Pain and Cooperation in Orthodontic Treatment

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Orthodontists generally assume that some discomfort is the norm for their patients and attribute patients' lack of cooperation to attitudinal factors. I have used a personality inventory test to assess patients' self-image, comfort with authority, and presence or absence of neurotic traits.[1-3] Although these tests are useful, many patients who should have been excellent cooperators according to their test scores turned out to be very poor patients. In trying to find a reason for these exceptions, I noticed that these patients brushed their teeth lightly and said the bristles were uncomfortable against their gums. This discovery led me to study pain tolerance and to conclude that attitude may have little to do with patient cooperation during treatment. Evidence suggests that a patient's pain threshold is closely related to inability to cooperate,[4,5] and that prostaglandins play an important role in the production of inflammatory pain.

Role of Prostaglandins

It has been suggested that stasis of capillary blood flow, which contributes to post-adjustment discomfort, could be alleviated by having the patient chew on a plastic wafer for 20 minutes following an orthodontic adjustment.[6] Although we have only empirical evidence that this treatment is effective, many patients have enthusiastically endorsed the plastic bite wafer for reducing post-adjustment discomfort. When this report was published (1972), little was known about the chemistry of inflammation. Since then, a great deal of information has come to light regarding the effect of prostaglandins and kinins on the inflammatory process.

Prostaglandins, which occur in almost all human tissues, were first found in high concentrations in the prostate glands of sheep, and this accounts for their name. They are biologically unusual because of their ubiquity, their broad spectrum of physiological action, their high potency, their variety of form, and their short life span.[9-11] They are not stored and must continually be biosynthesized. The site for their manufacture is thought to be the cell membrane. Functions of prostaglandins include opening airways by relaxing smooth muscles, regulating blood pressure by increasing urinary output and excretion of sodium ions, and vasoconstriction and vasodilation.[12-25]

Apparently, the release of prostaglandins greatly enhances the transmission of painful stimuli, because prostaglandins biochemically mediate the amount of cyclic AMP (adenosine monophosphate), which modulates norepinephrine at the neural synapse.[26] This localized effect of prostaglandins probably explains why some analgesic drugs, such as aspirin, indomethacin, phenylbutazone and extracts of aloe, are effective in combating prostaglandin-induced pain.

Although antihistamines and steroids are effective in reducing inflammation, they are not effective in reducing the accompanying pain, and often produce undesirable systemic side effects. Some well-known, over-the-counter drugs such as aspirin and aloe extracts have shown remarkable potency in combating prostaglandin biosynthesis. All of the non-steroidal anti-inflammatory agents (NSAIAs), including aspirin, apparently inhibit prostaglandin synthesis via acetylation and inactivation of the enzyme cyclo-oxygenase[28] (Fig. 1). Aspirin is active only in preventing prostaglandin production; once prostaglandins are formed, aspirin is incapable of breaking them down. Some of the newer NSAIAs are apparently capable of reversing prostaglandin synthesis.

The unique ability of prostaglandins to cause hyperalgesia may account for their major role in pain production in inflamed tissue, and may cause ordinary, non-painful stimuli to become painful.[29] Although there is evidence that intrapulpal pain may not be a feature of prostaglandin biosynthesis,[30] there is no doubt about its role in chronic periodontal inflammation.[24]

Orthodontic treatment provides the type of chronic periodontal insult that encourages a continual and increased production of prostaglandins. This may account for a number of problems such as poor oral hygiene, increased gingival sensitivity, chronic gingivitis, and inability to cooperate. Noncooperative orthodontic patients do not necessarily need an improved attitude as much as they need a reduction in synthesized inflammatory products and the accompanying pain.

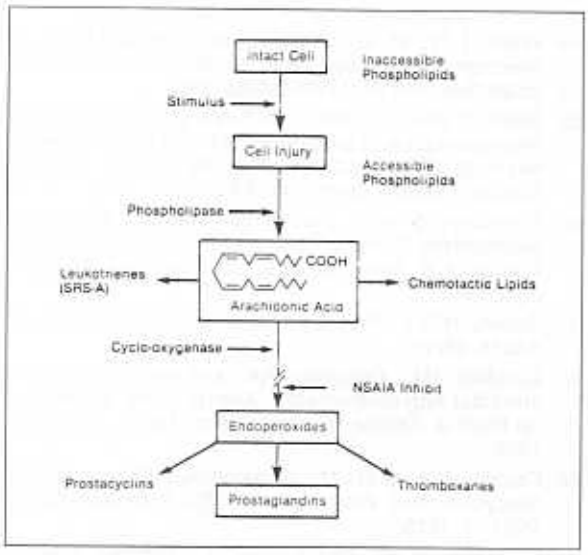

Fig. 1 Inhibition of prostaglandin production.

Effect of Analgesic Chewing Gum

To determine if the mechanical stimulation of the periodontium in combination with a weak analgesic—aspirin—might be able to decrease post-adjustment discomfort, 93 fully banded patients who had been in treatment several months were selected for the study. Because most post-adjustment discomfort is reported after archwire changes, participation was limited to those who had previously experienced archwire changes, so they would be able to compare the degree of post-adjustment discomfort.

Each participant was asked to chew two pieces of Aspergum for 20 to 30 minutes immediately following archwire changes, and to report the following day whether there had been more or less discomfort than usual with an archwire change or no difference in the level of discomfort.

Fifty-eight of the participants (63%) said they had experienced less discomfort after chewing Aspergum, 24 of the patients (25%) said the Aspergum had had no effect on their post-adjustment discomfort, and only 11 of the patients (12%) said their discomfort was increased after chewing the gum.

Discussion

Human pain perception can be so varied[31-37] that we would not expect an analgesic chewing gum to be a panacea for reducing orthodontic post-adjustment discomfort. Also, a study such as this, based on subjective findings, is likely to include some placebo effect. But any product that can reduce orthodontic pain in 63% of the patients studied should be considered seriously as a therapeutic adjunct. It is also easy to envision many new oral therapeutic products including toothpastes, mouthrinses, and ointments.

Aspirin's ability to prevent prostaglandin production may explain the success we had with our patients who chewed Aspergum immediately after their archwire changes. The aspirin may have prevented prostaglandin formation while the chewing kept the periodontal capillaries open so that nutritive and metabolic products could continue to move throughout the periodontium.

The results of our study suggest future investigations to determine if chewing on a plastic bite wafer could provide the same level of relief as Aspergum, and if a placebo chewing gum would evoke a statistically different response than the medicated gum.

Although Aspergum reduced post-adjustment discomfort in many patients in our study, it would be unwise to prescribe it as part of the regular regimen. Chronic ingestion of aspirin has been associated with enamel decalcification.[38] Infrequent use of the gum would probably not harm the enamel, but patients might take orthodontists' endorsement of the gum as a license to use it all the time, and only judicious use is advised. Perhaps a completely safe, effective NSAIA chewing gum will be developed. In the meantime, further research is needed into the interrelationships between patient comfort, oral hygiene, cooperation, and treatment time.

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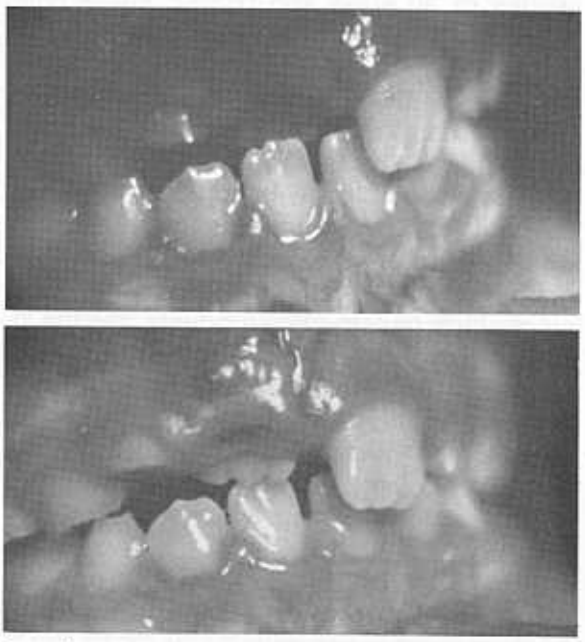
ANTERIOR CROSSBITE CORRECTION

If the horizontal and vertical overlaps are within 2-3mm, a tongue blade can be used to correct an anterior crossbite. The parent is advised in advance that this may be a two-hour appointment. Parent and child are seated in a treatment room and the parent is shown where to place the tongue blade, how much force to apply, and in which direction to apply it. Progress is checked periodically. Typically, the correction will take one to two hours, depending on the extent of the crossbite and the patient-parent cooperation. Once the correction is made, the occlusion will prevent a reversion of the crossbite.



Anterior crossbite correction after two hours of tongue blade pressure.

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