like to share with you. This girl had a cellular-immune deficiency problem. Her chief complaint was that her teeth would fall out with roots intact, and her clinical appearance was not unlike juvenile periodontitis. We tried mechanical treatments on her at first with curettage and scaling. Then we tried a course of tetracycline therapy followed by erythromycin therapy. After each course of therapy she still had gram-negative anerobic rods. We did not convert her microbiology to anything different from that, and we were using that as an index of treatment. Then we decided that if we eliminated all her pockets we could possibly alter the environment in her mouth and lose the anerobic gram-negative rods that she had.

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cellular-immune response in combination with a bacterial response, and that we altered the microbiology by our treatment.

Question:
How do you account for studies that do not show a linear correlation between the OHI, and indices such as GI and PI?

Answer: Dr. Ranney

I think there is a difference between children and adults in the way the gingival tissues respond, but we do not know what that difference is. Children don’t seem to follow the relationship of the plaque index to gingival index that adults do during the experimental gingivitis. I don’t know what the answer is, but I would be fascinated to learn what the difference is. I think that it would be a good research project.

Question:
Many clinicians do not recommend flossing for children in the primary dentition. They feel that the anatomical state of the teeth do not predispose to interproximal plaque accumulation. What are your comments? At what age do you start flossing children’s teeth? You seem to recommend dental tape rather than dental floss, why?

Answers: Dr. Axelsson

In Sweden, dental tape is much more accepted than dental floss, it seems that Swedish children prefer dental tape. However, we do not have success with children in the 8 to 14 year age group using floss. We try to train the parents to carry out flossing between the second primary molar and the first permanent molar just to prevent carious lesions from developing on the mesial surface of the first molar. We try to introduce floss from 10 to 13 years of age. By age 13, we stress flossing. We try to encourage self-diagnosis of gingivitis and interproximal caries and encourage effective plaque control.

Dr. Vanarsdall

Everyone here that sees a lot of children says the place that bleeds primarily is distal to the second primary molar moving toward the first permanent molar. I have parents floss all four quadrants to show the susceptibility of that area. It was very interesting listening to everyone say that the angular crest is a primary site for periodontal disease, and the angular crest is of a normal anatomy in the primary and mixed dentitions due to the occlusal-gingival dimensions of the teeth. You will always find more pocket depth in the average child distal to the second primary molar and mesial to the first permanent molar, and I have great cooperation from parents in flossing their children’s teeth.
**Question:**
Dr. Poulsen, is it recommended to choose the following criteria for a study: 1) gingival inflammation; 2) loss of attachment; or 3) gingival index based on bleeding? What procedure do you follow for the index of bleeding — bleeding on probing, or bleeding on drying the teeth with the air syringe? Would you measure your loss of attachment with a perio probe or with something thinner and more flexible, like a calibrated soft plastic device? Do you take 2, 4, or 6 measurements per tooth?

**Answer: Dr. Poulsen**

The first question was about bleeding: whether this was bleeding on probing, and bleeding using a periodontal probe which is used to stimulate the gingivae in children. The measurement of the loss of attachment is done with a thin periodontal probe, the Michigan probe, which is suitable for measuring a loss of attachment.

**Question:**
Dr. Steinberg, do you use a 0.5% stannous fluoride daily? Is there any discomfort or complication of staining, and is it a solution or a gel preparation?

**Answer: Dr. Steinberg**

We use a commerical gel preparation. We usually use a 1% solution and water it down. We didn’t notice any staining on the teeth during the time that we used it. At this concentration we really had not run into any problem — the only thing that really ever worried us was the actual amount of fluoride the patient would be swallowing. With the 1% stannous fluoride solution, you are getting, I think, something like about 10 mg of fluoride in each ml, and we tried to hold off using more than 1 cc applied to the gingival tissue and teeth. So even if the patient should swallow all of the material, that would only be about 2.5 mg. The child can tolerate that amount very well, because the ages of our population was from about 6 to 14. There is no gingival irritation either.

**Question:**
What current or future treatment modalities will the clinician have to control the host-immune response?

**Answer: Dr. Ranney**

There are, of course, some drugs which have a documented effect on the immune response but I think it’s going too far out on a limb and beyond the realm of my expertise to get into what specific agents might be used to correct the specific immune defects that may exist in a given person or population that we know about right now.

But, let me give a different type of example of the utility of that type of knowledge. Dr. Vanardsall is an excellent clinician who looks very carefully at the tissues in the cases he is going to treat orthodontically to make educated guesses as to which patients he's got to be most careful of. I think that it is quite possible that something along the lines of one or two of the perceived functional abnormalities that I talked about can be developed to the point where you can identify risk groups in that fashion, for example.

I would be very interested in knowing whether that was in a stage that had chemotactic defect or some other type of functional type of abnormality of the defense systems that could be tested. Now, I am not suggesting that will be coming down the road in the next year, because there is an awful lot that has to be learned before that is possible, but I think that will come before modulation of the immune system is a practicality. If you want to get away from antigen-specific or polyclonal-induced responsiveness of the lymphocyte series however, and look at the PMN chemotactic defect, I think that there is a possibility of earlier practical application for therapeutic manipulation. Several researchers at our university have made considerable progress in the last year in identifying a metabolic lesion in these PMN from the patients that would be in the cluster 3 that I described, in the most depressed group. Now, if that holds up, and if that metabolic lesion can be manipulated, then we will have a means with which we can test whether or not that PMN defect is, in fact, critical to the progress of the periodontal disease in those individuals. Then the next step, if that is true, is treating that defect.

Now we can serendipitously manipulate the defect in vitro without quite fully understanding all the aspects of the metabolic lesion. What we can manipulate in vitro at the moment isn’t a rationale for a clinical trial test in a human population. But I think the principle is there, and that particular type of functional-qualitative abnormality of PMN leucocytes may be manipulable. And if it in fact does have something to do with the disease, then you can get a handle on the disease. But we are still several steps away.