VIA MEDICA

ORIGINAL ARTICLE

Folia Morphol.
Vol. 62, No. 3, pp. 175–178

ISSN 0015–5659
www.fm.viamedica.pl

Left ventricle systolic volume in vasovagal syncope patients

Dariusz Kozłowski[1], Paweł Byrdziak[1], Wojciech Krupa[3], Marcin Gawrysiak[1], Grzegorz Piwko[2], Jacek Kubica[3], Grażyna Świątecka[1]

[1]*Second Department of Cardiac Diseases, Medical University of Gdańsk, Poland*
[2]*Department of Clinical Anatomy, Medical University of Gdańsk, Poland*
[3]*Department of Cardiology and Internal Medicine, Medical University of Bydgoszcz, Poland*

[Received 6 May 2003; Accepted 2 June 2003]

One of the hypotheses put forward concerning the mechanism of vasovagal syncope is that the vagal afferent fibres are activated during vigorous contractions against a partly empty left ventricle. The aim of the study was to confirm this hypothesis by using 2D echocardiography during a head-up tilt test. The study was carried out on 39 patients (17 male, 22 female, age range 21–64 years), all with a history of recurrent syncope. The patients were examined using a 2D echo to measure the end-diastolic and end-systolic volume before the head-up tilt test after the Westminster protocol (45min/60 grade) and every five minutes after tilting. T patients during head-up tilt test had a positive response and 32 proved negative. A reduction of both the end-diastolic and end-systolic volumes of the left ventricle was noticed. There was no significant difference in the degree of ejection fraction reduction. The difference in ejection fraction reduction between the two groups was similarly non-significant. It was also noticed that the patients with a positive response had more vigorous contractions than those with a negative test. The decision was therefore taken to use a different parameter for the left ventricle contraction, namely the LV posterior wall slope. As this parameter is partly dependent on time, its use in confirming the extremely vigorous nature of the contractions was considered appropriate. Only 6 patients were tested using this parameter. A tendency towards greater left ventricle posterior wall slope values, both before and during tilting was noticed in the group of patients with vasovagal reaction. Our data shows that vigorous contraction is probably less responsible for vasovagal syncope release than left ventricle volume reduction.

key words: LV systolic volume, vasovagal reaction, LV posterior wall slope

INTRODUCTION

Vasovagal syncope is one of a group of reflexive syncopes. One of the hypotheses explaining the mechanism for the occurrence of the vasovagal syncope is a paradoxical over-reactive response of the parasympathetic autonomic nervous system.

The non-mielin afferent fibres of the vagal nerve have their origin in receptors localised in the wall of the left ventricle and atrias. The reflexive reaction relies on inhibition of efferent sympathetic heart fibre activity and stimulation of the fibres of the vagal nerve running toward the heart [1] .The

Address for correspondence: Dariusz Kozłowski, II Department of Cardiac Diseases, Medical University of Gdansk, ul. Kieturakisa 1, 80–742 Gdańsk, Poland, tel/fax: +48 58 349 39 10, e-mail: dkozl@amg.gda.pl

incitement of these mechanoreceptors is mainly caused by changes in the pressure in the left ventricle (e.g. stretching of the left ventricle). From the physiological point of view, it is a positive phenomenon, an inhibiting heart-to-heart reaction that decreases heart work in circumstances of overload [5].

Nevertheless, in the case of vasovagal syncope the vertical position and redistribution of blood leads to a diminution of blood volume in the central nervous system with secondary adrenergic stimulation, which is suspected of evoking the rapid systoles of the scarcely filled LV, incitement of the previously-mentioned mechanoreceptors and, secondary to this, the reflexive response finally leading to syncope [1, 2, 10]. The purpose of this study was to determine the degree of left ventricle volume reduction in patients suffering from vasovagal syncope in comparison with the group without vasovagal syncope and to assess left ventricle systolic function during the head-up tilt test in both groups.

MATERIAL AND METHODS

The study was carried out on 39 people aged 21––64 years old, 17 men and 22 women, with recurrent syncopes in their history. All of them underwent the head-up tilt test after the Westminster protocol (45 min/60 degrees). The pressure was taken during the test with a sphygmomanometer, ECG and echocardiographic recordings on VHS tape (HP Sonos 2000 and Sonos 1000, 2.5 MHz head). The echocardiographic examination was carried out at the beginning, in the horizontal position, directly after the tilting and every 5 minutes until the end of the examination and finished because of the syncope or the completion of the procedure for the passive test. The VHS echocardiographic recordings were analysed after the head-up tilt test .The image acquired just before or during the syncope was hardly legible because of hyperventilation and thus could not be interpreted. The last recording of sufficient quality preceding the syncope was used for interpretation.

The end-diastolic and end-systolic volumes were evaluated in the apical four-chamber projection. The volume of the left ventricle concomitant with the Q wave in ECG was the end-diastolic one and the end-systolic volume was appraised at the moment of maximal movement of the interventricular septum toward the posterior wall of the left ventricle, which corresponded to the end of the T wave in ECG. It was noticed that in patients with vasovagal syncope the systoles of the left ventricle were more violent and this inclined us to expand the list of parameters observed to include the slope of the posterior wall of the left ventricle (LVPW-slope) in M-mode using parasternal, long axis projection. The measurement was taken before the test and every 5 minutes until its end. This particular parameter considers time as a significant element of systole rapidity estimation.

RESULTS

During the head-up tilt test 7 out of 39 patients experienced syncope that could be diagnosed as a vasovagal syndrome (4 — mixed type, 1 — cardiodepressive type, 2 — vasodepressive type). There were no significant differences in the reduction of end-diastolic and end-systolic volumes of the left ventricle in either the group with a positive result of the test, e.g. with syncope during the head-up tilt test, or the group without the syncope (Fig. 1). No significant dissimilarity was additionally observed between the groups in the various types of syncope. However, it was noticed that in the group of patients with a positive result of the head-up tilt test a tendency exists ($p > 0.05$) to reach larger LVPW-slope values (Fig. 2). This is present even in the preliminary examination before the test.

During the test we also observed an increase in the ejection fraction in all the groups examined (Fig. 3).

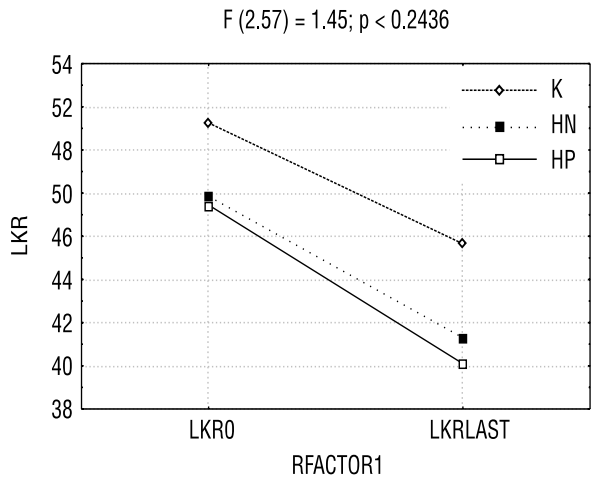

Figure 1. The velocity of left ventricle end-diastolic volume reduction during the head-up tilt test; LKR — left ventricle end-diastolic volume, LKR0 — at the beginning of the head-up tilt test, LKRLAST — at the end of the head-up tilt test, HP — patients with syncope in the history and during the test, HN — patients with syncope in the history and without syncope during the test, K — control group (patients without syncope in the history and within the test).

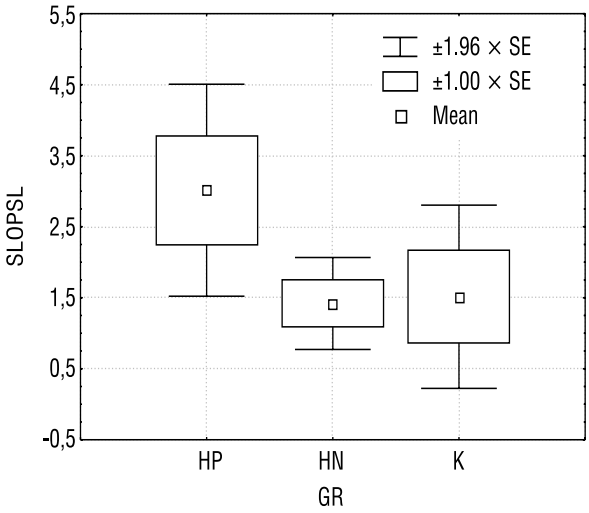

Figure 2. The increase of the left ventricle posterior wall slope during the head-up; SLOPSL — left ventricle posterior wall slope, HP — patients with syncope in the history and during the test, HN — patients with syncope in the history and without syncope during the test, K — control group (patients without syncope in the history and within the test).

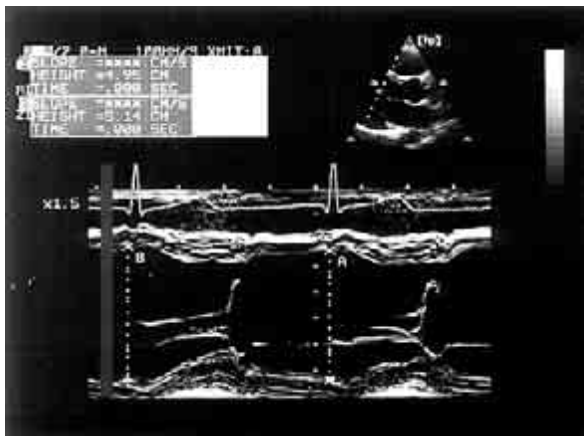

Figure 3. Increase of the ejection fraction during the head-up tilt test.

DISCUSSION

During the tilt test the input volume of blood reaching the heart is decreased due to its displacement to the lower parts of the human body. As a result of this phenomenon, strongly dependant on gravitational force, diminution of the end-diastolic volume of the left ventricle occurs. In accordance with one of the hypotheses explaining the trigger mechanism of reflexive reaction of the vagal nerve, such en effect is intensified in the group of patients suffering from vasovagal syncope [1, 10]. Thus, a more visible reduction of end-diastolic volume of the left ventricle should be observed in patients that experienced the vasovagal syncope during the test. We did not find any corroboration of such a difference in the degree of left ventricle end-diastolic volume reduction in groups with a positive result of the tilt test and with a negative result of the head-up tilt test. Neither did we manage to confirm any significant disparity between left ventricle end-systolic volume reduction degree in the group with a positive result of the test in comparison with patients without the syncope during the procedure.

The studies that have been carried out hitherto used different methods to estimate left ventricle filling and its contractility. Fitzpatrick et al. [4] evaluated the end-diastolic and end-systolic dimensions of the left ventricle in M-mode echocardiography. His results, compared to those for the control group, suggest a more intense reduction in the end-diastolic and end-systolic dimensions in the group of patients with vasovagal syncope during the test. Only two cases from the control group were analysed in that study.

On the other hand, Shalev et al. [8], who was the first to evaluate the changes in left ventricle dimensions and left ventricle function during the head-up tilt test with the use of 2-dimensional echocardiography, observed more lively contractility of the left ventricle and a significant reduction in the end-systolic dimension in the group of patients with a positive test in relation to the control group .

When Yamanouchi et al. [9] compared 7 patients with a positive result of the head-up tilt test to a group of 9 volunteers without any incidence of the syncope in their history there was no statistically significant difference between the two groups in the changes of left ventricle end-diastolic and end-systolic volume ratio. However, the dynamics of the reduction of left ventricle end-diastolic volume ratio and of the left ventricle ejection fraction was intensified during the tilt test in the group of patients with the vasovagal syncope. In their opinion the speed of left ventricle end-diastolic volume reduction can be important in stimulating the vasovagal reaction. The more pronounced reduction of the left ventricle ejection fraction in the group with the vasovagal syncope could also be a result and early response of the vagal nerve to the contractility of the left ventricle.

In contrast, Lee et al. [6] did not discern any dissimilarity in end-systolic and end-diastolic dimension between groups with positive and the negative results of the head-up tilt test. Nevertheless, he observed a significant increase in the shortening fraction and concluded that a lively systole of the left ventricle, not the contraction of the unfilled ventricle, is responsible for triggering the vasovagal reflex.

Liu et al. [7] compared the left ventricle volume and the shortening fraction of the left ventricle in patients during the tilt test (the parameters were registered before and during the first 5 minutes in a vertical position). They observed a reduction in both left ventricle volume and shortening fraction in the preliminary period of vertical position in patients with a positive result of the test. They suggested that if there were any paradoxical activation of the mechanoreceptors in left ventricle stimulation of the reflexive syncope, this would not be caused by excessive contractility of the left ventricle.

Deharo et al. [3], using a microaccelerometer at the end of an electrode placed in the right ventricle, found an increase in left ventricle contractility during the head-up tilt test. As can be seen, different studies have produced varying results.

The hypothesis explaining the stimulation of mechanoreceptors of vagal nerve C fibres in the left ventricle emphasises the rapidity of the systoles of the hardly filled left ventricle as the factor responsible for stimulation of the mechanoreceptors [10]. In our opinion none of the hitherto analysed parameters describing the contractility of the left ventricle i.e. left ventricle ejection fraction, shortening fraction and degree of end-systolic dimension reduction, takes time into consideration as the indispensable parameter for the evaluation of the rapidity of the systole. We therefore decided to introduce an additional parameter of echocardiographic examination of the left ventricular function during the tilt test, the slope of the left ventricular posterior wall movement (LVPW-slope). In LVPW-slope, time is considered as a parameter and, in our opinion, enables the rapidity of the systole to be evaluated, despite the fact that this has not been used in any published paper that was accessible for us. However, the number of patients examined who showed a positive result of the test is too small to allow any conclusions to be drawn. Nevertheless, there is a tendency towards higher values of LVPW-slope in the group of patients with positive test results and, interestingly, this is noticeable even in the examination preceding the patient's head-up tilt.

It seems that stimulation of the mechanoreceptors causing the paradoxical over reactive response of the parasympathetic system can occur during rapid systoles of quite a well filled and not, as was previously suggested, an unfilled left ventricle. The tendency to more rapid systoles of the left ventricle in patients with vasovagal syncope registered just before the test can be linked to the stronger sensitivity of these patients later to adrenergic stimulation. This can be conducive to the paradoxical reflexive response of the vagal nerve. However, there is a need for further studies with a larger group of patients suffering from vasovagal syncope compared to the control group of patients without any history of syncope.

REFERENCES

1. Abboud FM (1993) Neurocardiogenic syncope. N Engl J Med, 328: 1117–1120.
2. Benditt DG, Erickson M., Gammage MD, Markowitz T, Sutrton R (1997) A synopsis: neurocardiogenic syncope, an international symposium. Pacing Clin Electrophysiol, 20: 851–859.
3. Deharo JC, Peyre JP, Chalvidan T, Thirion X, Valli M, Ritter P, Djiane P (2000) Continuous monitoring of an endocardial index of myocardial contractility during head-up tilt test. Am Heart J, 139: 1022––1030.
4. Fitzpatrick AP, Theodorakis G, Vardas P, Sutton R (1991) Methodology of head-up tilt testing in patients with unexplained syncope. J Am Coll Cardiol, 17: 125–130.
5. Mark AL (1983) The Bezold-Jarish reflex revisited: clinical implications of inhibitory reflexes originating in the heart. J Am Coll Cardiol, 1: 90–102.
6. Lee TM, Chen MF, Su SF, Chao CL, Liau CS, Lee YT (1996) Excessive myocardial contraction in vasovagal syncope demonstrated by echocardiography during head-up tilt test. Clin Cardiol, 19: 137–140.
7. Liu JE, Hahn RT, Stein KM, Markowitz SM (2000) Left ventricle geometry and function preeceding neurally mediated syncope. Circulation, 101: 777–783.
8. Shalev Y, Gal R, Tchou PJ, Anderson AJ, Avitall B, Akhtar M., Jazayeri MR (1991) Echocardiographic demonstration of decereased left ventricle dimensions. J Am Coll Cardiol, 18: 746–751.
9. Yamanouchi Y, Jaalouk S, Shehadeh AA, Jaeger F, Goren H, Fouad-Tarazi FM (1998) Changes in left ventricular volume during head-up tilt in patients with vasovagal syncope: an echocardiographic study. Am Heart J, 131: 73–80.
10. White CM, Tsikouris JP (2000) A review of pathophysiology and therapy of patients with vasovagal syncope. Pharmacotherapy, 20: 158–165.