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MEASUREMENT

Comparison of Doppler derived haemodynamic variables and simultaneous high fidelity pressure measurements in severe pulmonary hypertension

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Abstract

***Objective*—To assess relations between right ventricular pressure measured with a high fidelity transducer tipped catheter and the characteristics of tricuspid regurgitation recorded with Doppler echocardiography.**

***Design*—A prospective non-randomised study of patients with severe pulmonary hypertension referred for consideration of lung transplantation.**

***Setting*—A tertiary referral centre for cardiac and pulmonary disease, with facilities for invasive and non-invasive investigation, and assessment for heart and heart-lung transplantation.**

***Patients*—10 patients with severe pulmonary hypertension being considered for lung transplantation.**

***Endpoints*—Peak right ventricular, pulmonary artery, and right atrial pressures; peak positive and negative right ventricular dP/dt; peak Doppler right ventricular-right atrial pressure drop; Doppler derived peak positive and negative right ventricular dP/dt; and time intervals of Q to peak right ventricular pressure and to peak positive and negative right ventricular dP/dt.**

***Results*—The mean (SD) pulmonary artery systolic pressure was 109 (29) mm Hg. The peak Doppler right ventricular-right atrial pressure drop underestimated peak right ventricular pressure by 38 (21) mm Hg, and by 21 (18) mm Hg when the Doppler value was added to the measured right atrial pressure (P values < 0·05). This discrepancy was greater for higher pulmonary artery pressures. The timing of peak right ventricular pressure differed, with the Doppler value consistently shorter (mean difference 16 ms, P < 0·05). Values of peak positive and negative right ventricular dP/dt and the time intervals Q-peak positive right ventricular dP/dt and pulmonary closure to the end of the pressure pulse differed between the two techniques in individual patients, but not in a consistent or predictable way.**

***Conclusions*—Doppler echocardiography significantly underestimates the peak right ventricular pressure and the time interval to peak right ventricular pressure in pulmonary hypertension, particularly when severe. These differences may be related to orifice geometry. Digitisation of Doppler records of tricuspid regurgitation provides useful semi-quantitative estimates of absolute values and timing of peak positive and negative right ventricular dP/dt. Clinically significant differences may exist, however, and must be considered in individual patients.**

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Echocardiographic assessment of right ventricular function and Doppler estimates of peak right ventricular and pulmonary artery pressure are increasingly common requests to echocardiography departments, particularly from units caring for patients with pulmonary hypertension awaiting lung transplantation. Early studies suggested that Doppler estimates of the peak right ventricular pressure obtained from tricuspid regurgitation were extremely close to those obtained with catheterisation,[1–3] even if not studied simultaneously. Furthermore, we and others are increasingly using the spectral display of mitral and tricuspid regurgitation to estimate peak rates of rise and decline of ventricular pressure.[4–6] This application of Doppler echocardiography has not yet been widely validated and not at all in severe pulmonary hypertension.

During a study to monitor ambulatory pulmonary artery pressure over a 24 hour period, we had noticed significant discrepancies between the invasively derived pressure and that obtained by applying the simplified Bernoulli equation to tricuspid regurgitant flow in the same patients. Previous studies have not specifically looked at patients with severe pulmonary hypertension; instead, a wide range of values has been presented. In this study we wished to look specifically at patients with very severe pulmonary hypertension: all but one had right ventricular systolic pressures greater than 100 mm Hg. Furthermore, our aims were to assess the interrelations between right ventricular pressure recorded with a high fidelity transducer tipped catheter, and the characteristics of tricuspid regurgitation recorded with Doppler echocardiography. We

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were particularly interested to compare the values of peak right ventricular pressure obtained from the two techniques, peak positive and negative right ventricular dP/dt, and to assess possible discrepancies with regard to the timing of events.

Patients and methods

STUDY POPULATION

Ten patients (seven women, three men) with severe pulmonary hypertension being assessed for lung transplantation were studied. The mean (range) age was 33 (21–46) years. The diagnoses were primary pulmonary hypertension in seven patients, atrial septal defect in two, and chronic pulmonary thromboembolic disease in one patient. All patients had given written informed consent to the study, which formed part of a larger investigation aimed at monitoring pulmonary artery pressure using a Gaeltec high fidelity transducer tipped catheter over a 24 hour period. The study was approved by the local ethics committee. All patients had functional tricuspid regurgitation recordable with Doppler, but in none was the regurgitation severe in volume terms, nor were 'v' waves present in the right atrial pressure trace.

METHODS

High fidelity pressure recordings

A high fidelity micromanometer tipped catheter (Gaeltec, United Kingdom) was placed in the proximal left or right pulmonary artery under radiographic screening.[7] The signal from the pulmonary artery catheter was amplified (frequency response of catheter and amplifier was greater-than 1000 Hz (–3 dB), and displayed on the screen of the echocardiograph, together with the *simultaneously* acquired echocardiographic and Doppler information. Paper recordings of pulmonary artery and then right ventricular pressure were made with the left ventricular M mode, transtricuspid, transmitral and transpulmonary flow, and tricuspid regurgitant flow, all at 10 cm/s. At the conclusion of the study the catheter was withdrawn to the right atrium, and the phasic and mean right atrial pressure were measured. Patients with severe tricuspid regurgitation were excluded from this study, thus 'v' waves were not prominent. The catheter was then externally and immediately calibrated using an air operated dead weight pressure balance 239P (Budenberg Gauge, United Kingdom) (limit of accuracy of the pressure balance was better than 0·1% of the applied test mass) such that standard pressures of zero and 100 mm Hg were displayed as a square wave deflection on paper.

M mode and cross sectional echocardiography

M mode and cross sectional echocardiograms were obtained with the patient in the standard left lateral position, using a Hewlett-Packard Sonos 1000 echocardiograph with a 2·5 or 3·5 MHz transducer. Simultaneous phonocardiograms were recorded with a Leatham microphone, with a low frequency filter. M mode echocardiograms were recorded with simultaneous electrocardiograms and phonocardiograms on a Honeywell (Ecoline 22) strip chart recorder at a paper speed of 10 cm/s, or on the dedicated Hewlett-Packard chart recorder. Aortic valve closure (A_2) was taken as the onset of the first high frequency vibration of the aortic component of the second heart sound recorded on the phonocardiogram and was checked for validity with the aortic echogram and the aortic closure artefact on the Doppler recordings. Left ventricular internal cavity dimensions and septal and posterior wall thickness were measured at end systole (at A_2) and end diastole (onset of the QRS complex on the electrocardiogram), using leading edge methodology, from the parasternal long axis view. Isovolumic relaxation time for the left ventricle was measured from A_2 to the initial separation of the mitral cusps on the M mode echogram. All measurements were made on three cardiac cycles and the mean taken.

Doppler echocardiography

We recorded Doppler signals with a Doptek Spectrascan and a 2·0 MHz transducer and a Hewlett Packard Sonos 1000 with a 3·5 MHz transducer. Peak forward transmitral and transtricuspid flow velocities were identified using continuous waves from the apex and were recorded in pulsed mode with a 3 mm gate and a 250 Hz wall filter. The peak velocities of early (E wave) and atrial (A wave) transmitral flow were recorded and the ratio E/A was derived. Tricuspid regurgitant flow was identified and recorded in continuous mode from the apex. The peak instantaneous systolic pressure drop from right ventricle to right atrium was calculated from the peak velocity of the tricuspid regurgitant signal using the modified Bernoulli equation ($\Delta p = 4V^2$). The tricuspid pressure drop at the instant of mitral cusp separation was also recorded. The RR interval was measured from the electrocardiogram, recorded simultaneously with the phonocardiogram. P_2 was taken as the first high frequency vibration of the second component of the second heart sound and checked on the pulmonary echogram and pulmonary closure artefact on Doppler recordings. The time interval from pulmonary closure (P_2) to the onset of forward tricuspid flow was recorded in all instances. Data were recorded on paper at 10 cm/s; all measurements were made on three cardiac cycles and the mean taken.

Digitisation of records

M mode and Doppler echocardiograms were manually digitised using a Terminal Display Systems TDS 20 digitising tablet linked to a Hewlett-Packard Vectra 486 computer. The digitising program allows pressure and Doppler recordings to be calibrated and digitised from the same RR interval. From the Doppler data the program derives a pressure trace based on the simplified Bernoulli equation, along with peak right ventricular-right atrial pressure drop, peak positive and negative right ventricular dP/dt,

Table 1 Pressure estimates. Values are mean (SD)

	Catheter	Doppler echocardiography
Pulmonary artery systolic pressure (mm Hg)	109(29)	
Pulmonary artery diastolic pressure (mm Hg)	52(14)	
Right ventricular systolic pressure (mm Hg)	115(30)	77(30)* 94(30)†
Right ventricular diastolic pressure (mm Hg)	18(12)	

*Doppler right ventricular-right atrial pressure drop.
†Doppler right ventricular-right atrial pressure drop plus measured right atrial pressure.

and the time intervals of Q wave onset to peak right venticular pressure, and to peak positive and negative dP/dt. The mean right atrial pressure measured by catheter was added to the peak right ventricular-right atrial pressure drop to obtain an estimate of peak pulmonary artery pressure. Peak pulmonary artery pressure and peak positive and negative right ventricular dP/dt were measured from the digitised catheter recordings, as were the time intervals of Q wave onset to peak right ventricular pressure and to peak positive and negative dP/dt.

STATISTICAL ANALYSIS

The statistical methods used for assessing agreement between two methods of clinical measurement were those of Bland and Altman.[8] Differences between mean values were compared by Student's t test, and a P value of $< 0{\cdot}05$ was taken as statistically significant.

Results

PRESSURE ESTIMATION

Table 1 gives the mean values for pulmonary artery and right ventricular pressure. Figure 1 shows an example of a simultaneously acquired right ventricular pressure pulse and a Doppler flow velocity recording of tricuspid regurgitation. The peak right ventricular-right atrial pressure drop measured with Doppler echocardiography consistently underestimated the peak right ventricular pressure measured invasively, the mean difference being 38 (21) mmHg ($P < 0{\cdot}001$). Figure 2 compares these two measurements as a comparative data plot. In Figure 3, the invasively measured right atrial pressure is added to the Doppler derived right ventricular-right atrial pressure drop and plotted against the peak right ventricular pressure measured by catheter. The discrepancy is reduced, but nevertheless remains significant, with the Doppler values underestimating the catheter value by a mean of 21 (18) mm Hg ($P < 0{\cdot}01$). There was a tendency for the greatest discrepancies to occur with the highest pulmonary artery pressures. Figure 4 shows a plot of the magnitude of the difference in right ventricular pressure against the mean right ventricular pressure measured by the two techniques. The mean difference of 21 mm Hg is indicated by a horizontal line and this equates to an underestimate of approximately 20%. The regression equation is

Doppler RV-RA pressure drop + RA pressure = 0·8 catheter RV pressure − 0·2 mm Hg

The slope of this regression equation is statistically different from 1·0 ($P < 0{\cdot}05$); however, the intercept is not statistically different from zero.

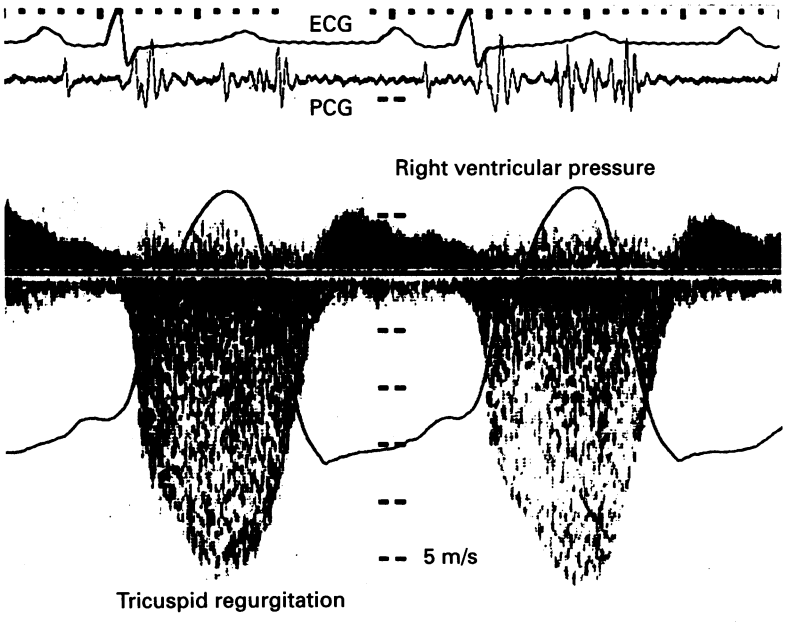

Figure 1 Simultaneously acquired high fidelity right ventricular pressure pulse with Doppler echocardiographic flow velocity display of tricuspid regurgitation from a patient with severe pulmonary hypertension. Simultaneous electrocardiogram (ECG) and phonocardiogram (PCG) are shown.

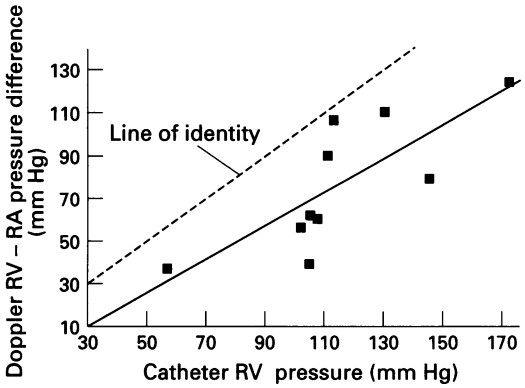

Figure 2 Comparative data plot of the Doppler derived peak right ventricular (RV)-right atrial (RA) pressure drop v the catheter peak right ventricular pressure. The regression line lies significantly below the line of identity and diverges at higher right ventricular pressure recordings.

DERIVED VARIABLES

Table 2 gives the values of peak positive and negative right ventricular dP/dt. No consistent differences between the two techniques existed although individual discrepancies did occur. Individual values for both peak positive ($r = 0{\cdot}81$; $p < 0{\cdot}01$) and negative ($r = 0{\cdot}78$; $p < 0{\cdot}01$) dP/dt were significantly correlated and related by the following regression equations

Doppler positive dP/dt = 87 + 1·03 catheter positive dP/dt

(standard error of intercept, 169 mm Hg/s; standard error of slope, 0·26; standard error of estimate, 163 mm Hg/s)

Doppler negative dP/dt = −110 + 0·99 catheter negative dP/dt

(standard error of intercept, 228 mm Hg/s;

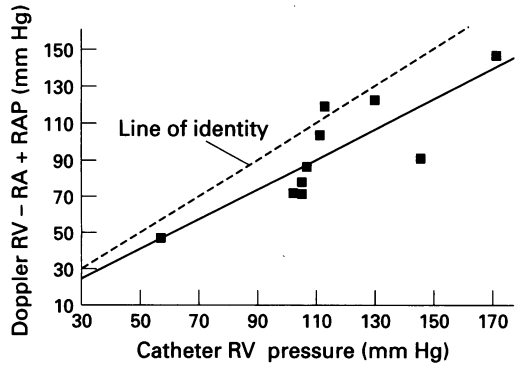

Figure 3 Comparative data plot of the Doppler derived peak right ventricular (RV)-right atrial (RA) pressure drop plus catheter measured right atrial pressure (RAP) versus the catheter peak right ventricular pressure. Once again the regression line is significantly below the line of identity.

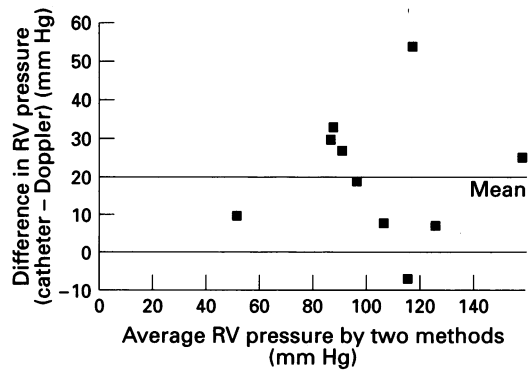

Figure 4 Bland-Altman plot of difference in right ventricular (RV) pressure measured by the two techniques v the average right ventricular pressure by the two methods.

standard error of slope, 0·28; standard error of estimate, 165 mm Hg/s). Neither of the slopes differed significantly from 1·0, nor the intercepts from zero.

TIMING OF EVENTS

The timing of peak right ventricular pressure with respect to the Q wave of the electrocardiogram also differed with the two techniques. The Doppler value was consistently less by a mean of 20 ms. There were not, however, significant or consistent differences between the methods in values of the time intervals of Q wave to peak positive and negative dP/dt, although small discrepancies did exist in individual patients. Table 3 gives the mean values from catheter and root mean square differences with Doppler. The value of the interval from pulmonary closure to the end of the ventricular pressure pulse, an interval we have previously shown to be directly related to the severity of the pulmonary hypertension,[9] differed by an insignificant amount between the two techniques.

LEFT VENTRICULAR FUNCTION

Left ventricular function was abnormal in our patients with a prolonged isovolumic relaxation time (80 (20) ms) and an abnormal transmitral Doppler A/E ratio (2·4 (2·8)). This we have previously shown to be directly related to a high right ventricular pressure at the instant of mitral valve opening.[10] Right ventricular pressure measured by catheter at this point was 38 (19) mm Hg, compared with a Doppler derived right ventricular–right atrial pressure drop of 21 (20) mm Hg ($P < 0·001$). The difference between methods became insignificant when the measured right atrial pressure was added to the Doppler right ventricular–right atrial pressure drop at the instant of mitral valve opening.

Table 2 Derived variables. Values are mean (SD)

	Catheter	*Doppler*
RV positive dP/dt (mm Hg/s)	625(211)	728(265)
RV negative dP/dt (mm Hg/s)	792(197)	673(249)

RV, Right ventricle.

Table 3 Timing of Events. Values are mean (SD)

	Catheter	*Doppler*	*Root mean square difference*
Q to peak RV pressure (ms)	275(25)	255(20)	*
Q to peak positive dP/dt (ms)	90(15)	95(10)	12
Q to peak negative dP/dt (ms)	380(30)	390(35)	16
Pulmonary closure to end of pressure pulse (ms)	125(45)	110(30)	15

*P < 0·05.
RV, Right ventricle.

Discussion

Doppler echocardiography has now become so widely used to assess valvular gradients and pressure drops that the principles on which such measurements are made are often forgotten. It is only when a large discrepancy occurs between an invasive and non-invasive measurement in an individual patient that clinicians address the principles on which estimates of pressure are made. The fact that invasive and non-invasive techniques measure different quantities has been compounded by the inappropriate use of the correlation coefficient to compare the two methods of clinical measurement.[8] Soon after Doppler echocardiography was introduced, it rapidly became adopted as a quick and accurate method for assessing pressure gradients. When applied to the estimation of right ventricular pressure, correlation coefficients of 0·96 and higher have been shown between catheter and Doppler measurements, with all measurements effectively lying on the line of identity.[2 11] In most studies, however, a range of patients with widely varying pulmonary artery pressures have been used. Data from such studies suggest that at the highest values of pulmonary artery pressure, Doppler echocardiography may underestimate the invasively determined pulmonary artery pressure[2]; this subgroup of patients has never been examined separately.

In our study we were not only interested in the relation between the peak right ventricular pressure measured by the two techniques, but also the derived variables dP/dt and time relations between the two pressure pulses. We found that Doppler echocardiography significantly underestimated the peak right ventricular pressure in patients with severe pulmonary hypertension and that the

discrepancy appeared to be greater (in absolute but not percentage terms) at the highest values of right ventricular pressure. The reasons for this discrepancy are not clear, but there are many possible explanations. We were careful to align perfectly with the jet of tricuspid regurgitation so as not to introduce an angle correction error. In our patients the right ventricle was dilated and formed the apex, so aligning with the tricuspid regurgitation was not difficult. Colour flow mapping was used to confirm the jet direction and clear spectral envelopes were obtained in every instance. The catheter measurements were made with a micromanometer tipped device with a high frequency response rather than fluid filled catheters, and were immediately and externally calibrated against a pressure balance, itself calibrated to UK National Physical Laboratory standards. Patients had only functional rather than haemodynamically significant tricuspid regurgitation and we cannot attribute the underestimates to an accentuated 'v' wave in the right atrial pressure. In any event, adding the 'v' wave to the peak pressure drop would tend to lead to an overestimate in peak right ventricular pressure because it occurs later in the cardiac cycle when the right ventricular pressure is decreasing.[1] We therefore believe the discrepancy to be genuine. In addition, underestimating the right atrial pressure would affect the intercept, not the slope, of the regression line between the invasive and non-invasive measurements. The intercept in our study is 0·2 mm Hg.

The more common error reported with Doppler echocardiographic assessment of pressure drops has been overestimation,[12] and explanations for this have been the fact that peak instantaneous gradients are by definition higher than peak to peak gradients measured by catheter withdrawal, and that velocities proximal to the stenosis are neglected by the Bernoulli equation. In addition, it has been postulated that localised gradients may not truly reflect the average gradient across the whole valve orifice.[13] It is more difficult to explain underestimates by Doppler echocardiography. For pulsatile flow in the cardiovascular system, the pressure difference between two points is derived from the simplified Bernoulli equation on the principle of the conservation of energy.[14] When applying the Bernoulli equation to clinical situations, a number of simplifications are made. The acceleration term (that is, the acceleration between the two points) from the full equation is assumed to be zero at peak systole and this is of course when the peak right ventricular-right atrial pressure drop occurs. It is well recognised that the simplified Bernoulli equation is invalid when proximal velocities cannot be ignored, or when stenoses occur in series. Neither of these apply in our patients. The problem may indeed be much more fundamental. In basic fluid dynamics it is well recognised that the peak velocity predicted by the simplified Bernoulli equation predicted from the pressure drop is not recorded across an orifice. The orifice coefficient (C) is therefore defined as

$$C^2 \cdot \Delta p = 4V^2$$

where Δp is the pressure drop between the two points.[15] An orifice coefficient of exactly 1·0 is uncommon, though this value is assumed for all cardiological applications. The coefficient itself depends critically on whether the orifice edges are sharp or rounded, or whether the region of constriction is short or tube-like.[15] If the tricuspid orifice coefficient at peak systole was less than 1·0—for example, 0·9—then the pressure difference calculated by the Bernoulli equation would be $C^2 = 0{\cdot}81$ of the true pressure drop, and this is just the order of magnitude of our underestimate. It is, in fact, highly unlikely that the tricuspid orifice has a coefficient of 1·0 and it is thus surprising that the slopes of the regression equations of invasive and non-invasive measurements have been so close to 1·0.[2 11]. In fact, using the magnitude of the pressure differences which might be expected in pulmonary hypertension, the approximate jet velocities and orifice areas, then from graphs of orifice meter coefficients, we might expect values of C to be 0·8–0·9.[15] Such calculations, however, are based on steady flow in straight circular tubes with rigid, sharpedged, circular orifices. Exactly how the orifice coefficent disturbs conventional Bernoulli calculations can only therefore be postulated, but it would seem that our data are entirely explicable on this basis. Segal *et al* reported directly measured orifice coefficients from an experimental model of aortic stenosis, using Plexiglass flow plates and restrictive nozzles, and a modified xenograft.[16] With the xenograft they obtained orifice coefficients of 0·9 at high Reynolds numbers and this would explain our results exactly.

The situation is even more complex when we try to extrapolate data from throughout the cardiac cycle. There is evidence that the orifice underlying functional mitral regurgitation changes size during the cardiac cycle,[17] and the same is likely to be true for the right side of the heart. If the shape changed as well then we might expect that the orifice coefficent might itself change throughout the cardiac cycle. This situation is very different to the concept of restrictive flow produced by fixed organic regurgitation, either occurring as the result of disease, or experimentally by inserting a grommet into a leaflet of the mitral valve.[5] In these situations, the orifice is fixed throughout the cardiac cycle and its nature is more predictable. Caution may be required therefore when extrapolating data from such models to the more complex situation in functional regurgitation.

From our data it is thus important to realise that the true right ventricular pressure may be approximately 20% higher than the Doppler echocardiographic estimate, but this does not necessarily invalidate the use of Doppler echocardiography for individual patient management. Furthermore, when arriving at the Doppler estimate in patients

with pulmonary hypertension, a minimum of 15 mm Hg should be added to the peak right ventricular − right atrial pressure drop (rather than the conventional 10 mm Hg), as the mean right atrial pressure is usually at least this.

We and others are increasingly using spectral Doppler displays of atrioventricular valve regurgitation to obtain the peak rates of ventricular pressure rise and decline.[4–6] We found no significant differences in the values obtained for these variables assessed by the two techniques. Individual discrepancies did occur, being of the order of 180 mm Hg/s, but these were not consistent. Thus using Doppler to assess right ventricular positive and negative dP/dt appears to be valid, at least semiquantitatively, and may be useful in following the effects of interventions on the rate of rise or decline of pressure. With regard to the timing of events, the right ventricular pressure pulses obtained by the two techniques were similar. Nevertheless, the value of the time interval of Q wave to peak right ventricular pressure was slightly but significantly less when measured from Doppler echocardiography than from the catheter recordings. There was no significant difference in determining any other time intervals, particularly that between pulmonary closure and the end of the pressure pulse, which we have previously shown to be directly related to the right ventricular pressure.[9]

In this study we were able to confirm the effects of prolonged right ventricular systole on left ventricular diastolic function, which we and others have shown to be abnormal in pulmonary hypertension.[10 18 19] Our patients had abnormally prolonged left ventricular isovolumic relaxation and an abnormal pattern of transmitral flow. The invasively measured right ventricular pressure at the instant of mitral valve opening was 38 mm Hg, thus reinforcing our previous observation that a prolonged decrease in right ventricular wall tension causes significant septal displacement and thus interferes with left ventricular filling.

Thus the peak tricuspid regurgitant velocity measured by Doppler echocardiography significantly underestimates the peak right ventricular pressure by around 20% in severe pulmonary hypertension. The method gives semiquantitative estimates of the peak rates of right ventricular pressure rise and decline, but the agreement in the timing of events is much closer. There is no doubt that Doppler echocardiography should remain the major non-invasive method for investigating such patients. Knowing its limitations will strengthen the technique as a clinical tool, an approach that seems preferable to the currently widely held belief that the values it gives are interchangeable with invasive measurements.

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