Commentary

This paper addresses a problem which has been a most controversial question in orthodontics for many years. It is well known that any malocclusion is a result of a multitude of factors rather than just one factor. The etiology of open bite tends to be categorized in *growing* patients as:

1. Skeletal pattern — A function of growth and genetic factors. This involves defined characteristics such as short ramus height, vertical maxillary excess, and any number of skeletal factors which are involved in open bite growth pattern.
2. The influence of soft tissue on open bite:
   a. tongue posture
   b. tongue thrust
   c. the influence of lymphoid tissue and its effect on:
      i. resting tongue posture
      ii. mandibular postural position
      iii. airway — and its potential influence on postural mechanisms as well.

In this study, the patient population was divided into two groups; one group which was judged to be actively growing during treatment and retention, and another group representing a nongrowing population. The distinction between these two groups is important in evaluation of the stability of orthodontic open bite correction, since the etiologic factors in open bite tend to be less numerous in the nongrowing group. For example, the authors report that in the group of 23 growing open bite patients that were successfully treated with cribs to positive over bite, 14 or 17.4 percent experienced significant relapse. Eighty-three percent were considered long-term successes. They also correctly point out other studies that show similar rates of bite closure in the mixed dentition with no treatment, a result of growth changes which cloud the single issue of the impact of tongue posture or function on open bite. It is most interesting, then, to analyze the use of crib therapy in the nongrowing group. This group essentially eliminates the factors of skeletal growth and lymphoid soft tissue influences. It is well documented that surgical correction of open bite has greater stability than does orthodontically corrected open bite, but the incidence of relapse is still as much as 21 percent (Denison). In the nongrowing group (n=7) using cribs, none of the subjects experienced relapse. While this is a small sample size, the fact that seven out of seven subjects were stable is significant itself and certainly deserves further inspection.

As in most studies of open bite treatment, this study raises more questions that need to be answered. The etiology of open bite may be very different from the relapse factors involved in treatment of open bite. Studies of open bite relapse often associate posterior tooth eruption with posttreatment relapse. This is a factor which was not analyzed in this study, therefore the question must be asked, what influence did posterior tooth eruption have on the relapse of these case treatments? There also was noted to be no relationship between the length of crib use and stability. This paper hypothesizes that the use of a crib may, in essence, retrain the tongue. If this is so, then, how long does it take for the tongue to learn the lessons it needs to learn? And once it learns a new position, why does it stay there?

Of course, this study analyzes crib treatment and must make extrapolations to tongue function. This has the potential of paralleling the speech therapy experience, that is, speech therapy was thought to be successful in retraining swallowing patterns until studies showed that their success rates were very close to no treatment at all in growing patients. This paper is a significant contribution to the further analysis of one of the more difficult problems in orthodontics. Further analysis of the nongrowing patient population is indicated as well.

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