THE VALSAVA MANEUVER IN CHAGASIC PATIENTS WITH LEFT VENTRICULAR SEGMENTAL WALL MOTION ABNORMALITIES AND NORMAL EJECTION FRACTION.

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Arterial pressure response during Valsalva maneuver (VM) is abnormal in patients with symptomatic cardiac failure and depressed ejection fraction (EF). Asymptomatic patients with Chagas' disease present abnormal segmental wall movement (SAC) with normal EF. In order to evaluate the arterial pressure response to VM with respect to segmental wall motion, we studied asymptomatic patients with Chagas' disease with SAC compared to normal controls. Thirty five percent of the patients with SAC presented abnormal responses. Our analysis showed a statistically significant relation ship ($p < 0.05$) between abnormal responses and SAC. VM could be useful in detecting incipient left ventricular disfunction

The normal arterial blood pressure response to the Valsalva Maneuver (VM) consists of an initial rise associated with the onset of straining (phase 1) followed by a sharp fall under baseline levels as the straining is maintained (phase 2). Release of strain (phase 3) is followed by an overshoot of the arterial pressure (phase 4). In this way a typical sinusoidal response is presented [1] (fig. 1A).

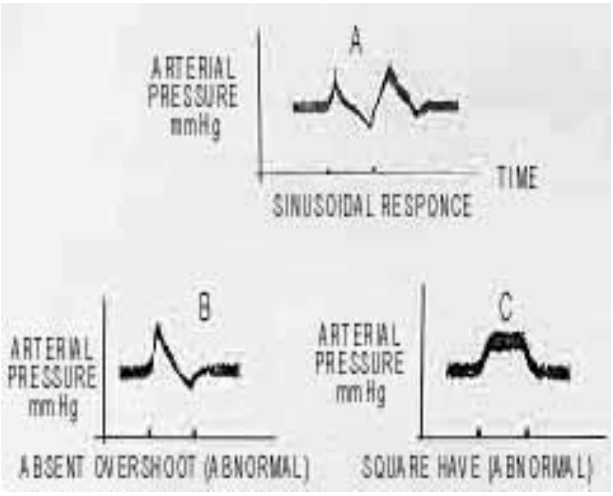

Fig. 1 - Arterial pressure responses to Valsalva Maneuver. The pressor phase is indicated between the marks in the horizontal axis. For description of responses see text.

Several explanations have been proposed for this phenomenon (increase in venous return, variations in sympathetic tone, afterload and contractility, etc.) but, so far, how and how much each of these factors intervenes remains unknown [2].

Whatever the explanation is, it has been demonstrated that in patients with overt congestive heart failure (CHF) the arterial pressure did not fall during phase 2 of VM[3]. Recently Zema et al. found two types of behavior which are in close relationship with a depressed ejection fraction (EF) and an increase in left ventricular end diastolic pressure (LVEDP): absent overshoot during phase 4 (fig. 1B) and a sustained elevation of arterial pressure during phase 2 which subsequently falls to control level without overshoot they called this phenomenon a "square wave response" (fig. 1C) [4-6].

In Venezuela chronic chagasic myocarditis (CCM) is a common cause of left ventricular disfunction and CHF. Carrasco et al. who studied the natural history of this disease, identified a subgroup of patients in the initial stages of the illness who have neither symptoms nor any abnormality detectable by non-invasive means. But in the ventriculogram these patients showed segmental alteration of left ventricular contractility (SAC) with normal EF and LVEDP[7].

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Zema et al. have the usefulness of VM in detecting left ventricular disfunction in patients who presented illnesses - other than chronic chagasic myocarditis - with overt cardiac failure, depressed EF and elevated LVEDP [4-6].

Our search of the literature did not reveal any previously reported of VM in patients with SAC, normal EF and LVEDP.

The present study was then carried out in order to investigate the response to VM in patients with CCM and SAC but with normal EF and LVEDP.

MATERIALS AND METHODS

Thirty two chagasic patients were studied and classified in the following groups after Carrasco's model [7]. They all signed a written consent before participating in the study Group 0: patients with a positive Machado-Guerreiro test who did not present any clinical nor paraclinical evidence of myocardial damage (i. e., the endomyocardial biopsy did not reveal any abnormality); Group 1A: patients whose endomyocardial biopsy shows ultrastructural damage but who do not present any evidence of cardiac damage in the neither non-invasive nor the invasive studies. Group 1B: Asymptomatic patients whose non-invasive cardiovascular explorations produces normal results and whose cineangiographic study reveals SAC or alteration of other hemodynamic parameters; Group 2: patients with arrhythmias and SAC, who already present alterations in the surface electrocardiogram but do not show signs of CHF; Group 3: patients who have or had signs of overt CHF, whose EF is depressed and who have dilated hearts.

The control was 1A as they presented a positive Machado-Guerreiro test, showed evidence of myocardial damage in the biopsy but no evidence of functional alteration. This makes it possible to compare these patients with those who do present alterned function in terms of their response to the VM. If the arterial pressure response to VM is different between control and patients, then the difference could be attributed to the functional damage and not to the CCM per se.

The VM was performed at the patients bedside while they rested in a quiet room. The patients were intructed to take a deep breath followed by a 10-second straining phase. At the physician's instructions they resumed quiet breathing. Following the procedure described by Zema et al. [4-6], the systolic blood pressure was always obtained while the patient was breathing quietly in the supine position. The cuff pressure was then raised 15 mmHg above the systolic pressure and the patient was asked to perform the VM. The effectiveness of the procedure was assessed by noting if the patient developed a florid face, distended neck veins, and increased abdominal wall muscle tone. While the cuff pressure was held inflated 15 mmHg above the previously determined systolic pressure during the straining phase of the VM, and for 15 seconds afterwards, the Korotkoff sounds were sought by auscultation over the brachial artery. Korotkoff sounds were heard at the initiation of the straining phase in all three types of pressure responses. In both the sinusoidal and absent overshoot responses these sounds were not perceived during the last part of straining phase of VM. In the square wave response, however, the Korotkoff sounds were heard for the entire duration of the straining phase. Patients with a sinusoidal response had return of Korotkoff sounds (overshoot) approximately there to 10 seconds after relaxation.

The VM was always assessed independently by at least two of the authors in a blind fashion. No discrepancy in the results was observed.

The correlation between the systolic blood pressure pattern during VM and SAC, the presence of arrhythmia or conduction disturbance, EF and left ventricular volumes, were analyzed by means of a Chi square test. An alpha value of 0.05 was assigned as the level of statistical significance.

The one-way variance analysis was used to compare the groups about basal arterial pressure, EF and left ventricular volumes.

All the patients had a complete haemodynamic study (including coronariography, left and right cineventriculography, recordings and right endomyocardial biopsy) before performing the VM. The mean time which elapsed between the VM and the above mentioned study was 57 months.

The patients clinical state was always assessed by one of the authors before performing the test. This permitted us to classify the patient in the above mentioned groups.

RESULTS

The VM did not give rise to any complication nor untoward effect for the patients.

In terms of their arterial pressure response to VM the 32 patients were distributed as follows: 9 patients in group 1A who had a sinusoidal (i. e., normal) response to VM: 9 patients in group 1B, 7 of whom had sinusoidal responses and 2 absent overshoot (i. e., abnormal responses), and 14 patients overshoot responses, and 2 square wave responses (table I).

TABLE I - Arterial pressure response to Valsalva maneuver by groups.

Group	Responses					
	Patients		Normal		Abnormal	
	N.º	%	N.º	%	N.º	%
1A	9	(100,0)	9	(100,0)	-	-
1B	9	(100,0)	7	(77,8)	2	(22,2)
2	14	(100,0)	8	(57,1)	6	(42,9)
Total	32	(100,0)	24	(75,0)	8	(25,0)

We observed a clear trend towards abnormal responses in patients classified in the more advanced groups, i. e., the greater the myocardial damage, the more frequent the abnormal responses (fig. 2). In group 1A, in which there was only ultrastructural damage we did not find any abnormal response. In the patients of group 1B, who had ultrastructural damage plus SAC, 22% of the responses were abnormal. Finally, in the patients of group 2, who had ultrastructural damage, SAC and arrhythmias or conduction disturbances, 42% of responses were abnormal

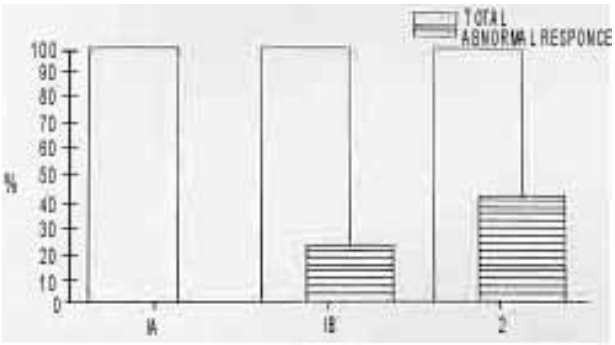

Fig. 2 - Percentage of abnormal responses in different groups of patients.

When comparing these three groups by means of a one-way variance analysis, differences with respect to EF and left ventricular volumes were found ($p < 0.05$). In other words, group 1A is normal in terms of function; group 1B only shows SAC. greater volumes and less EF, and group 2 exhibits SAC, greater volumes and less EF than group 1B and presents arrhythmias or conduction disturbances. In the three groups, in spite of the statistically significant difference between EF and ventricular volumes, the mean values of these parameters were still within normal limits (table II). No statistically significant difference was found by means of two-way variance analysis between the groups in terms of basal arterial pressure and stroke volume was found. However, a statistically significant difference was found in EF. ($p < 0.01$), end systolic volume ($p < 0.01$) and end diastolic volume index ($p < 0.05$).

The common factor between group I B and group 2 is SAC. This account for the difference between these groups (1B and 2) and group 1A which does not present functional abnormality.

In group 1A all the responses to VM were normal while in groups 1B and 2, 1/3 had abnormal responses (absent overshoot and square wave).

When comparing VM response with other variables, a positive correlation was found only in the presence of SAC which has a statistically significant association with an abnormal response ($p < 0.5$).

No statistically significant association was found between abnormal responses to VM and EF, left ventricular volumes conduction disturbances and arrhythmias (for all of them $p > 0.1$).

TABLE II - Mean values of ejection fraction and left ventricular volumes by groups.

Group	Ejection fraction	end diastolic vol. (index ml m^2 BS)	end systolic vol. (index ml m^2 BS)
1A	0.68	96	31
1B	0.62	115	43
2	0.52	135	55

Vol. = volume, BS = body surfaces.

DISCUSSION

It is evident that the VM is a simple test for both the physician and the patient. It does not require the use of any expensive equipment, it is reproducible, practical, and reliable because its correlation with intrarterial register is excellent as was demonstrated by Zema et al. [4].

Our findings have not been previously reported in the literature. This is due to the fact that we studied patients with an illness which has peculiar features. i. e., asymptomatic patients who have an abnormal contractile function and a normal EF. Such a state is difficult to identify in other cardiopathies such as ischaemic or dilated cardiomyopathy because, while the patient is asymptomatic, there is no clinical marker of the illness. In chronic chagasic myocarditis the Machado Guerreiro test permits the identification of the patients in early stages of the illness and their study in terms of myocardial damage.

In our group of patients, and in agreement with previous reports, we would expect normal responses to VM in all patients. In other words. we were expecting to obtain a sinusoidal response in all of our patients but this did not happen in 35% of them. This is mot to be expected from Zema's and others results [3-6,8].

Our findings suggest that the abnormal response to VM is not only the results of an important ventricular disfunction as that expressed in an abnormal EF or a dilated heart. but also of the presence of abnormal segmental contractility. Indeed, the only variable associated with an abnormal response to VM was the presence of SAC although the groups manifested significant differences regarding EF and cardiac size.

Little et al. found that the left end diastolic volume did not change during phase 2 of VM and that it kept in correlation with a square wave response in echocardiograms of patients with depressed EF and dilated hearts [9].

In patients with overt CHF the increased pulmonary vascular volume would not permit the diastolic filling to be reduced during phase 2. This, in turn, would not allow the cardiac index to fall, and consequently, the arterial pressure would remain elevated during this phase. This would explain, at least in part, the square wave response observed in patients with dilated hearts, depressed EF and CHF, but it could not explain such a response in our patients.

Labovitz et al. [10] studied patients with ischemic heart disease and dilated cardiomyopathy and com pared them with normal controls. All of them were submitted to left ventriculography and coronariography before and during the VM. They found that the segmental contractility was increased during VM in the patients with normal EF. Five of the patients presented a square wave response and an abnormal EF. Augmentation of the segmental contractility with VM did not occur in these patients, although only one of them failed to show a reduction of end diastolic and systolic volumes. This contradicts Little's finding and does not permit the explanation of the abnormal responses [10]. Thus, the mechanisms responsible for the responses to VM are still unknown.

In our study we failed to demonstrate any relationship between the response to VM and the presence of arrhythmias or conduction disturbances.

On this basis we are inclined to think that the abnormal segmental contractility without neither de pressed EF nor elevated LVEDP bears a direct relationship to the production of an abnormal response to the VM in chagasic patients.

Our work compares the responses to the VM with the hemodynamic state of the patient which was determined before performing the VM (mean 57 months). Between the time when the hemodynamic study was performed and the VM, the patient could have passed from group 1A to group 1B. But the findings of Espinoza et al. [11] do not support such an hypothesis. In deed, they showed that 94% of the patients from group 1A still belonged to the same group when they were submitted to a mew hemodynamic study 40 months after the first catheterism.

For the patients in the other groups the evolution from group 1B to group 2 could be detected by the initial evaluation performed before the VM.

In conclusion, the arterial pressure response during the VM could be a very simple and useful means for detecting and following the patients with chronic chagasic myocarditis. It is a non-invasive method available to any physician worldwide and it could help in identifying some asymptomatic patients in the initial stages of chronic chagasic myocarditis as well as other illnesses with similar behavior (for example, dilated cardiomyopathy, silent ischemic heart disease, etc.).

RESUMO

A resposta da pressão arterial durante a manobra de Valsalva é anormal em pacientes sintomáticos com insuficiência cardíaca e fração de ejeção diminuída. Pacientes chagásicos assintomáticos podem apresentar movimentos segmentares anormais da parede ventricular, com fração de ejeção normal.

Para avaliar a resposta pressórica durante a manobra de Valsalva em relação ao movimento segmentar, foram comparados pacientes chagásicos e indivíduos normais.

Trinta e cinco por cento dos pacientes chagásicos com SAC apresentaram resultados anormais, que foram estatisticamente significativos ($p < 0,05$). A manobra de Valsalva pode ser útil na detecção de disfunção ventricular esquerda incipiente.

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