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Letter to the Editor

Incidence of Early Reversible Nasal Obstruction

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Nasal obstruction is considered a risk factor in sleep-disordered breathing [1-3] which has a very negative impact on quality of life in children and adults with increased daytime sleepiness [4] this symptom resembles that of obstructive sleep apnea (OSA) caused by episodes of upper airway obstruction leading to episodic hypercapnic hypoxia which alters upper airway muscle structure and fiber type expression [5]. The most common clinical manifestations of OSA are nocturnal snoring, respiratory pauses, restless sleep and mouth breathing [6]. This disturbed breathing is known to produce lethargy, cognitive impairment and sleep impairment, especially in children [7,8]. Chronic nasal obstruction is a non-specific condition observed in many pathophysiological conditions e.g. allergic rhinitis, rhinosinusitis, adenoid hypertrophy and nasal polyps. Impaired nasal breathing results in obligatory oral breathing, which can be divided into two components: chronic absence of active nasal respiration that results in an olfactory deprivation [9] and chronic mouth opening [10]. Furthermore, in contrast to oral breathing, nasal breathing allows the optimal conditioning of inhaled air, clearing, moistening and warming the air before the gas exchange in the lungs [11,12].

Obligatory mouth breathing has been observed in human babies and has been associated with a number of conditions that could have both short and long term effects on the physiology and thus behaviour of these infants later on in adolescence. Decreases in oxygen saturation and respiratory frequency, with an increase in arousal were observed with nasal occlusion in preterm infants [13]. If untreated oral breathing in children can induce long narrow faces, narrow mouths, high palatal vaults, dental malocclusion, gummy smiles and other effects like skeletal facial profiles. These children do not sleep well at night and this lack of sleep can adversely affect growth and academic performance [14,8]. Oral breathing has been associated with increased net water loss in healthy subjects [15]. This would be expected given that oral breathing does not allow conditioning of the inhaled air or demoisturising of the exhaled air. However, in the case of suckling rat pups dehydration could be consequence of decreased feeding due to a difficulty imposed by oral breathing and suckling at the same time [16]. This could depend also on the position of the pups in the hierarchy on the nipple, males before females and stronger pups before weaker ones.

We have shown for the first time that in rat 4 days of forced oral breathing during early reversible nasal obstruction induced body weight loss associated with dehydration (increased plasma osmolality) and increased release of vasopressin. Associated with these changes in hydration in response to nasal obstruction was an intense stress response in rat pups. One day after implementing nasal obstruction, basal corticosterone levels had increased (by over 1000). The corticosteroid levels remained elevated when the signals of hydration had returned to normal [17]. Early nasal obstruction was associated with a decrease of oxygen consumption and carbon dioxide elimination [18]. Diaphragm muscle showed significant increases in adult isoforms (MHC 1, 2a) MHC neonatal and adult type 1 isoforms in two muscles involved with oral breathing, Masseter Superficialis (MS) and Anterior Digastric (AD) are increases. During this oral breathing period no changes were observed in the Levator Nasolabialis (LN) muscle involved with nasal breathing [19].

Early nasal obstruction was associated with a decrease in the vertical development of the nasomaxillary complex and in the development of the longitudinal skull base in both sexes [20].

We conclude that a 4 day nasal obstruction period in young rats leads to hormonal changes to Diaphragm myosin heavy chain structural and nasomaxillary complex adaptation.

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