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The use of isovolumic contraction velocity to determine right ventricular state of contractility and filling pressures
A pulsed Doppler tissue imaging study

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Abstract **Aims** Echocardiographic techniques have not so far been able to provide a good estimation of myocardial contractility in humans. Myocardial motion during the isovolumic contraction phase, measured by Doppler tissue imaging, has only recently in experimental models been shown to describe myocardial contractility. The aim of the present clinical study was to investigate the relationship between right ventricular isovolumic contraction velocity and right ventricular state of contractility.

Methods and results Doppler tissue imaging and cardiac catheterization were performed simultaneously in 26 consecutive patients with different cardiac diseases (18 males, mean age 52 ± 12, range 23—75 years). Peak isovolumic contraction velocity was measured using Doppler tissue imaging at 2 levels of the right ventricular free wall. During cardiac catheterization, the first derivative of right ventricular pressure related to the pulmonary artery peak pressure ($dP/dt/P_{max}$) was measured. Furthermore, right ventricular end diastolic pressure, right atrial mean pressure, pulmonary artery systolic pressure, and pulmonary artery resistance, were also measured. A significant relationship was found between isovolumic contraction velocity and $dP/dt/P_{max}$ ($r = 0.59$, $p < 0.01$). This relationship was strengthened after excluding patients with elevated right atrial pressures

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($r = 0.77$, $p < 0.001$). Furthermore, a correlation was also found between basal and mid cavity isovolumic contraction velocity and right ventricular end diastolic pressure ($r = -0.47$, $r = -0.49$, $p < 0.05$) as well as right atrial mean pressure ($r = -0.63$, $r = -0.55$, $p < 0.01$).

Conclusion Isovolumic contraction velocity is a reproducible and easily obtainable non-invasive parameter which correlates with invasive measurements of right ventricular state of contractility and right ventricular filling pressures.

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Introduction

Reduced right ventricular ejection fraction is a powerful predictor for adverse outcome.[1] Thus, assessment of right ventricular function is of clinical importance.[2] However, most non-invasive diagnostic techniques have shown limitations due to the complex anatomy and geometry of the right ventricle.[3] Doppler tissue imaging has previously been suggested to be a reliable tool for the evaluation of regional myocardial function throughout the cardiac cycle.[4] Myocardial motion during the isovolumic periods, which is easily detected by Doppler tissue imaging has recently attracted much interest.[5] The duration and the peak velocity for the isovolumic contraction phase are proposed to reflect myocardial function and correlate to increased right ventricular end diastolic pressure.[6,7] Recently, Vogel et al.[8] demonstrated by using Doppler tissue imaging, that acceleration of isovolumic contraction velocity is a reliable measurement of right ventricular contractility. Furthermore, this parameter is not affected by loading conditions and is therefore useful as an index of right ventricular function. All these studies, however, were carried out in animal models and have not been validated in a clinical setting in normal and diseased ventricles.[9] Therefore, the aim of the present study was to investigate the relationship between myocardial peak isovolumic contraction velocity and indices of right ventricular contractility in consecutive patients with different cardiac diseases using Doppler tissue imaging as validated to a simultaneous cardiac catheterization measurement.

Methods

Study population

Twenty-six consecutive patients (18 were males) referred for routine cardiac characterization due to different cardiac diseases were studied. Their mean age was 52 ± 12 (range 23–75) years. Two patients had hypertrophic cardiomyopathy, 10 with dilated cardiomyopathy, 4 with heart transplantation, 1 with systemic hypertension, 1 with mitral valve prosthesis dysfunction, 4 with primary hypertension, 1 with heart failure due to long-standing aortic stenosis, 2 with mitral valve disease, and 1 with ischemic heart disease (Table 1). All patients were in sinus rhythm and patients with right bundle branch block were excluded. Nine patients were on diuretics, 12 on ACE-inhibitors, 12 on beta-blockers, 1 on digitalis and 6 on calcium antagonist. All patients gave their 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 in stable haemodynamic condition and tracing were recorded during expiration. A commercially available ultrasound system (HP, Sonos 5500, Philips, Andover, Mass., U.S.) equipped with a multifrequency 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.[10] All recordings were made with a simultaneous superimposed ECG and phonocardiogram (PCG, HP 2105A, Philips, Andover, Mass., U.S.).[11,4] Recordings were made at a sweep speed of 50 and 100 mm/s and stored on magnetic optical discs. Values are presented as means of 3 consecutive beats.

Right ventricular myocardial Doppler tissue imaging

Myocardial systolic and diastolic velocities and the time intervals were recorded using pulsed Doppler tissue imaging technique. The sample volume was 6 mm. The acoustic power and filter frequencies were adjusted for detecting myocardial velocities.

Table 1 Patient characteristics

	DCM	Transplanted	sPHT	pPHT	HCM
n	10	4	6	4	2
Age (years)	49 ± 9	56 ± 8	60 ± 8	30 ± 9	64 ± 6
HR (bpm)	62 ± 12	76 ± 14	89 ± 16	74 ± 14	73 ± 1
SBP (mmHg)	118 ± 21	152 ± 31	131 ± 26	120 ± 30	128 ± 16
DBP (mmHg)	76 ± 11	92 ± 2	77 ± 13	68 ± 13	85 ± 7
Gender (m/f)	8/2	4/0	3/3	1/3	2/0
BSA (m^2)	2.07 ± 0.26	2.15 ± 0.17	1.88 ± 0.31	1.75 ± 0.21	2.15 ± 0.21
NYHA (I/II/III/IV)	0/4/5/1	2/2/0/0	0/2/2/2	0/2/2/0	0/0/0/2
PASP (mmHg)	47 ± 32	32 ± 3	71 ± 39	90 ± 26	42 ± 0
RAMP (mmHg)	7 ± 5	6 ± 2	12 ± 7	14 ± 10	8 ± 4
LVEF (%)	35 ± 11	54 ± 15	31 ± 23	72 ± 7	43 ± 19
LVDD (mm)	71 ± 11	52 ± 11	50 ± 7	43 ± 12	54 ± 18
RVS long axis (mm)	18 ± 5	17 ± 2	16 ± 9	13 ± 7	13 ± 1

DCM = dilated cardiomyopathy; sPHT = secondary pulmonary hypertension; pPHT = primary hypertension; HCM = hypertrophy cardiomyopathy; HR = heart rate; SBP = systolic blood pressure; SDP = diastolic blood pressure; BSA = body surface area; PASP = pulmonary artery systolic pressure; RAMP = right atrial mean pressure; LVEF = left ventricular ejection fraction; LVDD = left ventricular diastolic diameter; RVS = right ventricular systolic.

Cardiac catheterization

One experienced investigator performed all the cardiac catheterization studies (GW). Briefly, a balloon catheter was inserted through the right internal jugular vein or right brachial vein (Becton Dickinson Criticath SP5 107 HTD catheter). Pressures were registered with 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 internal diameter at end diastole (onset of the Q wave) as recommended by the American Society of Echocardiography.[12] From the 2D echocardiography registrations, ejection fraction was derived from Simpson's modified single plane method using 4-chamber view. Right ventricular outflow tract fractional shortening was calculated as the percentage fall in right ventricular outflow tract diameter in systole, as previously described.[13] Right ventricular systolic long axis amplitude was recorded from the apical 4-chamber view with the M-mode cursor positioned at the free wall of the tricuspid valve annulus.[14] Furthermore, right ventricular end diastolic and systolic areas were manually measured and right ventricular area change was calculated. All these measurements were done to compare dP/dt/P_{max} to systolic indices of right ventricular function.

Right ventricular myocardial Doppler tissue imaging

Doppler tissue imaging was used to measure right ventricular longitudinal myocardial segmental function.[15,16] From the basal and mid cavity level the peak systolic (Sv) and isovolumic contraction velocity (IVCv) was measured (Fig. 1).

Statistical analysis

A commercially available statistical program (SPSS 10.1 and 11.1) was used. All data are presented as the mean ± SD. Pearson's correlation and linear
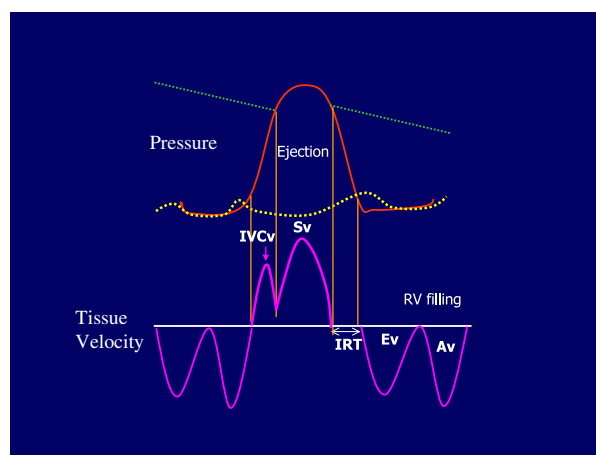
Figure 1 Schematic traces from Doppler tissue imaging with superimposed ventricular and atrial pressures. IVCv = isovolumic contraction velocity; Sv = systolic velocity during ejection period; Ev = early diastolic velocity; Av = atrial velocity; IRT = isovolumic relaxation time.

regression analyses were plotted to show certain relationships. A *p*-value less then 0.05 was considered significant.

Results

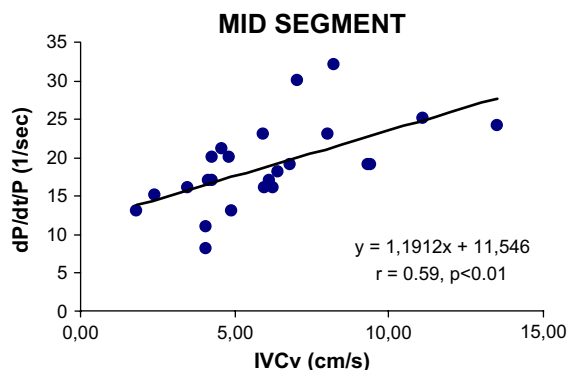### General characteristics of the study population

The patient's characteristics are presented in Table 1.

Right ventricular isovolumic contraction velocity and right ventricular contractility
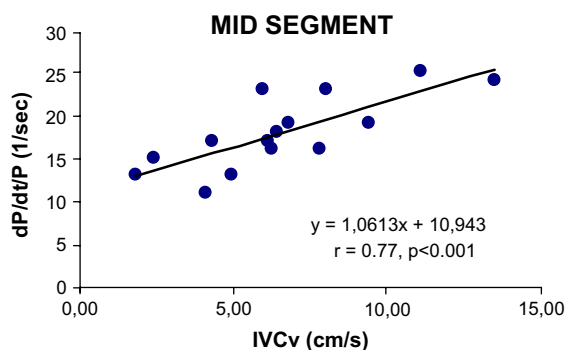
A significant relationship was found between $dP/dt/P_{max}$ and isovolumic contraction velocity at mid cavity of the right ventricular free wall ($r = 0.59$, $p < 0.01$, Fig. 2). Furthermore, when only patients with atrial pressure < 8 mmHg were taken into account the relationship of both basal and mid segmental levels were strengthened ($r = 0.69$, $p < 0.01$ and $r = 0.77$, $p < 0.001$, Fig. 3). The isovolumic contraction velocity at basal and mid segmental level did not correlate with neither right ventricular end diastolic dimension nor heart rate. We found no relationship between right ventricular systolic long axis, right ventricular area change, right ventricular outflow tract fractional shortening, Sv at any segment to $dP/dt/P_{max}$ neither in total group nor in the subgroup.

Right ventricular isovolumic contraction velocity and after-load dependence

Isovolumic contraction velocity measured from either basal or mid cavity segmental level did not
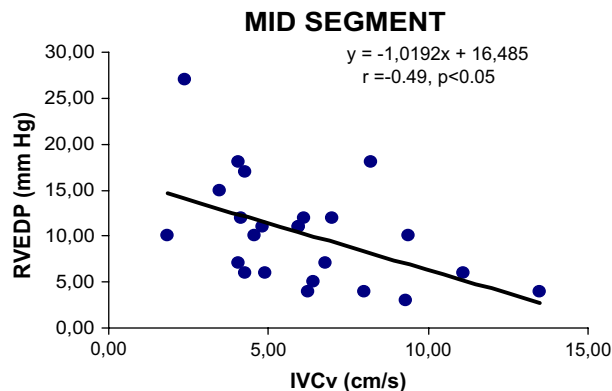
Figure 3 Correlation between isovolumic contraction velocities at the mid segment of the right ventricle and right ventricular $dP/dt/P_{max}$ in patients with right atrial pressures < 8 mmHg.

correlate to pulmonary artery systolic pressure or pulmonary artery resistance.

Right ventricular isovolumic contraction velocity and right ventricular filling pressures

Isovolumic contraction velocity measured from base and mid cavity level correlated significantly with right ventricular end diastolic pressure ($r = -0.47$ and $r = -0.49$, $p < 0.05$, for both, Fig. 4). We also found a relation between right atrial mean pressure and isovolumic contraction velocity at both segments ($r = -0.62$ and -0.54, $p < 0.01$, for both, Fig. 5). On the basis of the correlation between isovolumic contraction velocity at basal segment and right atrial mean pressure, we tested the sensitivity and specificity of a cut off value below 6 cm/s indicating an increased right atrial pressure (>6 mmHg). The sensitivity and specificity were 100% and 78%, respectively (Fig. 5).

Figure 2 Correlation between isovolumic contraction velocities at the mid segment of the right ventricle and right ventricular $dP/dt/P_{max}$ in all patients.

Figure 4 Correlation between isovolumic contraction velocity at the mid segment of the right ventricle and right ventricular end diastolic pressure in all patients.

BASAL SEGMENT

$$y = -0{,}7302x + 14{,}852$$
$$r = -0.62, p<0.01$$
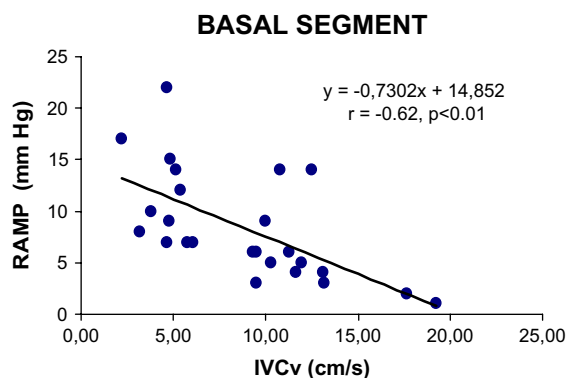
Figure 5 Correlation between isovolumic contraction velocities at the basal segment of the right ventricle and right atrial mean pressure in all patients.

Reproducibility of the data

Intra-observer and inter-observer variability of isovolumic contraction velocity measurements was tested by repeated measurements in 10 consecutive tracings. Variability was expressed as the coefficient of variation. Inter- and intra-observer variability for isovolumic contraction velocity was 7%, respectively.

Discussion

We have demonstrated that peak myocardial velocity of the right ventricular free wall, measured by pulsed Doppler tissue imaging, during the isovolumic contraction velocity phase can be used to estimate right ventricular contractility as well as right ventricular filling pressures. These findings may be of importance as disturbance of myocardial motion occurs predominantly during the isovolumic phases (i.e. contraction and/or relaxation), and may therefore be a sensitive marker for myocardial dysfunction.[5]

Cardiac anatomy

Buckberg[17] described in a recent review article the arrangement of cardiac fibres in terms of 2 different myocardial bands whereas the first spiral is designated as the basal loop and the second one as the apical loop. The basal loop fibres are running in a transverse or circumferential motion of the ventricles and perpendicular to ventricular long axis which is related to the apical loop. These 2 loops are proposed to be responsible for 4 cardiac motions including systolic narrowing and shortening followed by a diastolic lengthening and

widening. The chamber narrowing in transverse direction is starting from the right segment of the basal loop during isovolumic contraction. The spiral fibres of the right ventricle are located in the subepicardium.[18] Therefore, the velocity during the isovolumic contraction phase might well be derived from the initial transverse right ventricular free wall motion.

Right ventricular isovolumic contraction velocity and right ventricular contractility

An index of right ventricular contractility should ideally be sensitive to changes in the inotropic state but insensitive to heart rate as well as pre- and after-load conditions. Kaul et al. demonstrated a simple method to measure the systolic excursion of the tricuspid ring from base to apex.[19] This motion correlated with radionuclide derived ejection fraction. Recently, Meluzin et al. showed by using Doppler tissue imaging, that the peak systolic velocity of right ventricular myocardial free wall correlated to ejection fraction.[20] The major advantage of these measurements is the simplicity and the high reproducibility.[21,22] However, the use of peak systolic velocity in this respect is limited by being both load and heart rate dependent as well as being insensitive to alteration in the inotropic state.[8,23] dP/dt_{max} has traditionally been considered as a measurement of myocardial contractility because its maximum rate of pressure rise occurs before the aortic and pulmonary valve opening. The main limitation of dP/dt_{max} is its pre-load dependence as well as being influenced by cavity size and thickness index.[24] Therefore, $dP/dt/P_{max}$ was considered less sensitive to pre-load and a more reliable index for right ventricular contractility.[25]

In an animal study, Vogel et al. showed that IVC acceleration is a reliable measurement of right ventricular contractility and relatively load independent.[8] The study concluded that the acceleration was a more sensitive marker than IVC peak velocity. We found it difficult to accurately measure the acceleration of isovolumic contraction velocity as this is commonly multidirectional as supported by the anatomical explanation by Buckberg et al.[18] Therefore, we proposed that isovolumic contraction velocity would be the most appropriate measure.

In the present study, we found that isovolumic contraction velocity was influenced by the contractile state, especially at the mid cavity segments of the right ventricular free wall.

Right ventricular isovolumic contraction velocity and right ventricular filling pressures

Our present finding that isovolumic contraction velocity is dependent on the right ventricular end diastolic pressure indicates that isovolumic contraction velocity can be used to estimate filling pressures within certain limits. This is the consequence of the Frank Starling relationship where an increase in end diastolic pressure results in increased initial fibre (sarcomere) length. We propose that an isovolumic contraction velocity value below 6 cm/s can be used as a cut-off point for elevated filling pressures.

Study limitations

Although a small number of patients were investigated in this study, this was sufficient enough to disclose the physiological consequence of right heart failure. Most patients, however, suffered from severe heart failure, and only 14 showed normal right atrial pressures. In this study we used a thermodilution catheter; a tip manometer catheter with higher frequencies would have strengthened the results. However, the catheter used by us is a commonly used catheter in the daily clinical practice in our lab.

One should be conscious when interpretation IVCv in presence of elevated atrial pressure and presence of right bundle branch block since both elevated right atrial pressure and electromechanical disturbances are known to alter the isovolumic phases.[26,27] Therefore, the influence of different types of arrhythmias, bundle branch block and elevated atrial pressures needs to be further investigated.

Conclusion

Right ventricular contractility can be determined by measuring isovolumic contraction velocity, which is a reproducible and easily obtainable non-invasive parameter. Furthermore, isovolumic contraction velocity can also be useful in detecting patients with elevated right ventricular filling pressures.

Acknowledgements

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