Introduction

Although well established as a neurogenic reflex pursuing a cardiovascular protective control in physiological conditions, trigemino-cardiac reflex (TCR) mechanisms and its clinical significance remains unclear. This powerful brainstem oxygen-conserving autonomic reflex usually manifests as sudden onset of hemodynamic changes on heart rate (HR) and blood pressure (BP) and has been associated to arrhythmias, asystole, apnea and disturbed gastric mobility [1]. The automatic response can be activated by mechanical or chemical stimulation at any point in the course of the trigeminal nerve and their branches and usually gets abolished after the removal of the inciting stimulus. Sometimes however persistence of TCR can result in exaggerated response which may have fatal consequences, particularly in pediatric age and in elderly. This is of primary relevance when TCR occur in the chronic form which is largely underestimated and under reported [2]. Though the role of TCR have been investigated in surgical related patho physiological conditions, it was recently admitted that it can have a clear significance in the aim of several sleep disturbances as sudden infant death syndrome (SIDS), obstructive sleep apnea (OSA) and sleep related bruxism (SB) [3]. In a review published in these same issue of OJNBD, Ken Luco looked for establishing a relationship between TCR and Sleep Bruxism, a sleep related movement disorder characterized by transient further recurrent autonomic changes (sympathetic hyper activation) generating tachycardia, tachypnea and hypertension chronologically associated to rhythmic masticatory muscle hyper activation. The better knowledge of the TCR will help us to understand other factors influencing the autonomic modulation during sleep and its association with sleep quality, cardiovascular risk and neurocognitive functioning.

This relationship is of particular clinical interest because cardiovascular autonomic changes associated with SB can be directly and indirectly (by metabolic induced cardiovascular changes) related to an increased cardiovascular risk in these patients. This can happen because of sympathetic over ac-
tivation related to RMMA itself or because of the stress and pain associated to temporomandibular joint (TMJ) which may be secondary to RMMA or SB.

Depending on how this clinical entity is perceived by the clinical specialty which primarily follows the patient, SB could be intended as a mild to moderate, non-significant clinical odontoestomatomological problem affecting teeth, periodontal structures and eventually TMJ or a sleep related problem which is often of mild severity since complaints and objectively evaluated dysfunction can be discrete enough and then neglected and receiving little or no clinical attention at all [4]. However, if we consider that cardiovascular disorders are still one of the most prevalent and challenging conditions contributing to elevated rates of mortality and disability with increasing health related costs (either because of direct—e.g. pharmacological or indirect—e.g. absenteeism and reduced productivity related costs) it turns obvious the need of being aware on how can we predict an occurrence and that we should hardly try to do it. On the other hand we should certainly be conscious about the possibility of avoiding worse prognosis in terms of cardiovascular outcomes in the long term thus preventing either non-fatal or fatal events. Treating and managing SB should therefore be intended as a more challenging issue than it is currently perceived and the link between SB and TCR is probably one of the major phenomena requiring particular diagnostic considerations and playing an important role on integrated clinical decision making.

References