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TOPICS IN PEDIATRICS

Midface alterations in childhood as pathogenesis of obstructive sleep apnea syndrome☆

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Abstract The onset of nasal breathing sets a genetically determined impulse to aerate the face cavities or paranasal sinuses, which in turn initiate their growth creating a useful trafficable space for air during the development of the midface. Considering the evidence that the upper airway obstruction has a primary role in the pathogenesis of respiratory sleep disorders, any condition that causes a permanent difficulty to nasal airflow during breathing will cause hypodevelopment of the required amplitude in this passage, reducing the growth stimulation of the sinus cavities and altering the development of the whole midface.

PALABRAS CLAVE
Tercio medio facial;
Aireación;
Infancia;
Síndrome de apnea-hipopnea obstructiva del sueño (SAHOS)

Alteraciones del tercio medio facial en la infancia como patogénesis del síndrome de apnea obstructiva del sueño

Resumen El inicio de la respiración nasal marca un impulso genéticamente determinado para airear las cavidades de la cara o senos paranasales, que a su vez inician su crecimiento y forman el espacio útil transitable desde el punto de vista respiratorio durante el desarrollo del tercio medio facial. Considerando la evidencia de que la obstrucción de la vía aérea superior tiene un rol primordial en la patogénesis de los trastornos respiratorios del sueño, cualquier

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patología que cause dificultad permanente al flujo aéreo nasal durante la respiración llevará a un hipodesarrollo de la amplitud requerida en esta vía, disminuyendo la estimulación del crecimiento de las cavidades sinusales y alterando el desarrollo del tercio medio facial en su conjunto.

1. Introduction

Human breathing is a basic function of life. The human being is born conditioned to breathe through the nose and to feed through the mouth, and the process of breathing requires the free passage of air through the nasal and naso-oro-pharyngeal spaces. An adequate respiratory function associated with a correct masticatory, swallowing and labial and lingual muscular action will stimulate facial growth and development in its whole given that bone growth responds to the adequate functioning of the muscles and facial soft tissues, as it is described in Moss' theory.[1] Growth of the midface and the conformation of the dental arcade initiate from the first respiration and finalize at the end of the second childhood,[2] linked to the adequate ventilation of the paranasal sinuses in relation to the volume of air that can pass through the nose.[3] The midface is integrated by the bones that form the roof of the mouth, the floor and the lateral walls of the orbit, a large part of the nasal cavity, which accommodates the nasal septum, the inferior, middle and superior turbinates, as well as the multiple cavities of the maxillary and ethmoidal sinuses, which serve as a support and give shape to the soft tissues from which the external configuration of the face will depend. Therefore, these tissues have a great physiological and esthetic relevance. Any disease that involves these structures can contribute to an alteration of their growth and development.

2. Biomechanics of the upper airway

Previous research has outlined the dynamic alterations of permeability as a function of the intraluminal pressure throughout the flexible segments of the cardiovascular, gastrointestinal and genitourinary biological conduits.[4–8] In the case of the upper airways, the permeable and flexible segment, which corresponds to the pharynx, is connected through two rigid segments. The upstream segment corresponds to the nose and the downstream segment to the trachea (Fig. 1A).

The air conduits of the upper and lower segments of the flexible site have fixed diameters and resistances: upstream segment resistance (R_{US}) and downstream segment resistance (R_{DS}); and variable pressures—upstream segment pressure (P_{US}) and downstream segment pressure (P_{DS}). It is important to mention several characteristics of this model, known as Starling's model of resistance, highlighting the following concepts:

A) The pressure outside the rigid conduits and the flexible conduit is positive; inside, the pressure is negative, which allows the air current to flow freely through the conduits
B) The components of the system generate resistance to the passing of air; by increasing resistance, more pressure is required for the air to flow to the interior of the system
C) The rigid segments of the conduit do not have a risk to collapse, only the flexible portion. Based on this, a new concept is generated: the critical pressure (P_{CRIT}), which represents the risk of total or partial collapse of the flexible portion, and results in a greater or lesser obstruction
D) When the upper portion of the segment is obstructed, the pressure inside the conduit is modified, which increases the usual pressure from -18 to -10 cmH_2O to +4 cmH_2O, which closes or collapses the airway in the flexible segment during sleep.[10] When the P_{CRIT} is greater than the P_{US} and P_{DS}, which connect with the flexible segment, the intramural pressure is positive, the airways close and airflow ceases (Fig. 1B).

Flow can be reestablished by elevating the P_{US} above the P_{CRIT}. If the P_{US} and the P_{DS} are greater than the P_{CRIT}, the intramural pressure is negative, the airways open and allow an adequate airflow (Fig. 1C). In these conditions, flow through the upper airway is proportional to the pressure gradient through the entire airway, and it can be described by the tension-current relation of Ohm's law:

$$V_{IMAX} = \frac{P_{US} - P_{DS}}{R_{US} + R_{DS}}$$

where V_{IMAX} represent the maximal inspiratory volume.

In contrast, when the P_{US} is greater than the P_{CRIT} and the P_{DS} is lower than the P_{CRIT}, the airways operate in a flow-limited condition (Fig. 1D). Since the inspiratory cycle varies rapidly between closed and opened status, the pressure in the flexible segment remains almost constant in relation to the P_{CRIT}. If the pressure in the flexible segment is constant, airflow also remains constant. Under these circumstances, airflow becomes independent of the P_{DS} and reaches a level of V_{IMAX}. Given that the P_{CRIT} replaces the P_{DS}, this favors an effective inspiratory flow return. Therefore, the level of V_{IMAX} is determined by the gradient of the P_{US} and P_{CRIT} divided by the resistance through the upstream segment in accordance with the following equation:

$$V_{IMAX} = \frac{P_{US} - P_{CRIT}}{R_{US}}$$