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Heart rate changes during the Valsalva maneuver in patients with isolated aortic insufficiency

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Abstract

To determine the possible relationship between left ventricular dilatation and heart rate changes provoked by the Valsalva maneuver (Valsalva ratio), we studied 9 patients with isolated chronic aortic insufficiency. Left ventricular systolic function was assessed by two-dimensional echocardiography and cardiac catheterization. All patients were asymptomatic (functional class I of the New York Heart Association). The left ventricular internal diameters and volumes were significantly increased in all patients. The asymptomatic patients had either normal or slightly depressed ejection fraction (EF>0.40). The Valsalva ratio of these asymptomatic patients showed no significant correlation with the left ventricular volumes or with the left ventricular ejection fraction. In other words, parasympathetic heart rate control, as expressed by the Valsalva ratio, was normal in the asymptomatic patients with left ventricular dilatation and preserved left ventricular ejection fraction. Therefore, left ventricular dilatation may not be the major mechanism responsible for the abnormal parasympathetic heart rate control of patients with acquired heart disease.

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Introduction

Patients with congestive heart failure have cardiac parasympathetic abnormalities and neurohormonal activation (1-3). Cardiac parasympathetic heart rate control, as assessed by the heart rate changes during the Valsalva maneuver, is impaired in these symptomatic patients (4-6). The mechanism(s) responsible for these abnormalities is (are) unknown (7-9). It has been postulated that left ventricular dilatation may be one of the responsible mechanisms (10).

In patients who have suffered a myocardial infarction (11) and in most patients with dilated and chagasic cardiomyopathy (12,13), progressive ventricular dilatation takes place on a left ventricle with segmental wall motion abnormalities. Most of these patients, although still asymptomatic, have abnormal heart rate responses during the Valsalva maneuver (14-16).

Chronic aortic regurgitation also provokes a progressive dilatation of the left ventricle (17). Chronic volume overloading usually takes place in a previously healthy myocardium (18,19). This well-established fact distinguishes the left ventricle of chronic aortic insufficiency from that of most other cardiac diseases (20). Therefore, aortic insufficiency

offers a unique opportunity to study the effects of varying degrees of ventricular dilatation on the heart rate changes occurring during the Valsalva maneuver.

In the present investigation, we postulate that if cardiac parasympathetic heart rate control, as assessed by the heart rate changes during the Valsalva maneuver, is normal in asymptomatic patients with chronic aortic insufficiency, left ventricular dilatation may not be the major mechanism responsible for the cardiovascular parasympathetic abnormalities.

Patients and Methods

Nine patients with a clinical diagnosis of isolated chronic aortic regurgitation, no past medical history of rheumatic fever and no serologic evidence of syphilis were referred to our Institute because of the presence of a heart murmur. The clinical diagnosis of isolated aortic regurgitation was based on the presence of a high-pitched diastolic murmur along the left sternal border (21). This diagnosis was confirmed by cardiac catheterization and ventricular cineangiography. The severity of the aortic valve incompetence was established according to Grossman and Dexter (22). Patients with other cardiac, metabolic or systemic diseases were excluded. Eleven age-matched healthy sedentary subjects were used as normal controls.

Clinical and echocardiographic protocol

After informed consent was obtained, all patients had their clinical history taken and were submitted to a routine laboratory work-up, chest X-ray and M-mode and two-dimensional echocardiographic evaluation. The size of the left cardiac chambers was determined as follows: patients were positioned in a shallow left lateral decubitus. We used a Hewlett-Packard 77020-Ac two-dimensional echocardiographic instrument to measure areas and lengths and to estimate volumes. A 2.25 or 3.25 MHz transducer was used. The left ventricular and atrial internal diameters were measured by M-mode and two-dimensional echocardiography. A cursor was placed on the diameter of the chamber of interest in the parasternal short-axis or long-axis two-dimensional display of the heart (23).

Cardiac autonomic function tests

On the same day of the echocardiographic study, the patients were taken to a quiet semidarkened room and allowed to rest for 30 min in the supine position. The following sequence of cardiac autonomic function tests was performed.

Valsalva maneuver. The patients and the control subjects were instructed to maintain an expiratory pressure of 40 mmHg for 10 s by means of forced expiration into a mouthpiece connected to a sphygmomanometer. The EKG was recorded during the maneuver and 15 s thereafter. The Valsalva ratio was calculated by dividing the maximum heart rate observed during the maneuver by the minimum heart rate after the maneuver (24).

Standing up. The patients stood up after a verbal command. The maneuver was performed over a period of 2-3 s and the patients remained upright for 30 s. The EKG was recorded before, during and after the maneuver. The 30/15 ratio was calculated by dividing the minimum heart rate after the 30th beat by the maximum heart rate at the 15th beat (25).

Facial immersion. The patients were instructed to immerse their faces in a water basin at 18-20^{o}C, after a normal inspiration. The maneuver was performed over a period of 20-30 s, while the subjects were sitting in front of a table. The magnitude of the bradycardic response was estimated by subtracting the minimum heart rate observed from the baseline heart rate (26,27).

Both the echocardiographic evaluation and the cardiac autonomic function tests

were performed on all patients within 6 months of cardiac catheterization.

Statistical analysis

The left ventricular function parameters and the results of the cardiac autonomic function tests obtained for patients and normal controls were compared by the *t*-test for unpaired data. The left ventricular diameters and the heart rate changes induced by the cardiac autonomic function tests were correlated by standard regression and correlation analysis.

Results

Clinical, cineangiographic and echocardiographic characteristics

We studied 9 patients (7 men and 2 women) with isolated chronic aortic regurgitation. All patients were in functional class I (New York Heart Association, NYHA). The baseline heart rate was similar to that of the controls (Table 1).

The diagnosis of isolated chronic aortic regurgitation was confirmed by cardiac catheterization. All patients had severe aortic regurgitation (complete and dense left ventricular opacification in 1 beat with an eventual contrast density greater than that of the aorta). Left ventricular wall motion was normal in all patients. There were no segmental wall motion abnormalities upon a quantitative analysis of the left ventricular cineangiograms (21).

The echocardiographic evaluation performed on the same day of the assessment of cardiac autonomic function revealed no discrete areas of abnormal wall motion. However, there was marked left ventricular enlargement (Table 2).

The left ventricular ejection fraction was preserved and did not significantly differ from that of the normal controls (Table 1).

Cardiac autonomic function tests

Valsalva maneuver. When compared to the normal sedentary controls the Valsalva ratio was slightly lower in the asymptomatic patients with aortic insufficiency, although not significantly different (Table 2). There was no correlation between the Valsalva ratio and the left ventricular function parameters.

Standing up and facial immersion. The heart rate changes observed during standing up (30/15 ratio) and facial immersion (brady-

Table 1 - Clinical and echocardiographic characteristics.

Data are reported as means ± SD. *P≤0.01 compared to control (*t*-test).

Characteristics	Controls (N = 11)	Patients (N = 9)
Clinical		
Male	9 (82%)	7 (78%)
Female	2 (18%)	2 (22%)
Age	29 ± 13	28 ± 12
Baseline heart rate	82 ± 12	74 ± 10
Functional class I (NYHA)	–	9 (100%)
Echocardiographic		
Left ventricular function		
Diastolic diameter (mm)	44 ± 6.0	68 ± 6.3*
Systolic diameter (mm)	30 ± 7.8	45 ± 10*
Ejection fraction	0.63 ± 0.05	0.52 ± 0.10
Normal left ventricular wall motion	–	9 (100%)
Segmental wall motion abnormalities	–	–

Table 2 - Cardiac autonomic function tests.

Data are reported as means ± SD with the 95% confidence interval given below. There were no significant differences between patients and controls (ANOVA).

Autonomic test	Controls (N = 11)	Patients (N = 9)
Valsalva ratio	1.90 ± 0.49 1.63 ± 2.10	1.65 ± 0.33 1.39 ± 1.92
Standing up (30/15 ratio)	1.16 ± 0.16 1.07 ± 1.25	1.14 ± 0.11 1.04 ± 1.25
Facial immersion (beats/min)	-31 ± 15 -19 ± 43	-25 ± 22 -12 ± 38

cardic response) were similar in both groups of subjects. There were no significant correlations between the echocardiographic parameters of left ventricular function and the heart rate changes induced by these two cardiac autonomic function tests (Table 2).

Discussion

The Valsalva maneuver is a well-known and widely accepted test of cardiac parasympathetic function. This test, which has been used in multicenter trials to evaluate autonomic function, is reliable, consistent and operator-independent (24-28). The heart rate changes provoked during the maneuver and expressed as the Valsalva ratio are mostly dependent on cardiovagal integrity (29).

The Valsalva maneuver is abnormal in most asymptomatic patients with coronary heart disease and cardiomyopathies (14-16). The patients studied in these earlier clinical investigations had a distinct and common pathologic feature, i.e., left ventricular wall motion abnormalities (30-32). Most of these patients also had mild to moderate left ventricular dilatation. On the other hand, the ischemic and cardiomyopathic patients with a normal Valsalva ratio had either normal segmental wall motion or highly localized myocardial dyskinesis (i.e., apical aneurysms). Thus, asymptomatic patients with acquired heart disease of different etiologies and with an abnormal Valsalva ratio had segmental wall motion abnormalities and ventricular dilatation (33-37).

The mechanism(s) responsible for the abnormal heart rate changes during the Valsalva maneuver is (are) still unknown (1,4,5). We (36) and other investigators (10) have postulated that progressive ventricular dilatation may diminish the normal inhibitory action of cardiac vagal afferents on the brainstem vasomotor center which in turn may decrease the efferent parasympathetic traffic to the sinus node and partially explain the abnormal heart rate changes observed during the Valsalva maneuver (38,39).

In the present investigation, we studied patients with pure aortic insufficiency. We chose this clinical entity because, in asymptomatic chronic aortic insufficiency, myocardial fibrosis is discrete, does not affect systolic function and is of rather late appearance (18,20). Therefore, this clinical model may allow us to study the effects of left ventricular dilatation on parasympathetic heart rate control. Our results suggest that ventricular dilatation by itself does not significantly affect the Valsalva ratio in asymptomatic aortic insufficiency.

Since the two additional tests of cardiac parasympathetic function were also normal in our asymptomatic patients with ventricular dilatation and no segmental wall motion abnormalities, we are tempted to hypothesize that, in order to impair parasympathetic heart rate control, ventricular dilatation should be associated with segmental wall motion abnormalities and/or congestive heart failure (40).

Why would segmental wall motion abnormalities of different etiologies impair parasympathetic heart rate control? Recent experimental (41-43) and clinical investigations indicate that segmental myocardial abnormalities are very likely to be related to parasympathetic heart rate control (44,45). In these clinical studies ischemic patients with segmental wall motion abnormalities and impaired parasympathetic heart rate control were subjected to coronary angioplasty. Normalization of left ventricular wall motion by augmented coronary blood flow was followed by an improvement in parasympathetic heart rate control. The authors concluded that alterations in cardiac geometry, secondary to the presence of a noncontracting myocardial segment, may increase the discharge of sympathetic afferent fibers by mechanical distortion of their sensory endings. Similarly, an ischemic or necrotic myocardial segment may reduce the number of inhibitory cardiac vagal afferent fibers. Both

mechanisms may interfere with the tonic activity of the parasympathetic fibers directed to the sinus node (46,47).

In summary, our results indicate that, in asymptomatic patients with left ventricular dilatation and no segmental wall motion abnormalities, parasympathetic heart rate control as indicated by the Valsalva maneuver is not impaired.

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