SOFT TISSUES AND MALOCCLUSION

Sir, I was interested in the article by Sarah Turner, Claire Nattrass and Jonathan Sandy, published in the June issue, and support their conclusion that "the role of the soft tissue in the aetiology of malocclusion seems to be active but unquantifiable". Also that "it is likely that each malocclusion is caused by multiple factors acting synergistically".

The authors provide a balanced review of the literature, but in common with many others who have considered these issues, fail at times to apply sufficient reason to the evidence. For instance they say "there is a trend towards a reduction in jaw size". This is impossible: genes rarely change and jaws can only get progressively smaller within a single population if those with large jaws are being eliminated from the pool. They refer to a tongue as though it were inherited. We should remember that the tongue is almost 90% muscle and that muscle can change in volume by approaching ten times. Clearly large tongues are due to environmental activity. They refer to Ballards "strap-like lower lip" but this can be visibly associated with tongue and lip parafunction, and we know that lip form can at times change dramatically.

They reasonably conclude that "masticatory muscle strength is unlikely to play an important role in determining vertical facial height", but like most of our specialty neglect to mention the long-term open-mouth postures that afflict around 80% of industrialized children. What is likely to happen to the eruptive pattern of these children? Why do the cases with muscular dystrophy that they mention have long faces with the molars in contact and collapsed arches? What evidence (or reason) is there for this to depend on the existing vertical facial form?

It may be true that "increased nasal airflow resistance cannot be a factor in the development of a long face", but short-term nasal obstruction can break a child's natural lip-seal. Some subsequently learn to close their mouth, while some do not, rendering any research that does not allow for this useless.

The soft tissues may be unquantifiable, but every experienced clinician assesses them at every consultation. Where proof is not available we must not close our minds to rational probabilities. It is simple to recognise lip parafunction by the associated hypertrophy, and every patient with enlarged buccinator muscles will have collapsed arches. It is easy to recognise a child's resting posture by watching them talk; some lips hardly ever touch while others contact between most syllables and at rest. Few of the latter will have malocclusion. The authors consider that "soft tissues do not generally play a part in the aetiology of Class III malocclusion". Then why do all such patients push their tongues against the lower incisors and fail to rest it against the palate?

We should avoid misleading statements such as "there is no evidence to show that increased muscular forces might alter jaw and dental arch dimensions" when there is no evidence to show that this is not so. Few would doubt that a tongue-thrust swallow can at times maintain an open bite, but we have not the evidence to conclude that it is an adaptive phenomenon. Tongue between tooth swallows start young and are nearly always associated with malocclusion.

When I trained it was considered that 95% of malocclusion was genetic, now it is thought to be around 50%. But have always insisted that it is over 50% environmental, and one day I am sure this view will be justified. However those sitting on the fence until we have the proof, are likely to get very uncomfortable.

John Mew
Dental Surgeon
Sussex

THE AUTHOR'S REPLY

Sir, we are very grateful for the interest that Mr Mew has shown in this paper. We do not think that we can really comment on some of the issues he has raised. It is interesting to us that instead of moving towards evidence-based practice, Mr Mew is suggesting that if we do not have any evidence then we can move to "rational-probability based practice". This article arose after an essay and tutorial on soft tissue as part of the Bristol Postgraduate Programme.

It may have slipped Mr Mew's memory, but he did speak to the Bristol postgraduates in their last year. The Bristol Orthodontic Postgraduate Programme has always been open to as many views as possible from different clinicians. Therefore, it is hoped the education the postgraduates have received will enable them to discern what is credible and what is speculation.

I think as Mr Mew says this is a balanced review of the literature and the move towards "evidence-based practice" rather than "rational-probability (however derived) practice" is most likely to prevail.

Dr Jonathan Sandy
Consultant Senior Lecturer in Orthodontics
Bristol Dental School

TROUBLE WITH COVER SCREWS FOR IMPLANTS

Sir. The placement of cover screws for the Bränemark dental implants is usually straightforward. The authors of the recent implantology series encountered difficulty while placing one, and I would like to present this problem for discussion.

A fit lady in her forties underwent placement of six Bränemark dental implants. The author and colleagues placed all six Bränemark implants at one visit. This was followed by the placement of cover screws one after another. However, the author encountered difficulty screwing in one of the cover screws. Attempts by other colleagues were also futile. The author changed to a new cover screw thinking that there was a manufacturer's defect in the one tried earlier. However, it was also impossible to screw in.

The cover screw was eventually placed after irrigating the implant with copious amount of normal saline. No obvious cause could be found, but one of the hypothesis proposed to explain the problem is that because the implant was left exposed too long, a clot had formed inside the thread of the implant. This may hinder the action of screwing the cover screw.

Wei Cheong Ngeow
Lecturer, Department of Oral and Maxillofacial Surgery
University of Malaysia

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