

Letter in relation to “Patients with syncope remaining unexplained after a structured work-up share the characteristics of patients with neurally mediated syncope”

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In the recent paper by Froidevaux and colleagues, a significant percentage of the patients admitted to the emergency department for syncope in two major Swiss centres shared the features of neurally mediated syncope, which usually results in self-limiting bradycardia and/or hypotension [1].

Neurally mediated syncope is at the crossroads of two major clinical fields: neurology (since all the chronological sequences are mediated by a nerve and some of the accompanying symptoms are basically neurological) and cardiology (since most of the clinical effects of vagal reactions are heart rate mediated).

As to a pathophysiological explanation of this phenomenon, the parasympathetic innervation of the heart should be born in mind. The fibres of the right branch of the vagus nerve innervate the sinus node in the right atrium, thus inducing bradycardia, whereas the left branch fibres innervate the atrioventricular node and are responsible for atrioventricular blocks. An overlap in this distribution may occur. Furthermore, parasympathetic fibres innervate the outer layer of blood vessels (adventitia) promoting vasodilation. So, vagal activation typically leads to a drop in heart rate and/or blood pressure [2].

Brain magnetic resonance imaging showed that alterations at the elongated marrow in the central nervous system or cortical atrophy can result in parasympathetic hyperactivity with consequent bradycardia and hypotension [2]. This may occur at any stage of life [3].

In addition to the mentioned head-up tilt test and carotid sinus massage, heart rate variability is also a simple and non-invasive method to check sympatho-vagal balance at the sinoatrial node. The atropine test constitutes another useful tool. The ocular compression test is no longer performed owing to the risk of retinal detachment [2].

Regarding therapy, a personalised (“tailored”) approach is suggested, with drugs that are often administered off-label [2].

Death in patients without underlying cardiac conditions is rare. However, human autopsies revealed that in 10–15% of people who died after falling into water, little or no water was found in their lungs. It implies that their death may have been neurally mediated [2].

I agree that in refractory cases, permanent pacemaker implantation may be considered, although non-responder patients represent a severe challenge. Some exceptional cases of surgical approach were published, including denervation of the sinoatrial node or thoracic vagectomy.

On balance, little is still known about neurally mediated syncope. Froidevaux and colleagues have the credit of having showed its epidemiology, but extended human studies are strongly needed for a better understanding of its mechanisms and related therapies.

Disclosure statement

No potential conflict of interest relevant to this article was reported.

References

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