Non-Invasive Estimation of Mean Pulmonary Artery Pressure Using Pulsed Doppler Echocardiography (P.D.E.).

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SUMMARY

In an attempt to evaluate the reliability of Pulsed Doppler Echocardiography to predict mean pulmonary artery pressure, 18 patients with Valvar Heart Disease were assessed both by P.D.E. and cardiac catheterisation. Mean P.A. pressure was calculated using the formula:– Mean PA Pressure = 79 - (0.45 x.A.T.) where AT is acceleration time. The mean PA pressure was correctly predicted in 15/18 patients (83%). In I, it was grossly overestimated and in the remaining 2, it was slightly underestimated. (Mean PA Pressure÷Mean Systemic Pressure) Ratio was also evaluated. P.D.E. correctly estimated this ratio in only 11/18 (61%) and was slightly overestimated in 2(11%) and slightly underestimated in 5(28%). Thus mean PA pressure could be reliably predicted by P.D.E. but the Mean PA Pressure÷Mean Systemic Pressure Ratio was a less reliable index.

INTRODUCTION :

Traditionally cardiac catheterisation has been the mainstay in the accurate assessment of the severity of valve disease and pulmonary hypertension. M-mode and 2–D echocardiography was then used as an adjunct in the evaluation of the valve disease, but it has not replaced cardiac catheterisation. In Pakistan, where there is a high incidence of Rheumatic Heart Disease, there is a great need for a reliable non-invasive procedure to replace cardiac catheterisation. The advent of Doppler Ultrasonography has been a step in this direction. Using pulsed Doppler Echocardiography (P.D.E.), the severity of valvar stenosis and regurgitation can be assessed fairly accurately[1] but for the measurement of higher gradients, Continuous Wave Doppler (C.W.D) is required[2]. This latter technique can also be utilised to estimate ventricular systolic pressures. Recently, using a formula developed by Mahan et al[3], it has become possible to estimate the mean pulmonary artery pressure. If mean pulmonary artery pressure could be reliably estimated, then combined with the estimation of valve stenosis and regurgitation, P.D.E could replace cardiac catheterisation, a procedure not available at all the cardiac centres in Pakistan.

In this study, 18 patients with valvar heart disease underwent assessment by P.D.E followed by cardiac catheterisation. The purpose of this paper is to evaluate the reliability of P.D.E in the estimation of pulmonary artery pressure compared with those obtained at cardiac catheterisation.

POPULATION AND METHODS

Eighteen patients with valvar heart disease of different types underwent Pulsed Doppler Echocardiography (P.D.E) immediately prior to cardiac catheterisation. Three were 9 males and 9 females ranging in age between 14 and 33 years (mean 23 years). Seven of the patients had severe

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rheumatic mitral stenosis, 2 had severe mitral regurgitation, 6 mixed mitral and aortic valve disease, 2 mixed aortic valve disease and 1 atrio-ventricular septal defect. P.D.E. was performed using Honeywell Ultraimager with 2.25 or 3.5 MHz transducers. The sample volume was placed in the distal main pulmonary artery for the recording of the pulmonary flow. The pulmonary artery (PA) flow acceleration time (AT) was measured and an average of 4 values calculated. The mean PA pressure was then calculated using the formula[3].

Mean PA Pressure = 79—0.45 x A. T
where AT = time from the onset of ejectionto the peak velocity.

Systemic arterial mean blood pressure was calculated from the brachial artery pressure by the formula :

$$\text{Mean Arterial Pressure} = \text{Diastolic Pressure} + \frac{(\text{Systolic} - \text{Diastolic})}{3}$$

$$\frac{\text{The mean PA pressure}}{\text{mean systemic pressure}}$$ ratio was then obtained.

Right and left heart catheterisation was performed using the standard transfemoral Judkins percutaneous technique.

RESULTS:

Mean PA Pressure (Table – I)

In the whole group the average of the mean PA pressures was 38 ± 12 mmHg estimated by P.D.E. compared with 37 ±15 mmHg measured at cardiac catheterisation.

Three patients with normal mean PA pressures (< 22mmHg) at cardiac catheterisation were correctly identified by PDE.

Five patients were found to have mild to moderate pulmonary hypertension (mean PAP 22—40 mmHg) on P.D.E. This was confirmed at catheterisation in all except one case in whom the mean PA pressure was found to be lower (16 mmHg).

In 10 patients, P.D.E. predicated the presence of fairly severe pulmonary hypertension (mean PAP > 40 mmHg). Cardiac catheterisation confirmed this finding in all except 2 patients in whom the mean PA pressures were in the moderate pulmonary hypertension range (36 and 26 mmHg).

TABLE - I

PATIENT S. NO.	AGE	Mean PA Pressure mmHg DOPPLER	CATHETER	Mean PAP/Mean Systemic Ratio DOPPLER	CATHETER
1.	14	45	60	0.52	0.75
2.	28	45	45	0.45	0.56
3.	21	20	22	0.22	0.28
4.	32	53	45	0.73	0.66
5.	28	52	58	0.60	0.72
6.	30	28	38	0.38	0.50
7.	26	45	45	0.54	0.51
8.	25	25	22	0.40	0.44
9.	16	43	36	0.50	0.48
10.	15	42	50	0.55	0.67
11.	22	40	16	0.46	0.27
12.	15	50	58	0.55	0.82
13.	26	35	32	0.35	0.45
14.	33	45	26	0.58	0.35
15.	30	40	28	0.43	0.46
16.	15	10	9	0.12	0.10
17.	21	43	52	0.59	0.74
18.	15	20	22	0.50	0.55
Mean	23 ± 6 Years	38 ± 12	37 ± 15 Years	0.47 ± 0.14	0.52 ± 0.18

Thus, the mean PA pressure was correctly predicted in 15 out of 18 patients (83%). Out of the remaining 3, P.D.E grossly overestimated the mean PA pressure in only I patient (5.5%) and only slightly underestimated in 2 patients (11%).

$\frac{\text{Mean PA Pressure re}}{\text{Mean Systemic Pressure}}$ Ratio (Table – I)

This ratio was calculated for comparison with cardiac catheterisation to judge the severity of pulmonary hypertension in relation to the systemic pressure.

The overall average of this ratio on P.D.E was 0.47 ± 0.14 compared with 0.52 ± 0.18 at cardiac catheterisation.

Normal pulmonary pressure was defined by $\frac{\text{mean PA Pressure}}{\text{mean systemic pressure}}$ ratio of less than 0.4. Four patients had a ratio of < 0.4 at cardiac catheterisation. Two (50%) of these were correctly identified by P.D.E, and in the remaining 2, P.D.E slightly overestimated the ratio (0.46 and 0.58).

Eight patients had a ratio 0.4 – 0.6 at catheterisation. P.D.E. correctly predicted this ratio in 6 (75%) patients. In the remaining 2 (25%) the ratio was slightly underestimated (0.38 and 0.35).

Six patients had a ratio > 0.6 at catheterisation. In 3(50%), the ratio was correctly estimated by P.D.E and in the remaining 3 (50 %), the ratio was slightly underestimated (0.52, 0.55 and 0.55).

Thus P.D.E correctly estimated this ratio in

11/18 (61%) of the cases, and appeared to be less reliable an index of severity of pulmonary hypertension. It slightly overestimated in 2 (11%) and slightly underestimated in 5 (28 %).

DISCUSSION

Pulmonary artery pressure has been estimated using Doppler Ultrasonography in a variety of clinical settings, including tricuspid regurgitation & ventricular septal defects, with or without pulmonary valve stenosis[4]. However, these techniques may utilise the newer technique of continuous wave Doppler, which is not available in all the centres.

In pulmonary hypertension, the shape of the pulmonary flow velocity curve changes. The peak velocity may not differ from normal but the time taken to reach the peak velocity may be reduced[2]. Another method estimates the presence of pulmonary hypertension by dividing the acceleration time (AT) by the right ventricular ejection time[1,5]. The ratio of pre-ejection to ejection time (ET) may give an indication of the pulmonary artery diastolic pressure[1,5].

Mahan et al[3] measured the AT, ET and peak flow velocity and compared these with the mean PA pressure, logarithm mean PA pressure and pulmonary vascular resistance. They found that the best corelation was between mean PAP and AT and that the A.T could be used to estimate the mean PAP by the formula :—

Mean PA Pressure = 79 – (0.45 x A.T).

Our study was undertaken in patients undergoing cardiac catheterisation for valvar disease of different types. We found that the absoulte mean PA pressure was fairly correctly estimated in nearly 95% of cases by P.D.E. However, the mean PAP – mean systemic = ratio was less accurately predicted by P.D.E, being correct in 61% and either slightly underestimated or overestimated in the remaining 39%.

In conclusion, P.D.E is a reliable non-invasive method of estimating the mean P.A pressure. Further more, when combined with the ability of P.D.E to assess the severity of valvar stenosis or regurgitation, this technique should further reduce the need for cardiac catheterisation in patients with valvar disease of any aetiology.

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