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Sleep bruxism: The braking effect of GABA

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This article, in addition to reviewing our previous contribution to the literature on this subject, aims to explain bruxism in both its physiological expression, namely rhythmic masticatory muscle activity (RMMA), as well as in its pathological expression, characterized by tooth grinding and/or clenching during sleep. We will begin with a description of the mesencephalic trigeminal nucleus and then discuss sleep and its macro and micro structure in the non-rapid eye movement (NREM) stage. Finally, we will propose our hypothesis that during NREM sleep the trigeminal motor nucleus (Mo5) is activated: this activation is due to the acute effect of the inhibitory neurotransmitters gamma-aminobutyric acid (GABA) and Galanin, which are released from the ventrolateral preoptic nuclei (VLPO) and from the median preoptic nuclei (MnPO) located in the brainstem. Mo5 activates the mesencephalic trigeminal nucleus (Me5), which in turn releases glutamate. The release of glutamate curbs the inhibitory effect of GABA and Galanin of hypothalamic origin on some nuclei of the brainstem, in particular on those of reticular formation (FR), which is responsible for the ascending reticular activator system (ARAS). When the amount of GABA of hypothalamic origin is in excess, the activity of Mo5 continues; bruxism then becomes pathological.

Biography

Giorgia Andrisani has completed her PhD from Alfonso X el Sabio in Madrid and is now busy with her Master in Orthodontics. She works full time for a private practice in the Netherlands and part time for the private practice Andrisani. She is part of the ISPID organisation to discover the cause and prevent SIDS.

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