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EDITORIAL COMMENT

Reflex syncope: Insights into physiology

Síncope reflexa: reflexões sobre a fisiologia

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The authors[1] report on an interesting case describing an 80-year-old patient with recurrent cervical pain accompanied by transient loss of consciousness (T-LOC). The diagnostic workup revealed paroxysmal asystole as the cause of T-LOC and a pacemaker was implanted. Subsequently, the patient was diagnosed with glossopharyngeal neuralgia, and it was established as the trigger for the patient's cardioinhibitory syncope.

When taking into consideration a physiological reflex loop, one must bear in mind the trigger, the afferent loop, the efferent loop, and the effector. As such, in the presence of a pathological reflex such as neurocardiogenic syncope, one might find a variety of somatic or visceral triggers and afferent loops, and a common efferent loop in the form of the autonomic nervous system (ANS) with vagal efferents leading to splanchic vasodilation and bradycardia and sympathetic efferents leading to peripheral vasodilation. Ultimately, the presence of hypotension, bradycardia, or both, lead to decreased cerebral perfusion and transient loss of consciousness i.e., syncope.

Treatment must, therefore, address one or more components of this reflex loop. Treatment may be preventive, such trigger avoidance; modulation of the ANS with tilt-training; or blunting the vasodilatory response by optimization of the hemodynamic reserve with hydration or compression stockings. In addition, when a predominant cardioinhibitory mechanism is found, treatment options also include blunting of the effector loop by cardioneuroablation or treatment of the effector response by cardiac pacing. Exceptionally, the trigger itself may be subject to specific treatment, as was the case in this patient.

Although glossopharyngeal neuralgia is an exceedingly rare diagnosis and not part of the differential diagnosis in recurrent unexplained syncope, bradycardia and syncope have been described due to the glossopharyngeal vagal reflex. As the authors elegantly demonstrated, effective control of the somatic trigger led to disappearance of symptoms and most likely to prevention of recurrent syncope notwithstanding a pacemaker *in situ*.

Regrettably, not all patients with cardioinhibitory syncope have such a well-defined cause and complete removal of the trigger may be difficult or impossible. In such cases, cardiac pacing is currently a class I recommendation[2] as per the results of the SPAIN[3] and Bio-Sync[4] trials. The emerging field of cardioneuroablation,[5] where direct disruption by catheter ablation of cardiac autonomic ganglia can suppress reflex bradycardia, may lead to further treatment options in a subset of young patients who might not yet want to undergo pacemaker implantation. However, the long-term results of this novel intervention are yet to be established.

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Ultimately, an accurate diagnosis is the fulcrum of medical practice, and successfully treatment of bradycardia with an anticonvulsant medication demonstrates the complex interplay that makes medicine an art as much as a science.

Conflicts of interest

The author has no conflicts of interest to declare.

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