European Journal of Orthodontics 18 (1996) 665–668

Letters to the Editor

Sir,

I wonder if I could add a comment to the article by Kluemper *et al.* (1995). In keeping with other SNORT articles by these authors, it is a very workmanlike piece of research, and I find myself in broad agreement with their listed conclusions.

However, their presentation might encourage less informed clinicians to believe that nasal obstruction *per se* has no influence on facial growth. It only requires a short period of increased nasal resistance to break the natural lip seal of the neonate and once destroyed it may never be restored, regardless of the subsequent airway status. There are many other researchers and clinicians who would feel that long-term open mouth postures have a substantial impact on facial growth (Linder-Aronson *et al.*, 1993).

Mr J Mew
21 Foxley Lane
Purley
Surrey CR8 3EH, UK

Sir,

The article by Kluemper *et al.* (1995) is seriously flawed. Measuring airway resistance in 17 year olds and relating it to facial morphology is ill-conceived. By the age of 17 most of the lymphoid tissue in the nasopharynx has atrophied, so it is meaningless to measure nasopharyngeal patency on the lateral headplate at that age.

Children whose nasal airway has been compromised from age 4 to 11 may have developed excessive vertical facial growth during that time. By the age of 17 their nasal airway may no longer be compromised, yet their facial form is established.

This flaw seriously impugns the conclusion that there is no significant association between craniofacial morphology and mode of respiration.

Dr R M Rubin
302 E. Little Creek Road
Norfolk, VA 23505, USA

Sir,

In the paper by Kluemper *et al.* (1995), the cephalometric morphology was measured and the respiratory mode assessed. A detailed statistical analysis was made in order to test the strength of an association between these two variables. Unfortunately, the protocol was flawed.

Firstly, in nearly half the sample growth had stopped and an abnormal mode would have an immeasurably small effect at this late stage, especially in the case of the 53 year old.

Secondly, even if the entire sample was within the growth period, a single recording would be suspect as there are variations in respiratory mode due to the behaviour of common obstructive agents such as adenoids. In a hypothetical case of adenoidectomy for obstructive hypertrophy, the result of a SNORT recording taken at a point in time before the operation would differ considerably from one taken after. If an abnormal respiratory mode is to have any marked effect on the growth pattern it must be in place for a long period, preferably starting early. Yet there was no mention of the nasal history in the text. It should come as no surprise that a negative conclusion was reached.

Even if the result had been positive, no cause and effect mechanism could be demonstrated so (i) respiration might be responsible for morphology, (ii) morphology for respiration, (ii) both responding to an unknown third dynamic force and (iv) mere coincidence.

Mr D. J. Timms
Department of Orthodontics
Royal Preston Hospital
Sharoe Green Lane North
Fulwood
Preston PR2 9HT, UK

Sir,

We appreciate this opportunity to respond to comments by Drs Mew, Rubin and Timms following publication of our paper (Kluemper *et al.*, 1995). This recent paper, as well as a

substantial body of work from this laboratory, questions not only the clinical significance of such a relationship, but even whether impaired nasal breathing can be accurately identified by clinicians. The study design did not address whether diminished nasal breathing contributes to the development of malocclusion. By the time patients see an orthodontist they already have the condition. The question is whether such patients should have anything done about their breathing, if so then what, and which patients are 'at risk'. In modern evidence-based medicine, therapies are only accepted as efficacious after being tested in controlled clinical trials. The onus is on the proponent of treatment, not on the sceptic.

Interventions to increase nasal breathing have never been subjected to systematic study, nor shown to predictably yield any well-defined orthodontic benefits. Hence no evidence exists to support their effectiveness of efficacy. In spite of this, belief to the contrary persists. Given that even the need for orthodontics itself is questioned by some as constituting a valid 'health' service, any unnecessary auxiliary or adjunctive treatment of questionable utility can only detract from the perceived benefits of orthodontics to society.

The above letters question our contention that there are no strong associations between orofacial morphology and nasorespiratory parameters. These objections are based on the writers' perceptions concerning the validity of our methods, as well as on some of their opinions concerning respiratory physiology. Our paper is by no means the only evidence for a lack of association between malocclusion and breathing. If the 'weight of evidence' is measured by the sheer numbers of papers, ours is in the minority of dissenters. That, however, is not how the scientific basis is established. We will attempt to address each writer's major concern and then to provide a more comprehensive background to our studies, of which the recent paper was merely one of a large number of publications (Hartgerink, 1986; Hartgerink *et al.*, 1987; Drake *et al.*, 1988; Vig and Hartgerink, 1989; Vig *et al.*, 1991).

Dr Mew states 'even a short period of increased nasal resistance can break the neonates' natural lip seal, which once destroyed may never be restored'. This is erroneous, as neonates do not have an anterior oral seal at all but maintain nasal respiration using a posterior oral seal. In fact lip seal, or 'lip competence', does not occur in most children until much later in facial development, and certainly after the establishment of the mixed dentition (Vig and Cohen, 1979; McLain *et al.*, 1983). He is right in stating that many other researchers and clinicians *feel* that 'open mouth postures' have an impact on facial growth. However, over the past 15 years we have found that most subjects with lip separation at rest do not breath predominantly through the mouth. In fact 100 per cent nasal breathing can, and often does, occur while the lips are separated. Thus 'treatments' to modify nasal respiration may well have absolutely no effect on orofacial muscle posture. The real issue, and primary focus of our paper, is whether clinicians can reliably identify which individuals have clinically significant nasal airway impairment, and what objective criteria differentiate between the presence and absence of such a condition! No unambiguous criteria have been demonstrated and therefore clinicians can not identify patients at risk, or consequently those who would benefit from such treatment—*even* if such treatment were proven to be effective.

Dr Rubin maintains that our conclusions are seriously impugned, because children may have elevated nasal resistance and developed excessive vertical growth at 4–11 years, but may no longer have a 'compromised' nasal airway when older. Our paper does not purport to test an aetiological hypothesis. It does explicitly evaluate the strength of correlations between form and function and, more importantly, demonstrates the lack of precision of two tests that have been advocated to confirm the diagnosis of impaired nasal function. Our sample was confined to patients referred for orthodontic treatment and therefore encompassed a wide spectrum of ages. It is these patients who are at risk for having surgical or other treatments to 'improve' nasal breathing recommended, and not the young children in the pre-orthodontic age. Excessive vertical facial growth can not be clearly ascertained at 4 years of age, and growth prediction in early childhood is problematic at

best. Consequently, the orthodontic diagnosis of vertical excess is generally not confirmed until later. As there are no objective criteria for 'impaired nasal breathing' and all young children have nasopharyngeal lymphoid tissue, would Dr Rubin advocate prophylactic adenoidectomy for its possible long-term orthodontic benefit?

Dr Rubin also fails to note that nasal resistance was not the only parameter of respiration that was evaluated. We also reported on nasal airway cross-sectional area, peak nasal airflow rate and the percentage of air inspired and expired through the nose. None of these variables correlate strongly with morphology, and their intercorrelation is also weak. This indicates, among other things, that nasal resistance is not a valid measure of the extent of 'mouth breathing' nor is the peak airflow rate (which was the methodological basis for the conclusions of Linder-Aronson, 1970) on whose original work many rely to support the aetiological significance of mouth breathing for malocclusion.

Dr Timms makes a cogent point concerning cause and effect, with which we agree. He points out that even if a consistent statistical association were demonstrable that would still beg the question of causation or the direction of causation. Does impaired nasal breathing cause morphological change, or does morphology contribute to respiratory behaviour or neither? As he rightly states, even if our results showed strong positive correlations between breathing characteristics and morphology the case for causation would still remain in doubt. Equally however, the justification for the rationality of treating respiration for orthodontic reasons would still need to be made. Neither case has been made.

Dr Timms alludes to the otolaryngological history of our subjects and to the variability of measurements of respiratory behaviour over time. We have data on both. Subjects who have had nasopharyngeal surgery were excluded from the analysis. We also tested the reproducibility of respiratory measurements over time. Nasal resistance does in fact fluctuate whereas the respiratory mode ascertained by the Simultaneous Oral and Nasal Respirometric Technique (i.e. the per cent nasal/oral breathing) is remarkably constant over time. Clinical decisions to treat nasal airway patency are generally not based on the same rigorous and reproducible protocols as those that we employed.

By way of a more general perspective, at the outset our studies sought to establish if nasorespiratory function was a significant aetiological factor in malocclusion and, if so, whether certain treatments, by promoting nasal breathing in preference to the oral mode, could be justified either to enhance facial growth or the prognosis of orthodontics or orthognathic surgery. Progress towards these goals required the objective quantification of respiration that was previously unavailable. This led to our developing the apparatus and protocols for SNORT (Keall and Vig, 1987). An alternative method for assessing respiratory mode was later developed by Warren *et al.* (1986). This remains the only current alternative to SNORT for determining respiratory mode (i.e. %N, the percentage of nasal breathing).

We mention his simply to point out that the bulk of the literature concerning orthodontics and breathing does not in fact directly compare morphology and degrees of oronasal breathing, but rather some variables which were presumed to be accurate reflections of the mode of respiration. Therefore the greater part of the 'evidence' for the mouthbreathing theory is lacking in precision and validity.

Lacking criteria based on objective measures of both 'normal', or 'impaired' nasal breathing, clinicians relied either on clinical impressions, or on uncorroborated and imprecise inferences which were based either on subjective observations or on indirect measurement. The non-specific, indefinable and therefore meaningless term 'mouthbreathing' is still widely used as a diagnostic label which variously denotes some clinically significant 'abnormality' or 'disease state', and is implicitly associated either with 'impaired' nasorespiratory function or 'habit'. This concept and its effects on clinical practice and thinking persist.

Our findings over the past 13 years indicate that only weak associations exist between such characteristics as respiratory mode (%N), nasal resistance (Nrz), peak nasal airway flowrate for

inspiration and expiration (PkNF), and minimum cross-sectional area of the nasal airway (CA). Therefore, these measurements of respiratory function are not acceptable alternatives for, nor are they accurate predictors of, %N.

We would like to offer your readers a final thought. What experimental evidence would suffice to persuade clinicians that variations in nasal respiratory behaviour are not clinically significant causes of malocclusion? Equally, when should treatment of the airway be integral to orthodontics? While pondering these questions, they might also consider whether it is reasonable to demand a lower standard of scientific rigour when verifying the efficacy for various treatments, than when challenging the utility of those same treatments!

T. Kleumper, P. Vig and K. Vig
College of Dentistry
The Ohio State University
Postle Hall
305 West 12th Avenue
Columbus, OH 43210-1241
USA

References

Drake A F Keall H J, Vig P S, Krause C J 1988 Clinical nasal obstruction and objective respiratory mode determination. Annals of Otology, Rhinology and Laryngology 97: 397–402

Hartgerink D V 1986 The effect of rapid maxillary expansion on nasal airway resistance: a one year follow-up. Masters' thesis, University of Michigan, Ann Arbor

Hartgerink D V, Vig P S, Abbott D W 1987 The effect of rapid maxillary expansion on nasal airway resistance. American Journal of Orthodontics and Dentofacial Orthopedics 92: 381–389

Keall C L, Vig P S 1987 An improved technique for the simultaneous measurement of nasal and oral respiration. American Journal of Orthodontics and Dentofacial Orthopedics 91: 207–212

Kluemper G T, Vig P S, Vig K W L 1995 Nasorespiratory characteristics and craniofacial morphology. European Journal of Orthodontics 17: 491–495

Linder-Aronson S 1970 Adenoids: their effect on mode of breathing and nasal airway and their relationship to characteristics of the facial skeleton and dentition. Acta Oto-laryngologica Supplement 25: 1–132

Linder-Aronson S, Woodside D G, Hellsing E, Emerson W 1993 Normalization of incisor position after adenoidectomy. American Journal of Orthodontics and Dentofacial Orthopedics 103: 412–417

McLain B, Steedle R, Vig P S 1983 Face height and dental relationships in 1600 children. A survey. Journal of Dental Research 62: 203 (Abstract)

Vig P, Cohen A 1979 Vertical growth of the lips. A serial cephalometric study. American Journal of Orthodontics 75: 405–415

Vig P S, Hartgerink D V 1989 Lower anterior face height and lip incompetence do not predict nasal airway obstruction. Angle Orthodontist 59: 17–23

Vig P S, Spalding P M, Lints R R 1991 Sensitivity and specificity of diagnostic tests for impaired nasal respiration. American Journal of Orthodontics and Dentofacial Orthopedics 99: 3543–360

Warren D W, Hinton V A, Hairfield W M 1986 Measurement of nasal and oral: respiration using inductive plethysomograpy. American Journal of Orthodontics 89: 480–484