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Increased Intraventricular Velocities

An Unrecognized Cause of Systolic Murmur in Adults

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Objectives. **The purpose of this study was to determine the frequency, clinical features and echocardiographic characteristics of increased intraventricular velocities (IIVs) in patients referred to the echocardiography laboratory for systolic murmur.**

Background. **A subset of patients referred to the echocardiography laboratory for evaluation of a systolic murmur have IIVs in the absence of other recognized causes of systolic murmur.**

Methods. **We prospectively studied echocardiograms from 108 consecutive patients referred for evaluation of a systolic murmur. Clinical data were obtained from patient examinations and medical records.**

Results. **The sole explanation for systolic murmur was IIVs in 16.7% of referred patients. Compared with those without IIVs, patients with IIVs had a higher ejection fraction (EF) (58.7 ± 7.8% vs. 51.1 ± 12.5%, $p < 0.001$), percent fractional shortening (42.3 ± 9.7% vs. 31.0 ± 11.4%, $p < 0.0001$), left ventricular (LV) mass index (181 ± 70 vs. 152 ± 48 g/m^2, $p = 0.046$) and prevalence of hypertension (73.3% vs. 51.7%, $p = 0.043$) and a lower prevalence of segmental wall motion abnormalities (2.2% vs. 39.3%, $p < 0.001$).**

Conclusions. **Increased intraventricular velocities are a common cause of systolic murmur in this group of patients and should be included in the differential diagnosis of systolic murmurs in adults. The association of IIVs with LV hypertrophy should be a clinical consideration when these murmurs are identified.**

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The finding of a previously undocumented systolic murmur discovered on physical examination is a common reason for referral to the echocardiography laboratory. The frequency, auscultatory characteristics and associated clinical and pathologic findings of the valvular and congenital causes of systolic murmurs have been described in detail elsewhere (1,2). The frequency and clinical significance of functional systolic murmurs in adults, however, are unknown.

This study was prompted by the observation that a subset of patients referred to the echocardiography laboratory for evaluation of a systolic murmur had no demonstrable valvular or congenital heart disease. Many of these patients, however, were found to have increased intraventricular velocities (IIVs). The objectives of this study were to determine 1) the prevalence of IIVs in a cohort of patients referred for systolic murmur; 2) the prevalence of IIVs in patients with systolic murmurs who lack echocardiographic evidence of valvular or congenital abnormalities; 3) the auscultatory characteristics of murmurs associated with IIVs; and 4) the clinical and echocardiographic characteristics of patients with IIVs.

Methods

Patients. We prospectively studied 108 consecutive patients referred to the echocardiography laboratory of the Tucson Veterans Affairs Medical Center for evaluation of a systolic murmur of unknown etiology. The referrals were collected over a 14-month period between June 1995 and August 1996. The Tucson Veterans Affairs Medical Center supports advanced cardiovascular care facilities within a tertiary care hospital. Referrals for echocardiographic examination are received from physicians and nurse practitioners within the institution and from other regional facilities.

For all patients included in the study, initial evaluation of a systolic murmur was the sole or principal reason for the echocardiographic request. Patients were excluded if a systolic murmur was not listed as the primary reason for echocardiographic study or if the study was requested for follow-up of a known valvular or congenital lesion. Both outpatient and inpatient referrals were included in the study. Patients were not screened before referral to the echocardiography laboratory.

Clinical data collection. A review of medical records and the decentralized hospital-based computer program data base

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Abbreviations and Acronyms

EF = ejection fraction
IIV = increased intraventricular velocity
LV = left ventricular
RWT = relative wall thickness

was performed for each patient. Data were collected regarding age, gender, race, medication history and past medical history. The pharmacy record for each patient was reviewed to determine whether prescriptions had been filled for angiotensin-converting enzyme inhibitors, beta-adrenergic blocking agents, calcium channel antagonists, digoxin or hydrochlorothiazide. Individual medical records were screened for the following medical problems: hypertension, diabetes mellitus, coronary artery disease and renal insufficiency. Hypertension was considered present if the diagnosis was documented in the medical record or if the two most recent systolic blood pressures documented in the chart were >140 mm Hg. Likewise, diabetes mellitus was present if the diagnosis was documented in the medical record, if the patient was prescribed insulin or if a fasting blood sugar level >140 mg/dl was documented. Coronary artery disease was determined to be present if coronary artery disease, angina pectoris or a previous myocardial infarction was documented in the medical record. Coronary artery disease was also considered present if there was angiographic evidence of one or more coronary artery lesions (>70% lumen diameter) or a history of previous coronary revascularization. Renal insufficiency was determined to be present if a serum creatinine level >1.5 mg/dl was documented within the 6 months preceding the echocardiographic evaluation.

Echocardiographic examination. Complete M-mode, two-dimensional and Doppler echocardiographic examinations were performed on all patients using commercially available cardiac ultrasound equipment (VingMed 750, Milpitas, California). Imaging was performed using a 2.35-MHz transducer according to the guidelines established by the American Society of Echocardiography (3,4) and recorded on ½-in. S-VHS videotape or digitized onto optical disks. All echocardiographic and Doppler measurements were performed on-line by experienced cardiac ultrasonographers. Patients were evaluated for all potential valvular and congenital causes of systolic murmurs, and the severity of each finding was graded semiquantitatively on a scale of 1 to 4+. Standard criteria were used to define the presence or absence of hypertrophic cardiomyopathy (5). The presence of hypertension was not used to exclude the diagnosis of hypertrophic cardiomyopathy.

Left ventricular (LV) intracavitary velocities were measured from the apical four-chamber, two-chamber and long-axis views. Doppler color flow imaging was used to identify areas of increased flow velocity by aliasing of the color signal or turbulent flow. From the apical four-chamber view, pulsed wave Doppler velocities were sampled from the mitral valve leaflet tips into the LV apex. From the apical long-axis view, velocities were sampled within the LV outflow tract, around the interventricular septum and into the LV apex. Right-sided intraventricular velocities were assessed in a similar fashion using pulsed wave Doppler sampling from the tricuspid leaflet tips into the right ventricular apex. Increased intraventricular velocities were defined as: 1) peak systolic velocities >0.7 m/s within the ventricular cavity but outside the ventricular outflow tract (defined by the absence of aortic valve closure clicks); or 2) peak systolic velocities within the LV outflow tract >1.2 m/s. Continuous wave Doppler imaging was used to verify the maximal intracavitary velocities. Only peak intraventricular velocities were reported.

From the apical four-chamber view, LV end-systolic and end-diastolic volumes and ejection fractions (EFs) were calculated using the modified Simpson's rule (6). Left ventricular mass index (g/m^2) was calculated as

$$1.04[(\mathrm{LVID/d} + \mathrm{IVS/d} + \mathrm{PW/d})^3 - (\mathrm{LVID/d})^3] - 13.6 \text{ (7)}.$$

For men and women, 134 g/m^2 and 110 g/m^2 were used as the upper limits of normal, respectively. Relative wall thickness (RWT) was calculated as

$$(\mathrm{IVS/d} + \mathrm{PW/d})/\mathrm{LVID/d} \times 100\%$$

Fractional shortening was calculated as

$$(\mathrm{LVID/d} - \mathrm{LVID/s})/\mathrm{LVID/d} \times 100\%,$$

where IVS/d and PW/d are the interventricular septal and posterior wall thickness in diastole, and LVID/d and LVID/s are the LV internal dimensions during diastole and systole.

Patients with IIVs were classified into four categories based on the probable source of the murmur. *Category 1*: IIV present without any other identifiable source of murmur. In these patients, the murmur was considered to be caused by the IIV (high probability). *Category 2*: IIV present in association with trace regurgitation of the mitral or tricuspid valves, without any other identifiable source of murmur. In these patients, the IIV was considered to be the most striking echocardiographic finding (intermediate probability). *Category 3*: IIV present in association with grade 1/2+ mitral or tricuspid regurgitation or aortic sclerosis without turbulent flow through the valve. In these patients, it was not possible to determine the origin of the murmur (indeterminate). *Category 4*: IIV present in association with aortic stenosis, aortic sclerosis with turbulent flow through the valve, grade 1+ and higher mitral or tricuspid regurgitation, or both, or a definable congenital lesion. In these patients, the murmur was determined to be of an origin clearly unrelated to the IIV (low probability).

Auscultation. Sixteen of 18 patients in category 1 underwent physical examinations by the authors (P.H.S., R.O.B., G.D.P.). The findings were recorded and compared among observers. Selected category 1 patients returned for simultaneous auscultation and repeat echocardiographic Doppler examinations.

Data analysis. Data are presented as the mean values ± SD. Unpaired, two-tailed t testing was used to compare continuous variables between patients with and without IIVs.

Table 1. Echocardiographic Findings in Patients Referred for Systolic Murmurs

Echocardiographic Finding	n	Percent
Mitral regurgitation	47	43.5
Increased intraventricular velocities	47	43.5
Tricuspid regurgitation	37	34.3
Aortic stenosis	30	27.8
Aortic sclerosis	25	23.2
Hypertrophic cardiomyopathy	1	0.9
Mitral annular calcification	1	0.9
Ventricular septal defect (supracristal)	1	0.9
Apical false tendon	1	0.9

Noncontinuous variables were compared by using the chi-square test. Correlation coefficients were determined by the least-squares method. Significance was defined at $p < 0.05$.

Results

Frequency of IIVs. A total of 108 patients were examined, including 23 inpatients and 85 outpatients. Increased intraventricular velocities were identified in 47 patients (43.5%). The groups with and without IIVs were comparable for the proportion of inpatients (9 [19.1%] of 47 vs. 14 [23%] of 61, respectively, $p = 0.809$). Of the 47 patients with IIVs, 18 were determined to be in category 1 (16.7%), 1 in category 2 (0.9%), 6 in category 3 (5.5%) and 22 in category 4 (20.4%). Only one patient was found to have right-sided IIVs. Mitral regurgitation and tricuspid regurgitation were the most frequent valvular lesions found by echocardiography (Table 1). As defined, IIVs were found as commonly as mitral regurgitation. In the group without IIVs, 3 (4.9%) of 61 patients had no apparent valvular or congenital abnormalities. In contrast, 18 (38.3%) of 47 patients with IIVs had no valvular or congenital abnormalities visualized ($p < 0.001$ vs. group without IIVs). Thus, of patients without echocardiographic valvular or congenital abnormalities, IIVs were present in 18 (85.6%) of 21 patients.

Clinical characteristics. The clinical characteristics associated with IIVs are shown in Table 2. Patients with and without IIVs were comparable for age, weight, race and gender ($p > 0.05$ for each). The overall prevalence of hypertension in our patient cohort was 58.3%. Patients with IIVs were more likely to have hypertension than those patients without IIVs (73.3% vs. 51.7%, $p = 0.043$). The mean creatinine level was similar between the groups with and without IIVs (1.35 vs. 1.38 mg/dl, respectively, $p = 0.900$), but the IIV group had a higher prevalence of chronic renal insufficiency (22.7% vs. 17.5%, $p = 0.035$). There was no difference in the prevalence of diabetes or coronary artery disease ($p > 0.05$). Also, there was no association between IIVs and the use of angiotensin-converting enzyme inhibitors, beta-blockers, calcium channel antagonists, digoxin or hydrochlorothiazide ($p > 0.05$).

Echocardiographic and Doppler findings. All of the patients with IIVs (100%) and the majority of the patients without IIVs (93.4%) were in sinus rhythm at the time of their echocardiographic examination. The peak intraventricular velocities ranged from 0.7 to 5.2 m/s (mean peak 1.45 ± 0.73). The spectral pattern of the intraventricular signal peaked in mid to late systole, with either a late-peaking "dagger-shaped" appearance or a more symmetric bullet-shaped pattern. Color aliasing or flow turbulence was observed in the mid-ventricular cavity (11.6%), near the proximal interventricular septum (51.2%) or within the LV outflow tract (37.2%) (Fig. 1).

Table 2. Clinical Characteristics of Patients With Increased Intraventricular Velocities

	IIVs Present	IIVs Absent	p Value
General			
Age (yrs)	65.2	66.3	0.660
Weight (lb)	189.1	180.2	0.249
Race (%)			
White	85.1	78.7	0.548
Black	4.3	3.3	0.805
Hispanic	6.4	11.5	0.675
Native American	4.3	6.6	0.982
Female (%)	17.0	31.1	0.141
Medications (%)			
ACE inhibitors	27.2	17.5	0.353
Beta-blockers	22.7	22.8	0.820
Calcium channel antagonists	31.8	17.5	0.150
Digoxin	2.3	10.5	0.225
Hydrochlorothiazide	20.5	12.2	0.391
Medical history (%)			
Hypertension	73.3	51.7	0.043
Diabetes	22.2	39.7	0.094
Coronary artery disease	22.2	36.2	0.187
Renal insufficiency	22.7	17.5	0.035

ACE = angiotensin-converting enzyme.

Although there was no difference in the prevalence of LV hypertrophy between the groups with IIVs (29 [82.9%] of 35 patients) and without IIVs (26 [66.6%] of 39, $p = 0.185$, patients with IIVs were found to have an increased mean LVEF (58.7 ± 7.8% vs. 51.1 ± 12.5%, $p < 0.001$), percent fractional shortening (42.3 ± 9.7% vs. 31.0 ± 11.4%, $p < 0.001$), LV mass index (181 ± 70 vs. g/m^2 vs. 152 ± 48 g/m^2, and RWT (57.7 ± 16.4 vs. 45.7 ± 13.1, $p < 0.001$) (Fig. 2 and 3). Patients with IIVs had a decreased mean systolic LV cavity dimension and an increased mean septal and posterior wall thickness (Table 3, $p < 0.01$ for each), without differences in percent septal or posterior wall thickening ($p > 0.05$). A sigmoid septum was found more commonly, and the angle between the LV outflow tract and the interventricular septum was more profound in patients with IIVs. No patient in the group with IIVs had a ventricular septal-to-posterior wall thickness ratio >1.3. Also, there was a conspicuous absence of segmental wall motion abnormalities in patients with IIVs as compared with those without ($p < 0.001$). The groups with and without IIVs were comparable for the proportion of patients with aortic stenosis (11 [23.4%] of 47 vs. 19 [31.1%] of 61, respectively, $p = 0.500$). For patients with aortic stenosis, the mean transvalvular aortic gradient in the groups with and

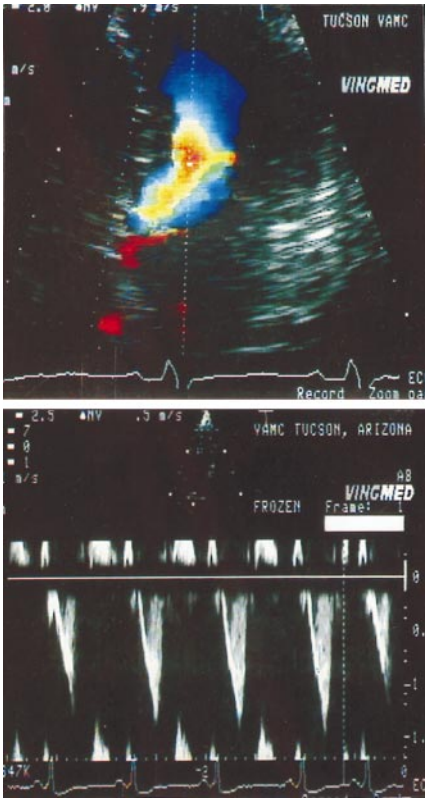

Figure 1. Upper panel, Doppler color flow imaging demonstrating color aliasing and turbulent flow around the proximal septum during systole. **Lower panel,** Pulsed wave Doppler interrogation at the mid-ventricle demonstrating an IIV. Note mid to late systolic peak and relatively lower velocities as compared with those obtained in hypertrophic obstructive cardiomyopathy.

without IIVs was comparable (22.7 vs. 20.0 mm Hg, respectively, p = 0.669).

To further examine the LV geometry and function associated with IIVs, peak intraventricular velocities were correlated with the continuous variables in Table 3 and Figures 2 and 3. Peak velocities were positively correlated with percent of septal thickening (r = 0.391, p = 0.022), percent fractional shortening (r = 0.625, p < 0.0001), LV mass (r = 0.439, p = 0.007) and LV mass index (r = 0.503, p = 0.002) and inversely correlated with LV systolic dimension (r = −0.439, p = 0.008).

Auscultatory characteristics of murmur. The murmurs heard in the patients with IIVs were typically grade II to III/VI. They were localized to the left lower sternal border, fourth intercostal space, but in selected patients they were also audible at the apex, at the base, or less frequently, at the right lower sternal border. There was no radiation to the neck or axilla. The crescendo-decrescendo, ejection-type murmur began after the first heard sound, peaked during mid to late systole and ended just before the second heart sound. The first heart sound was frequently prominent. With the Valsalva maneuver, there was a delayed augmentation of murmur intensity (five to seven beats) and a delayed diminution of the murmur after termination of the maneuver. The increase in the intensity of the murmur with the Valsalva maneuver was

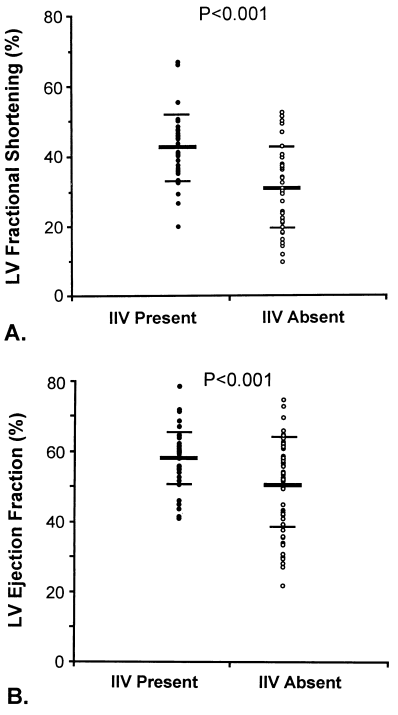

Figure 2. **(A)** Percent LV fractional shortening and **(B)** LVEF in patients with and without IIVs. Data points are presented with the superimposed mean value **(thick line)** and standard deviation **(thin lines).**

associated with an increased peak intraventricular velocity (Fig. 4). The murmur intensity and peak velocity were decreased with handgrip, markedly accentuated with standing

Figure 3. **(A)** Left ventricular mass index and **(B)** RWT in patients with and without IIVs. Data points are presented with the superimposed mean value **(thick line)** and standard deviation **(thin lines).**

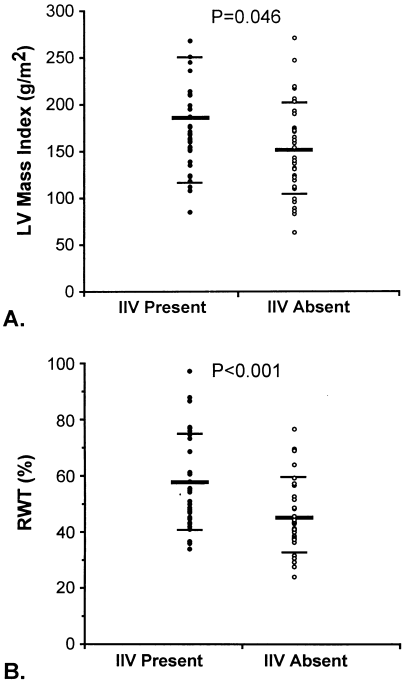

Table 3. Doppler Echocardiographic Measurements of Patients With and Without Increased Intraventricular Velocities

Echocardiographic Variable	IIVs Present	IIVs Absent	p Value
Sinus rhythm (%)	100	93.4	0.207
Segmental wall motion abnormalities (%)	2.2	39.3	<0.001
IVS/d (cm)	1.41 ± 0.29	1.16 ± 0.20	<0.0001
IVS/s (cm)	1.84 ± 0.39	1.56 ± 0.33	0.002
Septal thickening (%)	30.9 ± 16.8	36.4 ± 22.2	0.259
PW/d (cm)	1.39 ± 0.30	1.18 ± 0.22	0.0009
PW/s (cm)	2.16 ± 0.50	1.69 ± 0.33	<0.0001
Posterior wall thickening (%)	53.7 ± 24.5	43.3 ± 24.1	0.084
LVID/d (cm)	5.02 ± 0.753	5.32 ± 1.06	0.165
LVID/s (cm)	2.90 ± 0.717	3.81 ± 1.20	<0.001
Sigmoid septum present (%)	30.2	11.7	0.036
LVOT–septal angle	87 ± 12	100 ± 16	<0.0001

M-mode measurements of the left ventricle were obtained from a parasternal long-axis view. Data are presented as the mean value ± SD. IVS/d = interventricular septal thickness during diastole; IVS/s = interventricular septal thickness during systole; PW/d = posterior wall thickness during diastole; PW/s = posterior wall thickness during systole; LVID/d = left ventricular internal dimension during diastole; LVID/s = left ventricular dimension during systole; LVOT–septal angle = angle between the long axis of the left ventricular outflow tract and the long axis of the septum in an apical five-chamber view.

and markedly diminished or abolished with squatting or leg raises.

Discussion

This study implicates IIVs as a pathophysiologic mechanism of murmurs in adults. In our patient group a large number of patients referred for systolic murmurs had IIVs (43.5%), and IIVs were considered to be the sole Doppler echocardiographic explanation for the murmur in 16.7% of patients. Patients with IIVs were more likely to have hypertension or renal insufficiency and, echocardiographically, had a higher LV mass index, RWT and percent fractonal shortening. Also, patients with IIVs had increased wall thicknesses, a higher LVEF, altered septal–LV outflow tract geometry and a lower prevalence of segmental wall motion abnormalities. These data indicate that patients with IIVs have more vigorous systolic function and more severe concentric remodeling than patients without IIVs.

Only one patient of the 108 studied met the definition of hypertrophic cardiomyopathy (5), and this patient was excluded from the IIV group analysis. None of the patients with IIVs had asymmetric septal hypertrophy, defined as an interventricular septal to posterior wall thickness >1.3. It is possible that some of the patients included in the cohort had genetic abnormalities associated with hypertrophic cardiomyopathy (8) or that patients with IIVs may represent a *form fruste* of the disease (9). It is more likely, however, that concentric ventricular remodeling in the setting of long-standing hypertension establishes the cavity geometry necessary for IIVs. The high prevalence of hypertension in our Veterans Affairs patients may explain why IIVs and associated murmurs are particularly common.

The diagnosis of coronary artery disease was equally common between the patients with and without IIVs, but patients with IIVs were less likely to have segmental wall motion abnormalities. A plausible explanation is that IIVs disappear after myocardial infarction, a situation similar to the postinfarction disappearance of intraventricular gradients and systolic murmurs in patients with hypertrophic cardiomyopathy (10). Our data indicate that the prerequisites for IIVs include vigorous LV systolic function, concentric LV hypertrophy and small systolic cavity dimensions, without segmental wall motion abnormalities.

Relation to previous studies. In the absence of hypertrophic cardiomyopathy, IIVs are not a recognized cause of systolic murmur. Three standard textbooks of cardiology do not describe IIVs as a cause for systolic murmur, except in association with hypertrophic cardiomyopathy (11–13). One previous report of dynamic midventricular obstruction as a cause of systolic murmur in adults describes nine patients with

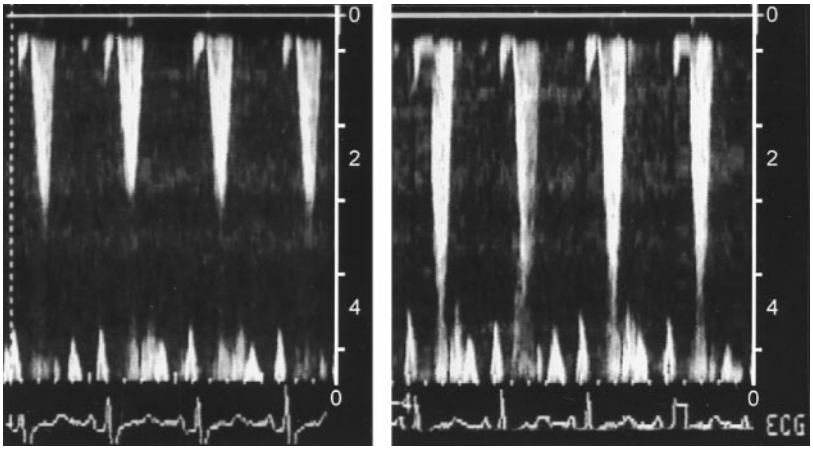

Figure 4. Pulsed wave Doppler representation of IIV before **(A)** and after **(B)** the Valsalva maneuver.

apical late systolic ejection murmurs associated with dynamic mid-LV obstruction (14). Two of the nine patients had hypertrophic cardiomyopathy.

Other investigators have referred to IIVs as intracavitary gradients (15), abnormal LV intracavitary flow acceleration (16) and midventricular systolic outflow obstruction (17). After aortic valve replacement, the presence of abnormal intracavitary flow acceleration defines a group of patients with increased RWT, higher EFs and increased postoperative morbidity and mortality (16).

In 1985, Topol et al. (18) described hypertensive hypertrophic cardiomyopathy of the elderly, a syndrome observed predominantly in elderly, black and female patients. Echocardiography demonstrated severe concentric LV hypertrophy with reduced cavity dimensions and supernormal systolic function. The study did not include Doppler measurements or the prevalence of systolic murmurs. The investigators concluded that this condition warrants recognition and tailored management, particularly avoidance of vasodilators. Subsequently, Pearson et al. (19) studied a series of 17 patients with similar demographic data and late-peaking LV outflow tract velocities ranging from 1.0 to 5 m/s. More recently, Harrison et al. (17) described a series of 10 patients with Doppler echocardiographic evidence of midventricular systolic obstruction and late-peaking intraventricular velocities ranging from 1.9 to 4.5 m/s. Eight of 10 patients were found to have systolic murmurs. Study subjects were predominantly elderly women with hypertension and concentric LV hypertrophy. In general, the prognosis of elderly patients with hypertensive cardiomyopathy is favorable (20).

The aforementioned studies differ from this study in several important aspects. The cohorts in the earlier studies were echocardiographically selected for the presence of mid-LV obstruction or concentric LV hypertrophy with supernormal systolic function. In contrast, our patients were referred for echocardiographic evaluation of a systolic murmur by practitioners with no knowledge of the study. Also, the patients in the current study are younger, predominantly male and predominantly white. This suggests that, independent of demographic data, the murmur of IIVs occurs when vigorous LV systolic function, LV hypertrophy and decreased systolic cavity dimensions coexist. The common variable between earlier studies and our Veterans Affairs patient cohort appears to be the presence of hypertension. This syndrome, therefore, may be more appropriately labeled hypertensive hypertrophic cardiomyopathy rather than hypertensive hypertrophic cardiomyopathy *of the elderly*.

Clinical significance of IIVs and systolic murmurs. In children, benign systolic murmurs are a common auscultatory finding (21) associated with a variety of echocardiographic findings, including increased aortic flow velocities (22). Before this study, the frequency of functional systolic murmurs in adults had not been estimated. Historically, these murmurs have been considered to be "benign" flow murmurs. In our study, patients with IIV-associated murmurs had a higher LV mass index, and the peak intraventricular velocities were positively correlated with LV mass index. Because LV hypertrophy is a strong independent risk factor for cardiovascular morbidity and mortality (23,24), the increased severity of concentric LV hypertrophy found in patients with IIVs suggests that these murmurs may not be "benign." Although most clinically significant valvular and congenital lesions can be reliably diagnosed from a thorough physical examination (25), concentric LV hypertrophy is not reliably diagnosed without echocardiographic examination. Echocardiography, therefore, may be indicated in evaluating patients with systolic murmurs that are consistent with IIVs.

Study limitations. Our study did not attempt to establish a direct cause and effect relation between IIVs and systolic murmurs, as this would have required a sophisticated, invasive approach. Nevertheless, the murmurs that we characterized by stethoscope had features that were strongly suggestive of an intraventricular source. First, the murmur intensity diminished with maneuvers that increased LV volume and increased with maneuvers that decreased LV volume. These changes were accompanied by parallel changes in IIVs, suggesting that the murmurs are ventricular and not valvular in origin. Also, the delayed changes in murmur intensity with the Valsalva maneuver are typical of murmurs originating from the LV (1). Second, this study defined a subset of patients referred for murmurs in whom IIVs were found, but in whom no valvular or congenital abnormalities were visualized. Finally, LV geometry and function differed between patients with and without IIVs. Taken together, these data implicate IIVs as a cause of systolic murmurs in adults.

Our study may have overestimated the prevalence of IIVs and associated murmurs in the general population because of the higher prevalence of hypertension and LV hypertrophy in the veteran population. For logistic reasons, cardiac auscultation was not performed on all patients and was often not performed on the same day as the echocardiogram. Also, our study did not attempt to identify the frequency of systolic murmurs in patients with IIVs, although earlier data suggest that the prevalence of systolic murmur in patients selected for IIVs is high (82%) (19).

Conclusions. This study demonstrates that a large proportion of veteran patients referred for echocardiographic evaluation of a systolic murmur have IIVs. A higher than expected number of patients had IIVs, without valvular or congenital lesions to explain the cause of the murmur. The findings of this study support the hypothesis that IIVs are a cause of systolic murmur. Patients with IIVs more commonly have hypertension and rarely have segmental wall motion abnormalities. Also, patients with IIVs have an increased LV mass index, increased wall thickness, more vigorous LV systolic function and a smaller systolic cavity dimension. Because of the association between IIVs and increased LV mass index, the presence of the characteristic systolic murmur on physical examination may constitute a marker for concentric LV hypertrophy. In addition to hypertrophic cardiomyopathy and valvular and congenital heart disease, the differential diagnosis of a systolic murmur in adults should be expanded to include IIVs.

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