Mouth breathing and orthodontic intervention: Does the evidence support keeping our mouths shut?

Sanjivan Kandasamy
St Louis, Mo, and Nedlands and Midland, Western Australia, Australia

It is believed that mouth breathing, particularly during early childhood, is associated with the development of various unfavorable craniofacial characteristics such as mandibular retrognathia, narrow high arch palates and alar bases, lip incompetence, increased vertical facial height, and mandibular plane angles. Today, there is significant debate and confusion pertaining to the role of orthodontists in identifying and managing mouth breathing, especially for young patients as early as 3 years old. Using the historical and contemporary evidence-based literature along with an ethical and pragmatic clinical perspective, the role of mouth breathing and early orthodontic intervention is put into perspective in this paper. (Am J Orthod Dentofacial Orthop 2025;167:629-34)

The debate pertaining to the role of orthodontists in assessing and managing patients with obstructive sleep apnea (OSA) has now also progressed to the importance of early detection of mouth breathing in all patients, including even patients as early as 3 years old.

Various clinicians around the world[1-4] believe that when children breathe through their mouths, this can lead to significant unfavorable craniofacial characteristics such as mandibular retrognathia, narrow high arch palates and alar bases, lip incompetence, and increased vertical facial height and mandibular plane angles (also referred to as the long face syndrome).[5] When the role of enlarged adenoids in causing nasal obstruction in association with mouth breathing and facial changes was first described by Wilhelm Meyer in the 1860s,[6] clinicians began labeling these associated facial changes as adenoid facies.

Center for Advanced Dental Education, Saint Louis University, St Louis, Mo. Dental School, The University of Western Australia, Nedlands, Western Australia, Australia. Private practice, West Australian Orthodontics, Midland, Western Australia, Australia
The author has completed and submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest, and none were reported.
Address correspondence to: Sanjivan Kandasamy, West Australian Orthodontics, Suite 9A, 401 Great Eastern Highway, Midland, 6056 Western Australia, Australia; e-mail, sanj@kandasamy.com.au.
Submitted, January 2025; revised and accepted, February 2025.
0889-5406

https://doi.org/10.1016/j.ajodo.2025.02.005

It is also believed that if a child is mouth breathing, there will be a lower posterior tongue posture which will result in the descent of the hyoid bone, which can impair mandibular growth.[1-4] These skeletal and dental malocclusions become more pronounced and complicated if mouth breathing persists into adulthood. If one continues to mouth breathe, there is a risk of developing unfavorable head and neck posturing,[7-9] which can lead to impaired cranial base flexing,[1] elongation of the face, and distal displacement of the mandible.[1] This has been suggested to result in cervical musculoskeletal issues in the long term.[10-12] There is the contention that when one breathes through their mouth at any age, there is a risk of behavioral and cognitive decline resulting in attention deficit hyperactivity disorder (ADHD).[13-17] It is also believed that when one mouth breathes during the day, this will inevitably lead to breathing issues during sleep.[1-4,18]

These authors propose early detection of mouth breathing and immediate attention with myofunctional therapy to establish an oral seal along with adequate tongue posture and appropriate speech and swallowing patterns. They advocate functional appliances to grow the mandible and change the trajectory of its growth, and maxillary expansion to increase the width of the maxilla and nasal cavity and reduce nasal resistance. All these treatments are essentially aimed at increasing the base of the nasal cavity, reducing nasal resistance, and increasing the space in the naso- and oro-pharynx, thereby facilitating nasal breathing. By

achieving this, it is believed that growing children will then be able to close their lips, breathe through their nose, and, in turn, grow and develop favorably.[1-4,18]

Despite the American Association of Orthodontists White Paper in 2019,[19] and subsequent publications[20,21] urging clinicians to adopt a conservative, evidence-based, and less invasive approach to these issues, there is a significant push by many convincing groups that mouth breathing is a significant cause of craniofacial and dental aberrations, orofacial musculature imbalance and function, sleep-disordered breathing, and unfavorable behavioral and cognitive issues.

As a result of this latest controversy, I was asked to put into perspective the current state of mouth breathing and early orthodontic intervention according to the evidence-based literature. This editorial will summarize that literature and hopefully will persuade clinicians to adopt an evidence-based approach to assessing and treating their young patients.

Before attempting to address these issues, it is pertinent to take a brief look at 2 frequently cited studies by Linder-Aronson[22] and Harvold et al[23,24] in the 1970s that have significantly influenced our specialty's beliefs surrounding mouth breathing, nasal obstruction, and craniofacial growth today. Conducted >40 years ago, these studies, despite their methodological limitations, have profoundly influenced many of the misconceptions about mouth breathing that continue to persist today.

In their studies, Harvold et al[23,24] induced nasal obstruction in growing rhesus monkeys (Macaca mulatta). The nasal obstruction increased anterior facial height and gonial angle, a steeper mandibular plane, and the development of various malocclusions ranging from open bites to Class II or III relationships.[25] After the removal of the nasal obstruction, there was some return to the original morphologic form; however, this outcome was variable.[26]

Several factors were not measured or assessed in those studies, and most of the changes observed were variable.[25] The primary issue with extrapolating these results and applying them to humans is that monkeys are obligate nasal breathers, which means that they cannot breathe through their mouths. They have an elongated velum that overlaps or connects with their epiglottis, thereby sealing off their oral cavity during nasal breathing and rendering it almost impossible to breathe orally. As a result of artificially inducing nasal obstruction, these animals are placed in a life-threatening situation, forcing them to posture their tongue and mandible forward and downward, exaggerating their posture to open up their oropharyngeal airway space. This extreme positioning would obviously lead to the dental and skeletal morphologic changes observed in these studies. However, humans only need to slightly open their mouths to breathe orally without any extreme posturing.[27] Humans are not obligate nasal breathers, and, therefore, to extrapolate what happens in monkeys into humans is profoundly unsubstantiated.[28] It is important to be aware of how this line of basic rationalization and deduction has developed and evolved over the past several decades into a commonly accepted (but unsubstantiated) belief that mouth breathing is the cause of significant vertical dental and skeletal changes in growing children.[27-29]

In the 1970s, Linder-Aronson[22] compared children with no adenoids with those with moderate adenoids and enlarged adenoids and then compared those subgroups against children who had their adenoids removed. The role of mouth breathing and tongue posture as a result of nasal obstruction was taken into consideration. However, there were many limitations and inconsistencies with this study, but the authors concluded that children with enlarged adenoids exhibited the least amount of nasal airflow and mouth breathing. Interestingly, the children who exhibited the least amount of nasal airflow did not exhibit any of the long facial characteristics of adenoid facies. However, the biggest issue with this study was that the mode of breathing was subjectively determined. Humans normally breathe through their nose and mouth interchangeably. No one is a complete mouth breather unless there is a total nasal obstruction preventing nasal respiration.

The key issue with many studies assessing the relationship between mouth breathing and dentofacial growth is what exactly defines mouth breathing, mainly because of the lack of objective criteria and tools for measuring breathing modes and the extent of nasal obstruction. Most of these assessments are crude and subjective based on patient reports, and as a result, they generally tend to overestimate the extent of mouth breathing. Only a handful of studies[30-32] have used objective and thorough means of measuring respiratory cycles along with oral and nasal airflow with high reliability and validity.

On the basis of the studies described above, only an associational relationship, at best, between severe nasal obstruction and dentofacial growth can be drawn.[27-29] However, an association does not mean causation. The simple act of having one's lips apart does not mean one is a mouth breather.[27] When objective means of measuring respiratory modes and the percentage of nasal airflow and impairment are used,[30-32] it would appear that a causal relationship between respiratory behavior and dentofacial growth cannot be drawn.

GROWING MANDIBLES

Several authors[1,2] have suggested that functional appliances positively affect the oropharyngeal airway by promoting forward growth of the mandible. In growing children, this can result in the recovery of suppressed growth hormone levels, further promoting mandibular growth, increased oxygen saturation, and improving OSA.[1,33] Interestingly, the discussion and rationale surrounding growth hormone and functional appliances were based on a study[33] on an adolescent rat model which is completely invalid and inapplicable to humans.

As previously discussed, the evidence-based literature[34-40] has already shown us that we cannot grow mandibles, and the mandible is going to grow to where it is going to grow. Functional appliances do not provide extra mandibular growth or change the trajectory of growth of the mandible and/or flex in the cranial base.

Do all mouth breathers have retruded mandibles? Do all mouth breathers have a long face? Can we change patients with a long (hyper/dolicho) face to a short (hypo/brachy) face? The simple answer is no.

Why has the specialty fixated on the effects of nasal obstruction in patients with a long face and pretended that this is not a concern in patients with a short face? Is it because this is not good for business? How do we explain the effects of significant nasal obstruction in growing patients with a short face? Do they develop anterior open bites or develop a long face as a result of their nasal obstruction? The answer is no. It is more complicated than that.

Interestingly, in most children, the vertical facial pattern is established as early as 4-5 years old.[41,42] In a small proportion of children, the vertical pattern may slightly change from normal to either long or short face. Furthermore, the masticatory muscle forces of children aged up to 10 years with long faces are the same as children with normal faces.[43] So, does chronic nasal obstruction and mouth breathing occur in all children with long faces before the age of 4 years, causing them to develop into a long face? The answer is no. There is a significant genetic component (form over function) to children with long faces, more than many would like to accept. It is more complicated than mouth breathing and nasal obstruction, leading to children with long faces.

EXPANSION

Several clinicians[1,2,44] advocate maxillary expansion in young children with mouth breathing with OSA as early as possible to establish nasal breathing to prevent irreversible changes in craniofacial growth.

It is well established that expansion, when there is no crossbite or transverse discrepancy in children purely on the basis of addressing mouth breathing or underlying sleep-disordered breathing, is unsubstantiated by the evidence.[19-21,45] Furthermore, if we expand when there is no crossbite in a patient with a long face, this may result in unnecessary extrusion of the overexpanded maxillary posterior teeth and lingual tipping of the opposing mandibular posterior teeth in the long term; in addition, it often leads to unwanted bite opening and dentoalveolar compensations. Maxillary expansion in these instances may increase the vertical facial height and may further create or worsen lip incompetence, mouth breathing, and OSA.

Continued growth and development and the resolution of lymphoid proliferation in childhood[46] generally lead to the spontaneous resolution of pediatric OSA. This further supports the decision not to carry out maxillary expansion routinely to address OSA and mouth breathing, especially when there is no transverse discrepancy.[47]

ATTENTION DEFICIT HYPERACTIVITY DISORDER

ADHD is a highly heritable complex childhood-onset neurodevelopmental disorder. Exposures to a range of prenatal and perinatal factors, environmental toxins, dietary factors, and psychosocial factors have also been associated with ADHD. It is characterized by developmentally distinct hyperactivity, inattention, and impulsiveness.[48-50]

There is a common misconception linking mouth breathing as a causative factor to ADHD. Many clinicians believe that mouth breathing leads to OSA, and in turn, this leads to ADHD.[13,15]

This is a far-reaching and very improbable claim. Although OSA-related fragmented sleep can potentially contribute to a child exhibiting signs and symptoms similar to ADHD, it does not cause ADHD. Chronic nasal obstruction or OSA needs to be screened during initial examinations, but it is critical that clinicians do not overdiagnose ADHD in children and carry out unnecessary and unsubstantiated treatments such as expansion or growth modification on the sole basis that the child might exhibit mouth breathing.

Myofunctional therapy is promoted as a means to correct improper tongue position, speech and swallowing patterns, mouth breathing as well as alleviating OSA and promoting mandibular growth.[1,44] What exactly qualifies as an unfavorable tongue position? Can we genuinely grow mandibles and stop mouth breathing with myofunctional therapy? Why has the orthodontic specialty not embraced this modality of treatment to cure the world of retrusive mandibles and mouth breathers? Because we cannot.[21,45] Although

these benefits may appear quite compelling, how feasible is it to engage very young children, as young as 3-4 years old, in these exercises and ensure their long-term adherence? What happens when they stop therapy? What happens to these children when they are asleep, a state of unconsciousness in which voluntary control of these trained muscles ceases? Does this truly prevent mouth breathing or OSA? Is it that simple? The answer is no.

Humans normally breathe through their nose and mouth, and this activity interchanges throughout the day. No one is a complete nasal or mouth breather unless there is significant obstruction present. There is no doubt that when we breathe through our nose, the air is warmed, filtered, and moistened, allowing the lungs to function and absorb oxygen more efficiently, along with its immunoprotective effects.[51,52] But what happens when we breathe through our mouth? The air is less filtered, moistened, and warmed. Clearly, we need to be able to breathe through our mouths in times of stress, exercise,[53] and when there is nasal congestion or obstruction. If patients experience chronic nasal obstruction and have to breathe through their mouth, we must aim to identify the cause of this nasal obstruction and manage this accordingly to improve the quality of life and facilitate normal nasal breathing along with mouth breathing.

CONCLUSIONS

On the basis of the findings from those early studies by Linder-Aronson and Harvold and associates, many practitioners in both the medical and dental specialty have adopted the belief that mouth breathing is indicative of nasal obstruction leading to detrimental postural and dentofacial changes. Over the many decades, clinicians around the world who accepted those beliefs and have been influenced by significant confirmation and cognitive biases have assessed and managed patients within this framework of belief and bias.

Many clinicians genuinely believe that the early detection of mouth breathing is an indication to establish an oral seal and an adequate tongue posture along with functional appliances to grow the mandible and change the trajectory of its growth and maxillary expansion to increase the width of the maxilla and nasal cavity and reduce nasal resistance.

We have to appreciate the possibility that some clinicians are using a widely relatable concept such as mouth breathing and oversensitizing the general public about its unsubstantiated detrimental consequences, especially to young children. On the surface, mouth breathing appears to seem less commercially driven, more holistic, and an indirect method for airway-dentistry advocates to promote and grow their practices. It presents an invaluable financial opportunity for clinicians to draw patients into their practices and use it to potentially overdiagnose OSA, narrow maxillae, and retruded mandibles. As a result, they may overservice these ailments under the guise of curing or preventing any underlying breathing issues. However, this practice is not supported by the evidence and is unethical.

On the basis of the available literature, we can accept that when the nasopharyngeal or oropharyngeal airway spaces are small or obstructed, this may lead to exaggerated postural responses in some patients, and this may influence dentofacial growth unfavorably. We need to identify these patients.

Simply asking a child or parent if one is a mouth breather is not a scientifically objective method. Diagnosing a child as a mouth breather based on dentofacial characteristics such as a long face or adenoid facies alone does not provide sufficient evidence to confirm chronic nasal obstruction.

Nasal obstruction is real. It can manifest itself as swelling and congestion of the intranasal tissues because of a variety of reasons, such as allergies, irritants and infections, or physical obstruction, such as structural abnormalities or growths. As part of our screening process of OSA, we need to screen for signs and symptoms of nasal obstruction, provide the necessary referrals, and address any orthodontic issues appropriately without conflating this with treating nasal obstruction. It is that simple. Promoting misinformation and fear by suggesting mouth breathing is linked to many underlying health problems, potentially leading to various growth and developmental issues, is baseless, scientifically flawed, and misleading.

It would appear that our mouths should never be shut when it comes to being accountable to the evidence on mouth breathing. We need to do better for our patients and our specialty.

CREDIT AUTHORSHIP CONTRIBUTION STATEMENT

Sanjivan Kandasamy: Conceptualization; Data curation; Formal analysis; Investigation; Validation; Visualization; Writing – original draft; Writing – review & editing.

REFERENCES

1. Yoon A, Gozal D, Kushida C, Pelayo R, Liu S, Faldu J, et al. A roadmap of craniofacial growth modification for children with sleep-disordered breathing: a multidisciplinary proposal. Sleep 2023; 46:zsad095.

2. Kim KA, Kim SJ, Yoon A. Craniofacial anatomical determinants of pediatric sleep-disordered breathing: a comprehensive review. J Prosthodont 2024; https://doi.org/10.1111/jopr.13984.
3. Torre C, Guilleminault C. Establishment of nasal breathing should be the ultimate goal to secure adequate craniofacial and airway development in children. J Pediatr (Rio J) 2018;94:101-3.
4. Valcheva Z, Arnautska H, Dimova M, Ivanova G, Atanasova I. The role of mouth breathing on dentition development and formation. JofIMAB 2018;24:1878-82.
5. Schendel SA, Eisenfeld J, Bell WH, Epker BN, Mishelevich DJ. The long face syndrome: vertical maxillary excess. Am J Orthod 1976; 70:398-408.
6. Ruben RJ. The adenoid: its history and a cautionary tale. Laryngoscope 2017;127:S13-28.
7. Solow B, Kreiborg S. Soft-tissue stretching: a possible control factor in craniofacial morphogenesis. Scand J Dent Res 1977;85: 505-7.
8. Solow B, Ovesen J, Nielsen PW, Wildschiødtz G, Tallgren A. Head posture in obstructive sleep apnoea. Eur J Orthod 1993;15: 107-14.
9. Solow B, Siersbaek-Nielsen S, Greve E. Airway adequacy, head posture, and craniofacial morphology. Am J Orthod 1984;86: 214-23.
10. Mahmoud NF, Hassan KA, Abdelmajeed SF, Moustafa IM, Silva AG. The relationship between forward head posture and neck pain: a systematic review and meta-analysis. Curr Rev Musculoskelet Med 2019;12:562-77.
11. Okuro RT, Morcillo AM, Ribeiro MÂGO, Sakano E, Conti PBM, Ribeiro JD. Mouth breathing and forward head posture: effects on respiratory biomechanics and exercise capacity in children. J Bras Pneumol 2011;37:471-9.
12. Okuro RT, Morcillo AM, Sakano E, Schivinski CIS, MÂGO Ribeiro, Ribeiro JD. Exercise capacity, respiratory mechanics and posture in mouth breathers. Braz J Otorhinolaryngol 2011;77:656-62.
13. Kalaskar R, Bhaje P, Kalaskar A, Faye A. Sleep difficulties and symptoms of attention-deficit hyperactivity disorder in children with mouth breathing. Int J Clin Pediatr Dent 2021;14:604-9.
14. Schredl M, Alm B, Sobanski E. Sleep quality in adult patients with attention deficit hyperactivity disorder (ADHD). Eur Arch Psychiatry Clin Neurosci 2007;257:164-8.
15. Huang YS, Guilleminault C, Li HY, Yang CM, Wu YY, Chen NH. Attention-deficit/hyperactivity disorder with obstructive sleep apnea: a treatment outcome study. Sleep Med 2007;8:18-30.
16. Jung JY, Kang CK. Investigation on the effect of oral breathing on cognitive activity using functional brain imaging. Healthcare (Basel) 2021;9:645.
17. Sano M, Sano S, Oka N, Yoshino K, Kato T. Increased oxygen load in the prefrontal cortex from mouth breathing: a vector-based near-infrared spectroscopy study. NeuroReport 2013;24:935-40.
18. Nestor J. Breath- The new science of a lost art. Dublin: Penguin Random House Ireland; 2021.
19. Behrents RG, Shelgikar AV, Conley RS, Flores-Mir C, Hans M, Levine M, et al. Obstructive sleep apnea and orthodontics: an American Association of Orthodontists White Paper. Am J Orthod Dentofacial Orthop 2019;156:13-28.e1.
20. Kandasamy S. Sleep disordered breathing and dentistry: waking up to the reality. Semin Orthod 2019;25:296-303.
21. Kandasamy S. Obstructive sleep apnea and early orthodontic intervention: how early is early? Am J Orthod Dentofacial Orthop 2024; 165:500-2.
22. Linder-Aronson S. Adenoids. Their effect on mode of breathing and nasal airflow and their relationship to characteristics of the facial skeleton and the denition. A biometric, rhino-manometric and cephalometro-radiographic study on children with and without adenoids. Acta Otolaryngol Suppl 1970;265:1-132.
23. Harvold EP, Vargervik K, Chierici G. Primate experiments on oral sensation and dental malocclusions. Am J Orthod 1973;63: 494-508.
24. Harvold EP, Tomer BS, Vargervik K, Chierici G. Primate experiments on oral respiration. Am J Orthod 1981;79:359-72.
25. O'Ryan FS, Gallagher DM, LaBanc JP, Epker BN. The relation between nasorespiratory function and dentofacial morphology: a review. Am J Orthod 1982;82:403-10.
26. Vargervik K, Miller AJ, Chierici G, Harvold E, Tomer BS. Morphologic response to changes in neuromuscular patterns experimentally induced by altered modes of respiration. Am J Orthod 1984;85:115-24.
27. Hartgerink DV, Vig PS. Lower anterior face height and lip incompetence do not predict nasal airway obstruction. Angle Orthod 1989;59:17-23.
28. Warren DW. Effect of airway obstruction upon facial growth. Otolaryngol Clin North Am 1990;23:699-712.
29. Vig KW. Nasal obstruction and facial growth: the strength of evidence for clinical assumptions. Am J Orthod Dentofacial Orthop 1998;113:603-11.
30. Warren DW. A quantitative technique for assessing nasal airway impairment. Am J Orthod 1984;86:306-14.
31. Gurley WH, Vig PS. A technique for the simultaneous measurement of nasal and oral respiration. Am J Orthod 1982;82: 33-41.
32. Drake AF, Keall H, Vig PS, Krause CJ. Clinical nasal obstruction and objective respiratory mode determination. Ann Otol Rhinol Laryngol 1988;97:397-402.
33. Wang S, Ye L, Li M, Zhan H, Ye R, Li Y, et al. Effects of growth hormone and functional appliance on mandibular growth in an adolescent rat model. Angle Orthod 2018;88:624-31.
34. Batista KB, Thiruvenkatachari B, Harrison JE, O'Brien KD. Orthodontic treatment for prominent upper front teeth (Class II malocclusion) in children and adolescents. Cochrane Database Syst Rev 2018;3:CD003452.
35. Keeling SD, Wheeler TT, King GJ, Garvan CW, Cohen DA, Cabassa S, et al. Anteroposterior skeletal and dental changes after early Class II treatment with bionators and headgear. Am J Orthod Dentofacial Orthop 1998;113:40-50.
36. O'Brien K, Wright J, Conboy F, Sanjie Y, Mandall N, Chadwick S, et al. Effectiveness of early orthodontic treatment with the twin-block appliance: a multicenter, randomized, controlled trial. Part 1: dental and skeletal effects. Am J Orthod Dentofacial Orthop 2003;124:234-43.
37. O'Brien K, Wright J, Conboy F, Chadwick S, Connolly I, Cook P, et al. Effectiveness of early orthodontic treatment with the twin-block appliance: a multicenter, randomized, controlled trial. Part 2: psychosocial effects. Am J Orthod Dentofacial Orthop 2003; 124:488-94.
38. O'Brien K, Wright J, Conboy F, Sanjie Y, Mandall N, Chadwick S, et al. Effectiveness of treatment for Class II malocclusion with the herbst or twin-block appliances: a randomized, controlled trial. Am J Orthod Dentofacial Orthop 2003;124:128-37.
39. Tulloch JFC, Phillips C, Koch G, Proffit WR. The effect of early intervention on skeletal pattern in Class II malocclusion: a randomized clinical trial. Am J Orthod Dentofacial Orthop 1997; 111:391-400.
40. Tulloch JFC, Proffit WR, Phillips C. Outcomes in a 2-phase randomized clinical trial of early Class II treatment. Am J Orthod Dentofacial Orthop 2004;125:657-67.

41. Bishara SE, Jakobsen JR. Longitudinal changes in three normal facial types. Am J Orthod 1985;88:466-502.
42. Nanda SK. Patterns of vertical growth in the face. Am J Orthod Dentofacial Orthop 1988;93:103-16.
43. Proffit WR, Fields HW. Occlusal forces in normal- and long-face children. J Dent Res 1983;62:571-4.
44. Huang YS, Guilleminault C. Pediatric obstructive sleep apnea and the critical role of oral-facial growth: evidences. Front Neurol 2012;3:184.
45. Sheats R, Masse JF, Levine M, Aarab G, Meira e Cruz MM, Simmons M, et al. Novel therapies for preventing, managing and treating obstructive sleep apnea and snoring in pediatric and adult patients. J Dent Sleep Med 2024;11:2.
46. Scammon RE. The measurement of the body in childhood. In: Harris JA, Jackson DG, Peterson DG, Scammon ER, editors. The measurement of Man. Minneapolis: University of Minnesota Press; 1930. p. 173-215.
47. Fernández-Barriales M, Lafuente-Ibáñez de Mendoza I, Alonso-Fernández Pacheco JJ, Aguirre-Urizar JM. Rapid maxillary expansion versus watchful waiting in pediatric OSA: a systematic review. Sleep Med Rev 2022;62:101609.
48. Thapar A, Cooper M, Jefferies R, Stergiakouli E. What causes attention deficit hyperactivity disorder? Arch Dis Child 2012;97:260-5.
49. Thapar A, Cooper M. Attention deficit hyperactivity disorder. Lancet 2016;387:1240-50.
50. Drechsler R, Brem S, Brandeis D, Grünblatt E, Berger G, Walitza S. ADHD: current concepts and treatments in children and adolescents. Neuropediatrics 2020;51:315-35.
51. Djupesland PG, Chatkin JM, Qian W, Haight JS. Nitric oxide in the nasal airway: a new dimension in otorhinolaryngology. Am J Otolaryngol 2001;22:19-32.
52. Lundberg JO, Settergren G, Gelinder S, Lundberg JM, Alving K, Weitzberg E. Inhalation of nasally derived nitric oxide modulates pulmonary function in humans. Acta Physiol Scand 1996;158:343-7.
53. Lörinczi F, Vanderka M, Lörinczová D, Kushkestani M. Nose vs. mouth breathing- acute effect of different breathing regimens on muscular endurance. BMC Sports Sci Med Rehabil 2024;16:42.