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EDITORIAL COMMENT

A pallid paroxysmal event in children: it is vagal anoxic seizure, it is treatable, and it is not "epilepsy"

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There is sometimes diagnostic confusion in children presenting with loss of consciousness, sudden falls, or other paroxysmal motor phenomena. In general, the vagal reactivity has received scarce attention in the pediatric literature. Both neurophysiological mechanism and physiopathological data concerning the vagal effects are almost absent from pediatric, cardiological, and neurological textbooks. However, vagal reactivity is at the crossroads of two major clinical fields: first, neurology, due to the fact that all the chronological sequences are mediated by a nerve and that some of the paroxysmal events related to increase of the vagal reactivity are basically neurological; second, cardiology, since most of the clinical effects of vagal reactions are heart rate mediated. Some authors even use the term of neurocardiology to demonstrate the intrinsic involvement of both systems. Recent investigations established that a third road has to be added to the abovementioned crossroads: the respiratory vagal effect which will probably lead to adjusting the terminology to neuropneumocardiology when considering vagal effects [3, 4]. The vagal reflex that has been the most extensively described in the literature is the "Hering-Breuer reflex". It is a vagally mediated respiratory reflex. In its initial description it was demonstrated that sustained lung inflation was able to inhibit inspiration in animals and that the reflex could be abolished by vagotomy. The cardiac effects of a vagal stimulation had been extremely well documented in animal experiments: precise cardiac response has been obtained at titrated electrical stimuli of the vagus nerve. The parasympathic system was considered for many years to be of little, if any, importance in the control of cardiac function.

A great number of histological and physiological studies now sustain the critical importance of the parasympathetic innervation of the heart. Despite the fact that many of these studies and techniques used on animals contributed significantly to a better understanding of the vagal reactivity but they are nevertheless of little use in clinical human studies.

As pointed out by Stephenson [8] almost 30 years ago, up to 25% of patients diagnosed as having epilepsy in childhood have in fact non-epileptic attacks. It is extremely important to avoid making an erroneous diagnosis not only to avoid the label of "epilepsy" but also to stay clear of the side effects of anti-epileptic drugs for those patients who do not need them. Certain of these conditions are quite common and will be encountered by pediatrician or pediatric neurologist during their career. The concept that seizures are not necessarily epileptic may be unusual or strange to some of us.

One of the consequences of specialization of medicine is that the pediatric neurologist tends to see patients with seizures and pediatric cardiologists those with faints. Pediatricians and general practitioners have the advantage of seeing both, the fits and the faints.

The article published in this journal by Bassareo et al. [1] on the use of homatropine methylbromide as anticholinergic medication, in the prevention of vagal hypertonia, is a well-documented evaluation of this drug in one of the most spectacular non-epileptic symptom related to fits and fails. The strength of this article is to provide clear clinical observations of patients with increased vagal reactivity treated by homatropin. The children became significantly better under treatment with less symptoms and, according to the authors, with less associated risk of presenting a sudden infant death syndrome in this age group.

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This article focuses on the clinical symptoms of vagal faints due to overactive vagal activity; the clinical signs and symptoms can vary but some of them are called pallid breath holding spells, venipuncture fit, bathing epilepsy, reflex anoxic seizures, and others. History acquisition is of paramount significance to diagnose reflex anoxic seizures [5]. Prolonged interrogation of bystanders, individual assessment, and family history is of importance. It is equally important to discover if these fits appear to have been following an acute stimulus to initiate the apparent seizure; in some hypervagotonic paroxysmal events a recognizable stimulus is the rule rather than the exception [7]. In some instances video recording of a replication of the event is necessary to witness and determine whether the event was epileptic or related to one of the different forms of anoxic seizures. In some cases this item needs to be clarified by dynamic reproduction of the event. This can be obtained by a standardized ocular compression test.

This test is not very often proposed but literature on the standardization (pressure, timing, and circadian rhythm) is available and was published in several articles in the 1990s [2, 6].

The technique of ocular compression test differs from one author to the other, some attributing more importance to the applied pressure and others to the effect of surprise or to the degree of pain evoked during the procedure. Even the threshold to provoke a clinical anoxic seizure due to vagal reaction can be measured on the duration of the asystole on the ECG (9.7 + 0.7 flat EEG time in seconds). Often this test does not need to be performed and anamnesis, EEG analysis together with ECG can be sufficient to confirm the diagnosis. Many therapeutic approaches have been proposed in the past like atropine, atropine sulfate, transderman scopolamine, and disopyramide. Some exceptional cases of surgical approach have been published including the denervation of the sino-atrial note or thoracic vagectomy. The authors propose here a treatment with homatropine methylbromide as parasympathicolyticum. The good results obtained in this study confirm some of the old studies related to this subject.

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