Potential use of isovolumic contraction velocity in assessment of left ventricular contractility in man: A simultaneous pulsed Doppler tissue imaging and cardiac catheterization study

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Cardiac catheterization; Doppler tissue imaging; Isovolumic contraction velocity; Left ventricular contractility

Abstract  Aims: Echocardiographic techniques have so far provided suboptimal estimates of myocardial contractility in humans. Longitudinal myocardial motion during the isovolumic contraction (IVC) phase, measured by colour tissue Doppler imaging (TDI), has recently been shown in experimental animal models to reflect the state of myocardial contractility. The aim of the present study was to investigate the relationship between left ventricular (LV) isovolumic contraction velocities (IVCv) using pulsed Doppler tissue imaging (DTI) and global LV contractility as measured during cardiac catheterization.

Methods and results: Cardiac catheterization and pulsed DTI were simultaneously performed in 16 consecutive patients (13 males, mean age 55 ± 10 years) with a variety of cardiac diseases. Relationships between the peak positive IVCv as measured at basal levels of the lateral, septal, anterior and posterior walls and the first derivative of LV pressure (+dP/dtmax), were investigated. Peak IVCv measurements were obtainable in 81–100% of the four LV wall segments. Statistically significant linear relationships were found between IVCv and +dP/dtmax at the lateral (r = 0.58, P < 0.05), septal (r = 0.66, P < 0.01), anterior (r = 0.73, P < 0.01) and posterior (r = 0.81, P < 0.001) segments of the LV.

Conclusion: IVCv of the basal four LV walls correlates strongly with peak +dP/dt. IVCv is a readily obtainable non-invasive parameter, which correlates with the classical

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Introduction

In common clinical practice, 2D and M-mode echocardiography provide qualitative and semi-quantitative measurements of ventricular systolic function. Such data are generated from the ejection phase which is load dependent and therefore not ideal for the determination of ventricular contractility. However, the load independent state of ventricular contractility can only be assessed by high temporal resolution techniques timed to record during the isovolumic contraction (IVC) period which normally occurs over a brief time interval (approximately 80 ms), although this may vary in disease states. Furthermore, wall motion during IVC shows a biphasic pattern in normals but this may be altered when ventricular contractility is impaired. Currently, tissue Doppler imaging (TDI) provides information regarding regional myocardial function with high spatial and temporal resolution. Vogel et al. showed that myocardial acceleration during IVC was a sensitive marker of the global state of contractility and was not particularly dependent on pre- or afterload. A number of other studies have supported this observation. More recently however, Lyseggen et al. showed that there was no consistent relationship between peak IVC acceleration and regional myocardial contractility as measured by sonomicrometry.

All of the above studies were carried out in animal models and have not been validated in healthy human volunteers or in patients with diseased ventricles. Therefore, the aim of the present study was to investigate the relationship between myocardial peak isovolumic contraction velocity and invasive indices of global left ventricular contractility in a consecutive series of patients with a variety of cardiac diseases by using simultaneous DTI and cardiac catheterization measurements.

Material and methods

Study population

Sixteen consecutive patients (13 of whom were males) referred for routine cardiac catheterization were studied. Their mean age was 55 ± 10 (SD; range 35–70) years. Two patients had hypertrophic cardiomyopathy, 5 dilated cardiomyopathy, 4 heart transplantsations, 1 atrial septum defect, 1 primary pulmonary hypertension, 1 combined mitral and tricuspid valve disease, and 2 ischemic heart diseases (Table 1). Ten patients were in sinus rhythm, 4 had atrial fibrillation or atrial flutter, 1 had a pacemaker implanted and 1 had left bundle branch block. All patients gave written consent to participate in the study, which was approved by the local ethics committee.

Echocardiographic examination

Doppler echocardiographic examinations were performed simultaneously with cardiac catheterization with patients lying in the supine position. All patients were stable haemodynamically. All tracings were recorded during expiration. A commercially available ultrasound system (HP, Sonos 5500, Philips, Andover, MA, USA) equipped with a multi frequency phased array transducer and pulsed Doppler tissue imaging technique was used. Parasternal and apical views were obtained according to the recommendations of the American Society of Echocardiography. All recordings were made with a simultaneous superimposed ECG and phonocardiogram (PCG). Recordings were made at a sweep speed of 50 and 100 mm/s and stored on magnetic optical discs. Values are presented as means from three consecutive cardiac cycles. Peak myocardial velocities were recorded using the pulsed Doppler tissue imaging technique. The sample volume was 6 mm. The acoustic power and filter frequencies were adjusted and optimized for detecting myocardial velocities.

Cardiac catheterization

One experienced investigator (G.W.) performed all the cardiac catheterization studies. Briefly, retrograde left ventricular catheterization was performed via the brachial or radial artery (Becton Dickinson Criticath SP5 107 HTD catheter). Pressures were registered using a Cathcor® system 3.3 (Siemens Elema AB, Electromedical Systems Divisions, Solna, Sweden).
Measurements

The following measurements were made from the 2D and M-mode echocardiography recordings: left ventricular (LV) internal diameter at end-diastole (onset of the Q wave), and fractional shortening as recommended by the American Society of Echocardiography. Using 2D echocardiography, ejection fraction was derived from Simpson’s modified single plane method using a 4-chamber view. Left ventricular long axis motion was measured from the Q-wave on the ECG to the highest amplitude of the second heart sound from the PCG at lateral, septal, anterior and posterior basal segments of the left ventricle. Velocity time integrals were measured at the LV outflow tract. All catheter measurements of $+dP/dt_{max}$ were compared and correlated with indices of the ejection phase of left ventricular systolic function. Doppler tissue imaging was used to measure left ventricular longitudinal myocardial segmental function. The systolic and isovolumic contraction peak positive velocities were measured at left lateral, septal, anterior and posterior basal levels of the left ventricle (Fig. 1).

Statistical analysis

A commercially available statistical program (SPSS 10.1 and 11.1) was used. All data are presented as mean ± SD. Spearman’s correlation and linear regression analyses were utilized to display relationships. A $P$ value of less then 0.05 was considered statistically significant.

Results

General characteristics of the study population

The patient’s characteristics are presented in Table 1.

Left ventricular regional myocardial functional and global left ventricular contractility

A significant positive relationship was found between $+dP/dt_{max}$ and peak positive isovolumic contraction velocity at all four left ventricular segments: basal lateral ($r = 0.58$, $P < 0.05$), septal ($r = 0.66$, $P < 0.01$), anterior ($r = 0.73$, $P < 0.01$) and basal posterior segments ($r = 0.81$, $P < 0.001$) (Fig. 2).

We found a significant positive correlation between all single measurements of IVCv and $+dP/dt_{max}$ at all segments ($r = 0.66$, $P < 0.001$) (Fig. 3).

We also found a robust correlation between $+dP/dt_{max}$ and systolic peak positive velocities at the posterior segments ($r = 0.72$, $P < 0.01$).

### Table 1 Patient characteristics

<table>
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<tr>
<th>Patient</th>
<th>Cardiac disease</th>
<th>Age (years)</th>
<th>HR (b/m)</th>
<th>SBP (mmHg)</th>
<th>DBP (mmHg)</th>
<th>Gender</th>
<th>NYHA</th>
<th>LVEF (%)</th>
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DCM, dilated cardiomyopathy; XP, heart transplantation; ASD, atrial septum defect; MR, mitral regurgitation; TR, tricuspid regurgitation; PPHT, primary pulmonary hypertension; HCM, hypertrophic cardiomyopathy; IHD, ischemic heart disease; b/m, beats per minute; HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure; NYHA, New York Heart Association class; LVEF, left ventricular ejection fraction.
However, correlation between IVCv and SV was seen only at the posterior segment ($r = 0.63$, $P < 0.05$).

Left ventricular systolic function and global left ventricular contractility

A statistically significant though weak correlation was noted between $+\frac{dP}{dt_{\text{max}}}$ and LV fractional shortening ($r = 0.59$, $P < 0.05$). There was no significant relationship between $+\frac{dP}{dt_{\text{max}}}$ and long-axis motion at any segment, left ventricular outflow velocity time integral or LV ejection fraction.

Peak positive isovolumic contraction velocity and its relation to indices of pre- and afterload

IVCv did not correlate significantly with either left ventricular end diastolic pressure or systemic vascular resistance at any of the left ventricular segments.

Reproducibility and feasibility

Reproducibility as coefficient of variation for inter-observer variability was found to be 6% for lateral, 6% for septal, 9% for anterior and 7% for posterior walls. Intra-observer variability was 3% for lateral, 3% for septal, 4% for anterior and 2% for posterior walls. Feasibility of measurements was found to be 81% for lateral, 100% for septal, 88% for anterior and 94% for posterior segments.

Discussion

The main finding of the present study

We have demonstrated for the first time in patients that peak positive myocardial velocity of the left ventricular free walls, measured by pulsed Doppler tissue imaging during the IVC phase, correlates well with left ventricular global contractility. These observations have the potential to be of clinical value since disturbances of myocardial motion occur predominantly during the isovolumic phases, (i.e. during contraction and/or relaxation), and such non-invasive recordings might therefore be a sensitive marker of myocardial dysfunction.5

Left ventricular contractility

Myocardial systolic function depends upon the interaction of myocardial contractility, preload and afterload. Myocardial contractility refers to the property of the heart muscle that accounts for alternations in performance induced by biochemical and hormonal changes and has classically been regarded as independent of preload and afterload. Isovolumic indices such as the maximum rate of raise LV systolic pressure, $dP/dt$, has been used as a measure of myocardial contractility. In clinical practice this is difficult since it require simultaneous recording of ventricular pressure and volume.

In our study we found no relationship between IVCv to either end-diastolic pressure or systemic vascular resistance. In an animal study, Vogel et al. showed that IVC acceleration is a reliable measurement of right ventricular contractility and is relatively load independent.7 These authors concluded that IVC acceleration was a more sensitive marker of contractility than IVC peak positive velocity. We found it difficult to measure accurately the acceleration of isovolumic contraction velocity as this was commonly multidirectional which supports the anatomical explanations of Buckberg.18 Therefore, we propose that the peak positive isovolumic contraction velocity may be the most appropriate measure of contractility in humans. Recently, Hashimoto et al. used a computerized method to measure acceleration9 and this approach might improve the accuracy of such measurements.
Isovolumic contraction velocity

In the present study and in clinical setting, we demonstrate the feasibility and reproducibility of IVC peak positive velocities for measurement of the left ventricular contractile state, which was found to be significant in particular for the septal and posterior segments. Differences in regional contractility have previously been shown. The reason why the relationship between IVCv and $+\frac{dp}{dt}$ varied according to the ventricular segment might be explained by segmental differences of the biphasic positive and negative IVCv, which is hypothesized to be due to asynchronous deformation patterns of subendocardial and subepicardial layers. We measured only peak positive velocities and the results might have differed had we measured both positive and negative velocities. Recent studies have shown that myocardial acceleration during IVC did not correlate with the state of contractility. However, that study compared acceleration during IVC with regional contractility where as we measured global contractility.

It is important to mention that we did not perform visual assessment of regional wall motion abnormalities in the studied population (normal; hypokinetic, akinetic, dyskinetic). We believe that wall motion score is strongly limited by a low frame rate (20–30 frames/s), which limits the sensitivity. In contrast pulsed Doppler tissue imaging is preferable in wall motion analysis because it is related to a very high frame rate (>200 frames/s).
Study limitations

The study has a number of limitations:

1. We acknowledged that a relatively small number of patients were investigated and confirmatory data is needed. Nevertheless, we were able to disclose statistically significant relationships between peak positive IVCv and global LV contractility. Also our study population was heterogeneous; it would have been better if our patient group had been homogeneous.

2. We did not measure myocardial acceleration during IVC and we measured only the peak positive velocity during IVC. It may have been more accurate to measure both positive and negative velocities and this again requires formal assessment.

3. Four of our patients had atrial fibrillation or flutter so the mean of three beats is suboptimal and at least six consecutive beats should have been averaged.

4. Finally, in this study we used a thermo-dilution catheter, whereas a tip manometer catheter with higher frequency responses would be expected to have strengthened the results. The latter premise, however, needs to be assessed formally.

Clinical implications of our study

Doppler tissue imaging could provide a useful tool for early detection of cardiac dysfunction. Doppler tissue imaging indices may complete the conventional analysis of the left ventricular function in different cardiac diseases. We propose IVCv as an additional non-invasive tool to be taken into consideration for complete assessment of left ventricular contractility. However, the usefulness of IVCv in the early detection of myocardial dysfunction needs to be clarified.

Conclusion

Left ventricular contractility can be determined by measuring peak positive isovolumic contraction velocity, which is relatively load independent. The positive IVCv is a readily obtainable, non-invasive parameter. It seems that there are regional differences in wall motion during IVC when Doppler tissue imaging is used to determine the state of LV contractility.

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References