Phenotypes in obstructive sleep apnea

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The Obstructive Sleep Disorders (OSD) includes upper airway resistance syndrome (UARS) and obstructive sleep apnea (OSA). Although the pathophysiology of OSD is not yet fully elucidated, it is known that anatomical abnormalities (craniofacial abnormalities and obesity) increase the susceptibility of the pharynx to collapse. However, non-anatomical mechanisms are now described to explain the presence of OSA. The four mechanisms currently described involved in the pathophysiology of OSA are: Anatomical changes/collapsibility of the upper airway, low arousal threshold, muscle factor and "high loop gain". Upper airway collapsibility is associated with the imbalance of extrinsic and intrinsic pharyngeal pressures and changes in critical closure pressure (Pcrit); muscular factors are associated with good or poor response depending on the negativity of pharyngeal pressure or gas changes such as hypoxemia or hypercapnia; the low arousal threshold is related to increased arousability at less negative intrapharyngeal pressures and, finally, the "high loop gain" is associated with hyperventilation (reduction of CO2) and sometimes, as a consequence, central apnea. Accurate and individualized diagnosis has been the key to success in choosing the right treatment for OSA. Thus, current research seeks to define the mechanisms that lead patients to develop sleep apnea, the so-called pathophysiological phenotypes or also known as the endotypes of OSA. The term "phenotype" is defined by the expression of an individual characteristic resulting from genetic and environmental interaction, whereas the endotypes represent the different pathophysiological processes that culminate in OSA (anatomical alterations/collapsibility of the upper airway, low arousal threshold, muscle factor (muscle response) and the "high loop gain").

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