EDITORIAL

Evaluation of the Behavior of the Time Interval from the Onset of the QRS Complex to the Onset of Radial and Carotid Pulse Waves with the Result of the Tilt Test

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In the 1980s, the first findings on the effect of prolonged orthostatic exposure in patients with unexplained syncope caused excitement among physicians, and generated great expectation on the possibility that a method could detect susceptible individuals and reveal the mechanisms of the reflex syncope, hitherto virtually unknown. The reflex or neurally mediated syncope is the main cause of syncope in the general population, and despite it has a good prognosis, it may compromise life quality of those affected by it. (1)

An adequate orthostatic tolerance depends on the association of several cardivoascular and cerebrovascular regulatory mechanisms, and the reflex syncope represents a sudden and severe failure in them. Immediately after an individual adopts a standing position, the stroke volume remains stable despite the decrease in venous return, probably because of the volumen of blood remaining in the pulmonary circulation. Gradually, within minutes, the fall in blood filling and cardiac output causes changes in the (cardiopulmonary and carotid) receptor activity of low and high pressure, which trigger an increased sympathetic influence on the cardiovascular system. The result is increased heart rate, cardiac inotropism, and splanchnic and systemic vasoconstriction. Remaining in a standing position then causes the release of other neurohormonal factors, like renin-angiotensin-aldosterone (RAA) and vasopressin. (2)

The coordination or failure of all these mechanisms determine the individual's orthostatic tolerance. It is known that the greater the depletion of blood volume, the greater the activation of RAA system, and that alterations in the systemic arterial and cerebral compliance interfere with orthostatic circulatory compensation. Other factors indirectly related to the baroreflex also influence on circulatory compensatory mechanisms, such as age and use of drugs with cardiovascular effects.

Over the past 20 years, a lot has been learned about the hemodynamic and neurohumoral factors involved in the pathophysiology of the vasovagal syncope, due to the studies performed on patients with syncope induced by the tilt testing. However, despite its position –which is already established in the diagnostic research of syncope– the tilt test, as well as other subsidiary methods, shows limitations regarding its diagnostic capacity, probably due to the variety of triggering factors and the complex elements involved in the neurally mediated reflex.

If we consider those patients susceptible of vasovagal syncope with no other comorbidities, it is believed that redistribution of blood and restoration of the central blood volume during orthostatism is particularly affected by limited vasoconstrictor reserve. This mechanism has been demonstrated in studies that analized the muscle-nerve sympathetic activity and the vasoconstrictor capacity on patients exposed to negative pressure chamber in the lower body, pharmacologically sensitized with vasoactive drugs. (3)

In their study, Villamil et al. (4) propose a new method for the assessment of hemodynamics during tilt testing, which would allow for early identification of individuals prone to vasovagal reflex, induced by the exposure to prolonged standing and early termination of the test. They consider that the induction of syncope during the test is an unpleasant experience for the patient, but this statement is questionable. In our experience, the fact that the patient experiences the symptoms during the test has not been a negative point but a relieving one, because of the certainty of the diagnosis and the prospect of an adequate treatment. On the other hand, the existing autonomic monitoring systems used during the tilt test analyze not only blood pressure and heart rate: through a bioimpedance technique and a special software, hemodynamic parameters like peripheral resistance, cardiac output, stroke volume,RR variability, and baroreflex sensitivity have shown different mechanisms during presyncopal period in response to different kinds of neurally mediated syncopes.

The use of autonomic observation provides a deeper understanding of the pathophysiology of the reflex syncope, and by applying the concept of individualized therapy for each type of mechanism, a more specific treatment for each patient can be administered. (5)

The analysis of the delay of the radial and carotid pulse wave in relation to QRS is certainly a non-invasive method easy to perform, which could be included in the hemodynamic parameters during tilt test to as-

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Revista Argentina de Cardiología / _vd _77_n"_5 / sip eimbii_dc ubii_2009

sess patients with syncope. However, due to the reasons stated above, we do not consider that the methods should be exclusive. The delay can be interpreted as the result of the failure of several possible stages in the central and peripheral regulatory mechanisms during the ejection or pre-ejection period, emerging in the first minutes of orthostatic exposure and interfering with circulatory compensation. Its real meaning is still unclear.

On the other hand, we cannot fail to mention the clinical characteristics of the studied population, which differ from those in most patients with neurally mediated syncope, who are usually young and do not present comorbid conditions. Older age, hypertension, and use of antihypertensive drugs may influence considerably on stroke volume, baroreflex response, arterial compliance, and peripheral resistance, and may determine hemodynamic responses which are different from the usual ones. The study lacks a control group, a fact that the authors consider a limitation of this pilot study, but that, in their opinion, will be solved once the next study in progress is over.

Thus, we believe the proposed method is promising, especially when analyzed with other hemodynamic measures and tested in larger populations, due to the possibility of providing more information about the pathophysiology of orthostatic intolerance.

However, it seems premature to suggest that this methodology is enough to identify patients susceptible to vasovagal syncope, and do without conventional tilt test.

We believe that the objectives of the studies on neurally mediated syncopes, with their methods for autonomic assessment, should be directed not only to identify susceptibles, but also to elucidate the mechanisms involved, and to offer solutions.

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