

Non-invasive estimation of pulmonary artery systolic pressure with Doppler ultrasound

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SUMMARY Systolic pressure in the pulmonary artery was estimated from the interval between pulmonary valve closure and tricuspid valve opening, and the heart rate using a nomogram previously described. The timing of valve movements was recorded by Doppler ultrasound. The estimated pressure correlated well with that obtained at catheterisation in 45 of 48 patients with pulmonary hypertension. Instantaneous variations in pressure and changes with treatment and during exercise could be measured. The method was easy to apply in all age groups, and was found useful both in detecting pulmonary hypertension and in the follow-up of patients. It may help to determine the optimal time for surgery or the effect of treatment.

A non-invasive method for the estimation of pulmonary artery pressure based on the duration of the interval between pulmonary valve closure and tricuspid valve opening (P_c-T_o interval) was described by Burstin.¹ Pulmonary valve closure was determined from the phonocardiogram and tricuspid valve opening from jugular venous tracing or right ventricular apexcardiogram. The pressures estimated from a nomogram based on the P_c-T_o interval and the heart rate correlated well with those obtained at catheterisation.

In this study Doppler ultrasound was used to record pulmonary valve closure and tricuspid valve opening—and Burstin's nomogram was used to estimate pulmonary artery pressure in subjects with and without pulmonary hypertension.

Subjects and methods

The method¹ rests on the assumption that the P_c-T_o interval increases proportionally with the systolic pressure in the pulmonary artery (Fig. 1) and is inversely related to heart rate. The rate of fall of right ventricular pressure during isometric relaxation is assumed to be proportional to heart rate—but constant regardless of the height of right ventricular pressure. An increase in right atrial pressure will shorten the interval, but this pressure remains within narrow limits for a long period in

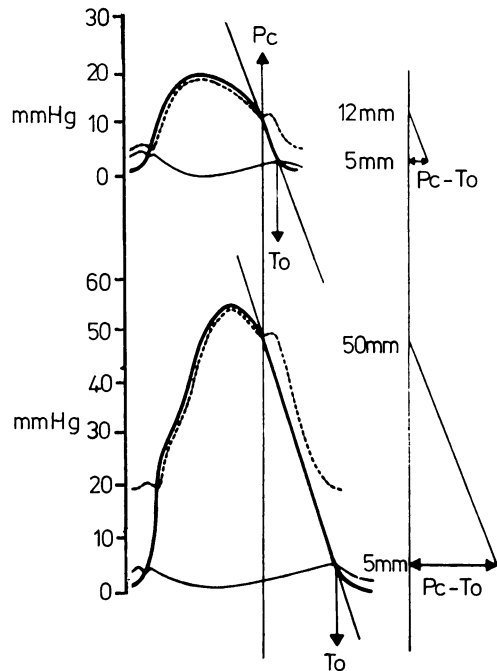


Fig. 1 The proposed mechanism for prolongation of P_c-T_o interval with normal and raised systolic pulmonary artery pressure. Right atrial, right ventricular, and pulmonary artery pressure. (Reproduced from Burstin¹ by permission.)

Received for publication 16 January 1980

most cases with pulmonary hypertension. The estimation of pulmonary artery pressure will thus be significantly vitiated only by severe right heart failure or tricuspid regurgitation. Fig. 2 shows the nomogram produced by Burstin.

In the present study the P_c-T_0 interval was obtained by using the Doppler ultrasound technique. The instrument used is described in detail elsewhere.^{2,3} The ultrasonic frequency is 2 MHz. It can be used in either a pulsed or a continuous mode. In the pulsed mode velocities are measured in a cylindrical volume of 7 mm diameter and 7.5 mm length. The instrument has a mean and a maximum frequency estimator. Mean velocity (directional) and maximum velocity (non-directional) as well as the amplitude of the Doppler signal can be recorded simultaneously on an ordinary paper recorder.

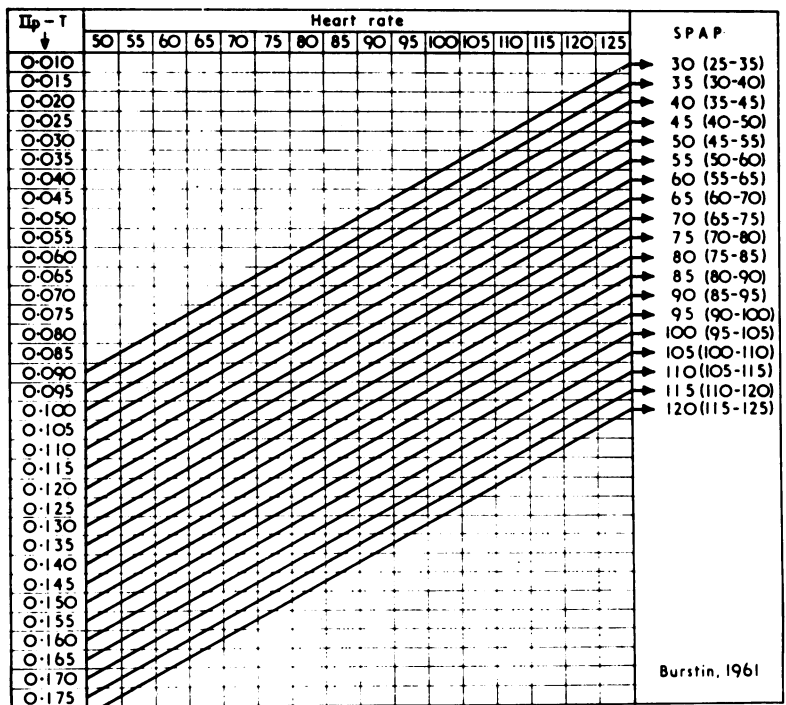
The audible Doppler signal from moving valves can be clearly distinguished from that of blood flow, but cannot always be clearly separated on the velocity tracings; the amplitude of the Doppler signal easily distinguishes the two since valve movements reflect far more energy (Fig. 3).

The ultrasonic recordings were made from the left sternal border, locating tricuspid and pulmonary flow by directing the transducer to the

right and upwards, respectively. The position and depth were adjusted until both blood flow and valve movements were clearly heard. Tricuspid and pulmonary flow were then recorded on an Elema Mingograph together with the electrocardiogram and phonocardiogram at a paper speed of 100 mm/s. Usually, both mean and maximum velocity were recorded together with the amplitude of the Doppler signal to ensure optimal position, and recordings were made on both sides of the valves to detect possible valve stenosis or regurgitation. As the measured P_c-T_0 interval varied slightly with respiration a mean of 10 beats was used; in atrial fibrillation the mean of 20 beats was taken. Systolic pulmonary artery pressure was estimated from the P_c-T_0 interval and heart rate using Burstin's nomogram. Extrapolation was made when the heart rate exceeded 125 (Fig. 2).

Since pulmonary and tricuspid flow were not recorded simultaneously, the phonocardiogram was useful, especially in atrial fibrillation with its varying length of systole. Localising pulmonary valve closure from the phonocardiogram alone was not always easy in adult patients. The timing of pulmonary valve closure on the amplitude curve was noted on the phonocardiogram and then used to identify pulmonary closure on the phonocardiogram

Fig. 2 Table produced by Burstin to calculate systolic pulmonary artery pressure from P_c-T_0 interval and heart rate. It is based on a proportional increase in this interval with increasing pressure and an inverse relation to heart rate. (Reproduced from Burstin, by permission.) SPAP—systolic pulmonary artery pressure.



recorded with the tricuspid flow curve.

Right heart catheterisation was carried out as previously described⁴; some patients were studied before surgery, others during the evaluation of vasodilator treatment for chronic left ventricular failure.

The P_c-T_o interval was recorded in 15 normal subjects, in 50 patients with various forms of heart disease but without pulmonary hypertension, and in 48 patients with pulmonary hypertension verified by heart catheterisation. In 14 of the patients with pulmonary hypertension, pressure and ultrasonic recording were done simultaneously. In the others the ultrasonic recording was made on the day before catheterisation in most, but in a few some days before.

Repeat ultrasonic recordings were done in 14 clinically stable patients, in 12 patients during treatment for heart failure, and in one premature infant with ventricular septal defect and pulmonary hypertension. Of the 14 stable patients, six had chronic heart failure caused by coronary heart disease in four and congestive cardiomyopathy in two, four had mitral regurgitation, one pulmonary heart disease, one primary pulmonary hypertension, and two congenital heart disease. Of the 12 patients being treated for heart failure, eight had coronary heart disease with previous myocardial infarction, two had congestive cardiomyopathy, one had aortic valve replacement for aortic regurgitation and one

had mitral valve disease. In seven patients measurements were done both before and after operation. In 12 patients with mitral stenosis the P_c-T_o interval as well as the maximal velocity in the mitral jet were recorded both at rest and during exercise. The pressure drop across the valve can be calculated from maximal velocity in the mitral jet.^{4,5} Patients with severe right heart failure or with significant tricuspid regurgitation were not included.

Results

Fig. 3 and 4 show the ultrasonic recording of pulmonary and tricuspid flow in one patient with normal and one with raised systolic pulmonary artery pressure. The mean velocity curve in Fig. 3 shows that pulmonary flow is recorded away from the transducer (negative) while tricuspid flow is recorded towards the transducer (positive). Valve movements are best seen in the lower curve (amplitude). Pulmonary valve opening is not shown here. By recording 0.5 to 1 cm farther away from the transducer, the valve opening can be clearly seen, but the closing movement is then less prominent. The difference in P_c-T_o interval between a normal subject (Fig. 3) and a patient with pulmonary hypertension (Fig. 4) is clearly seen. The varying P_c-T_o interval with atrial fibrillation is seen in the latter, changes with respiration usually being of smaller degree.

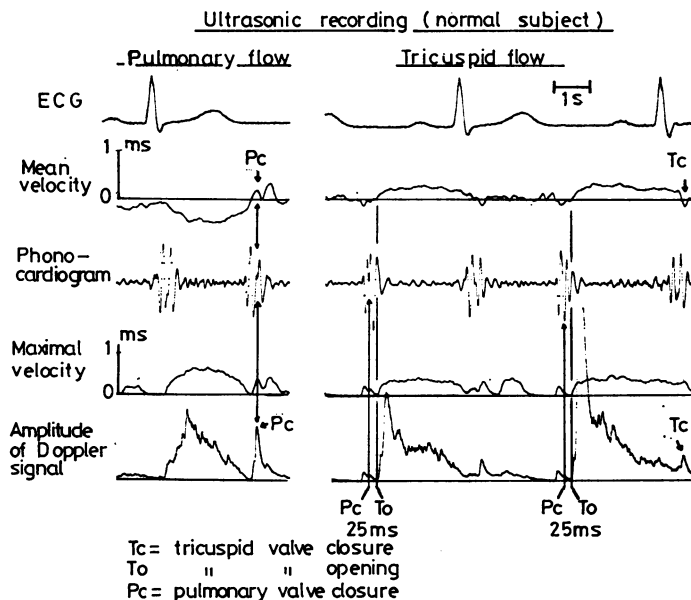


Fig. 3 Pulmonary and tricuspid flow recorded non-invasively by Doppler ultrasound. Mean velocity is directional, maximal velocity non-directional. Valve movements are best seen in the lowest curve. The P_c-T_o interval is 25 milliseconds (ms).

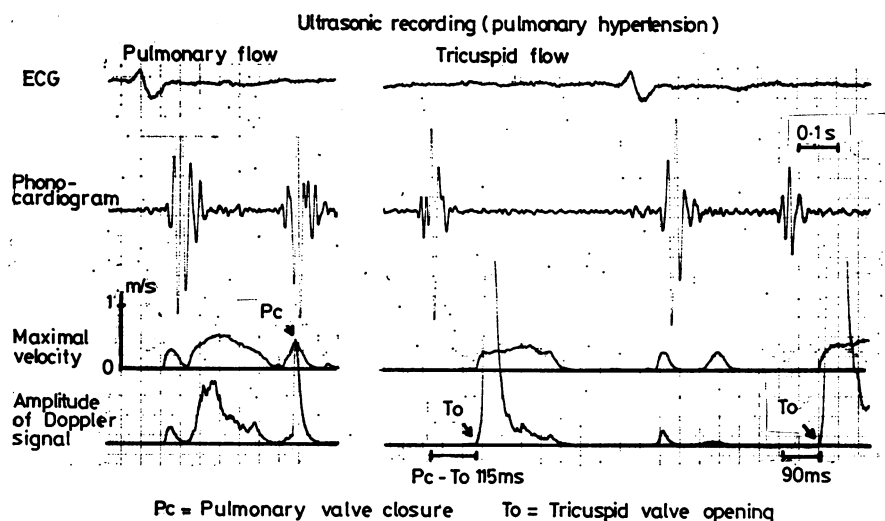


Fig. 4 Pulmonary and tricuspid flow in a patient with pulmonary hypertension. The P_c-T_o interval is prolonged.

CONTROLS

In the 15 healthy subjects (aged 4 to 29 years), the P_c-T_o interval was 10 to 50 (mean 22) ms. It was above 30 ms only in two with heart rates between 50 and 60 beats/min. In 25 children aged 1 to 17 years with various forms of heart disease and normal pulmonary artery pressures at catheterisation, the P_c-T_o interval ranged from 10 to 35 ms (mean 17 ms) with varying heart rates, indicating a normal systolic pulmonary artery pressure in all. In 25 adults aged 20 to 68 years with valvular or coronary heart disease and normal pulmonary artery pressure at catheterisation, the P_c-T_o interval ranged from 10 to 60 ms with varying heart rates, indicating a pulmonary artery systolic pressure below 30 mmHg in all.

PULMONARY HYPERTENSION

Of the 48 patients with pulmonary hypertension, seven were children and 41 were adults, with an age range of 1 to 75 (mean 51) years. The cause of pulmonary hypertension was valvular heart disease in 22, left ventricular failure caused by coronary heart disease or cardiomyopathy in 16, congenital heart disease in seven, pulmonary disease in two, and primary pulmonary hypertension in one.

Fig. 5 compares the pulmonary artery pressure estimated by Doppler ultrasound with that obtained at catheterisation. There was good correlation in most instances, particularly when the readings were obtained simultaneously rather than on different days. One patient, a child, had a heart rate of 160

which was above that found in the nomogram. With extrapolation the estimated pressure was very close to that obtained at catheterisation (90 and 87 mmHg, respectively). In three patients, however, there was a discrepancy between the pulmonary artery pressure obtained by ultrasound and by catheterisation. In patient (a) who was admitted with acute mitral regurgitation and severe pulmonary oedema, ultrasound performed on the day of admission disclosed a very high pulmonary artery pressure, but two days later when the clinical condition was much improved the pressure measured at catheterisation was lower. The lower pulmonary artery pressure recorded at catheterisation in patient (b) is attributable to sedation given for this procedure but not for the ultrasound examination. In the third patient (patient c) there was no apparent cause for the lower recording obtained on ultrasound.

Fig. 6 shows how closely changes in the P_c-T_o interval may follow changes in right heart pressure. In this patient with cor pulmonale and greater than usual variation in right ventricular pressure with respiration the P_c-T_o interval showed cyclic variations of 50 to 70 ms with respiration, with minimal changes in RR interval. This indicated a pulmonary artery systolic pressure varying between 42 and 60 mmHg. The actual change in the simultaneously measured right ventricular pressure was 47 to 59 mmHg. Similar but greater variations in pressure were seen in one patient with Cheyne-Stokes respiration where changes in P_c-T_o intervals

during periods of hypernoea and apnoea indicated corresponding changes in pulmonary artery systolic pressure ranging from 40 to 75 mmHg. The same variation was found on direct pressure recording.

REPEAT MEASUREMENTS

In the 14 clinically stable patients the ultrasonic recording and estimation of pulmonary artery pressure were repeated one or several times a day on consecutive days (Fig. 7). The repeat measurements showed only small variations in individuals. Twelve patients were followed during the treatment of heart failure (Fig. 8). The fall in pulmonary artery systolic pressure after vasodilator treatment was confirmed by direct pressure recordings.

Fig. 9 shows the results of repeat ultrasonic recording in a prematurely born child with ventricular septal defect. A gradual decline in pressure is seen, except for the recording at 12 months of age when he was crying and less co-operative than on the other occasions.

In seven patients, measurements were done both before and after operation. Fig. 10 is taken from a patient with mitral regurgitation. The estimated pulmonary artery systolic pressure before operation was 70 to 75 mmHg. Three weeks after mitral valve replacement the estimated pulmonary artery pressure was <30 mmHg. Table 1 shows the results in all seven patients treated surgically, an early,

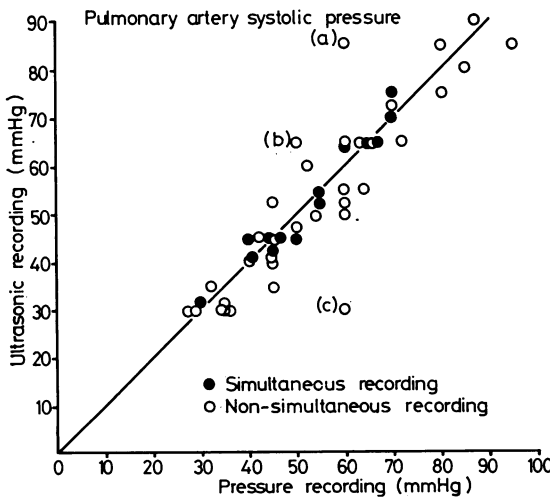


Fig. 5 Pulmonary artery systolic pressure estimated from ultrasonic recording of P_c-T_o interval compared with pressure recording. Correlation coefficient 0.89. Simultaneous measurements were done in 14 patients. See text for comments on patients marked (a), (b), and (c).

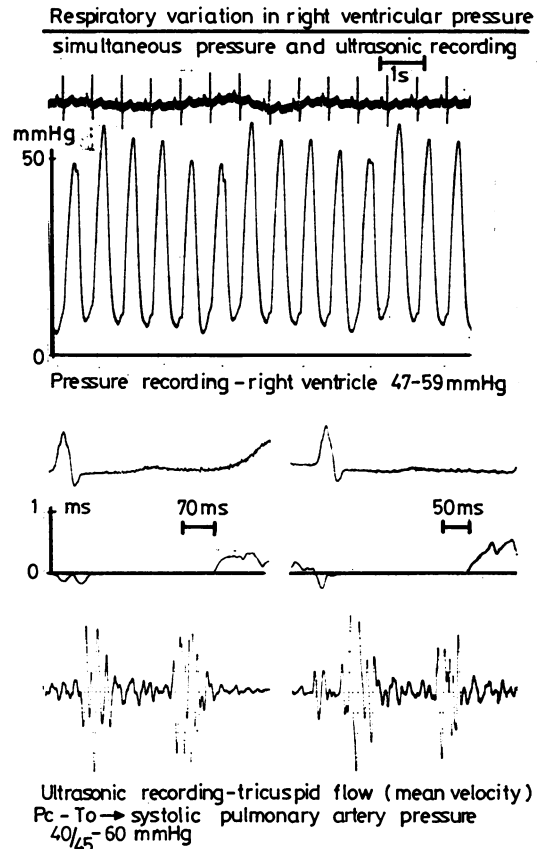


Fig. 6 Simultaneous recording in patient with pulmonary hypertension caused by pulmonary disease. Both methods showed the increased variation in pressure with respiration.

sizeable decrease in pulmonary artery pressure being seen in most. In two patients (cases 6 and 7), one of whom had been in cardiac failure for more than a year before operation and the other who still had a residual left-to-right shunt, the pressure fall was slower and less dramatic.

EXERCISE

In 12 patients with mitral stenosis, pulmonary artery systolic pressure was estimated both at rest and during exercise (bicycling in the supine position). Tricuspid flow was easily recorded during exercise, and pulmonary flow was also recorded in most patients. In the remaining patients, the phonocardiogram was used to determine pulmonary valve closure during exercise (pulmonary flow was recorded at rest in all). The P_c-T_o interval shortened with exercise, but with increases in heart rate the

estimated pressure increased in nine of the 12. Table 2 shows the increase in mean pressure drop across the mitral valve with exercise as well as the increase in estimated pulmonary artery systolic pressure.

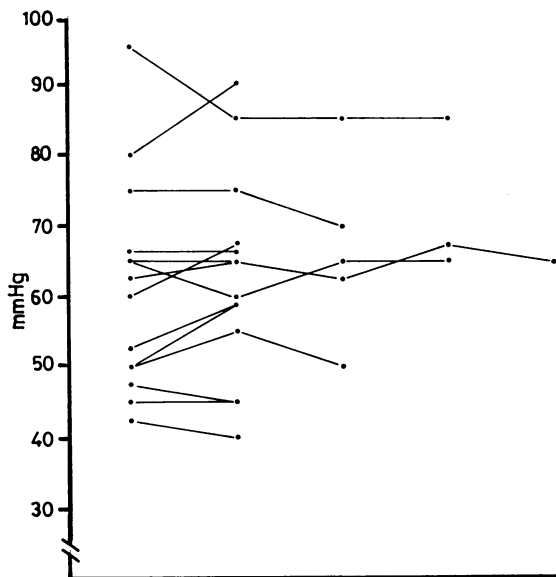


Fig. 7 Estimated pressure from repeat ultrasonic recording of P_c-T_o interval in 14 patients.

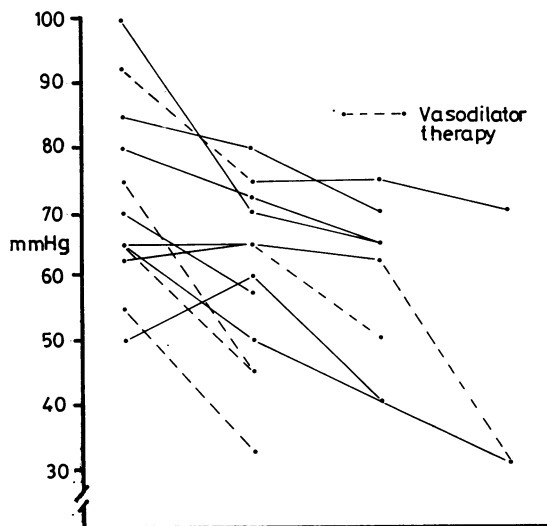


Fig. 8 Repeat estimations during treatment in 12 patients with left heart failure. In patients with vasodilator treatment direct pressure recording confirmed the non-invasively estimated fall in pressure.

SOURCES OF ERROR

The presence of significantly raised end-systolic right atrial pressure in right heart failure or tricuspid regurgitation will diminish the pressure difference between pulmonary artery and right atrium. If this is not taken into account the estimation of pulmonary artery pressure may be too low. Two other possible sources of error are the presence of either pulmonary regurgitation or cardiac failure with a very low cardiac output. In the upper part of Fig. 11 flow towards the transducer is seen shortly after the second heart sound when recording in the right ventricle 4 cm from the transducer, but tricuspid valve opening is recorded later in diastole. The patient had pulmonary hypertension with pulmonary regurgitation which caused flow towards the transducer before tricuspid valve opening. The recording at 5 cm depth, that is closer to the tricuspid orifice, shows only tricuspid flow. Since pulmonary regurgitation is easily detected by Doppler ultrasound this error should easily be avoided.

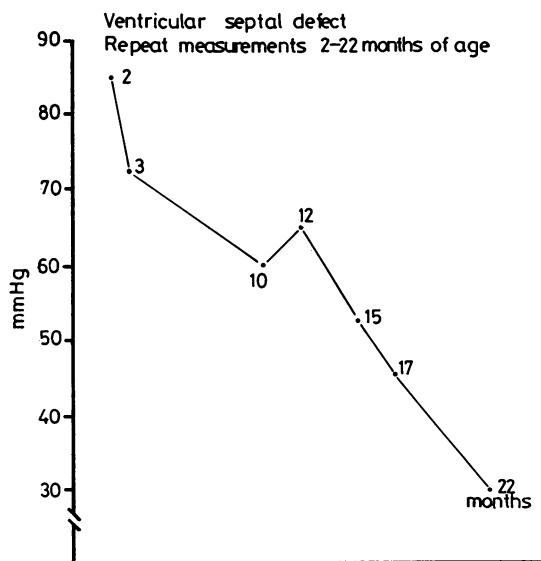


Fig. 9 Repeat ultrasonic recording of P_c-T_o interval in a child with ventricular septal defect.

The lower part of Fig. 11 is taken from a patient with severe left ventricular failure, very low cardiac output, and diastolic filling occurring mainly during atrial contraction. In the first and third diastole tricuspid valve opening is recorded very late, only after atrial contraction. In such patients the opening of the tricuspid valve at atrial systole may

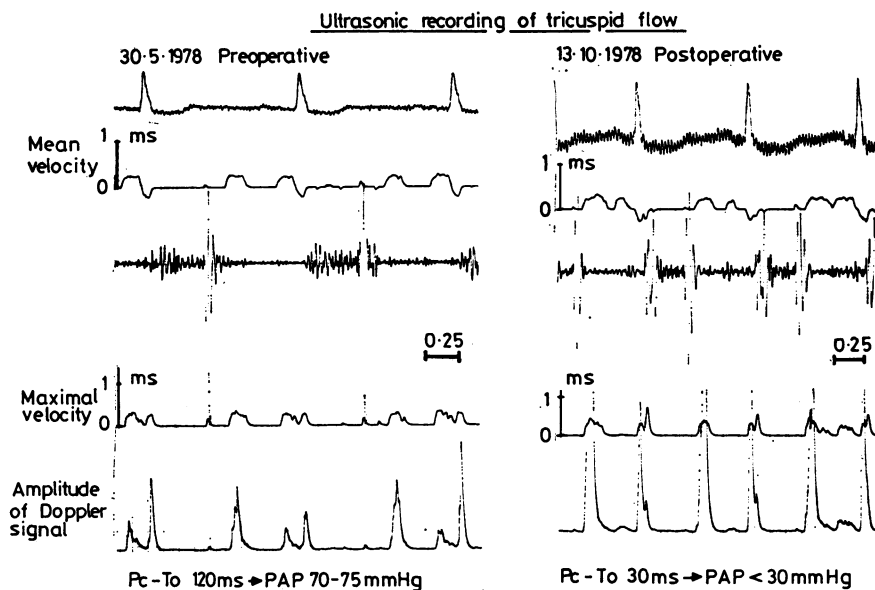


Fig. 10 Ultrasonic recording with estimation of pulmonary artery systolic pressure (PAP) before and three weeks after mitral valve replacement in a patient with mitral regurgitation.

be more easily detected than the initial opening earlier in diastole and this may then be overlooked. The early diastolic filling in this patient (Fig. 11) was so much less, and with less valve movement (2nd diastole) that it had to be specially looked for. In the first and third diastole the signals from the initial opening and early diastolic flow have been too weak to be recorded, whereas in the second beat it is recorded probably owing to increased flow during inspiration. Since it is the time of the initial opening that is needed and not of the reopening at atrial systole, one should be alerted to search for this when valve movement and diastolic filling at first is recorded only at atrial contraction. With a prolonged PQ interval and/or very rapid heart rate, however, this may occur simultaneously with atrial systole,

but in such cases tricuspid valve opening occurs early in diastole.

Discussion

Accurate recording of the P_c-T_0 interval is essential, as can be seen from the nomogram (Fig. 2). A difference in the interval of 10 ms will result in a 10 mmHg difference in estimated pressure. Ultrasound can record valve movements directly and is an easy and accurate way to obtain the interval. Localisation of pulmonary valve closure from the phonocardiogram alone was difficult in many adult patients as the initial deflection of P_2 was often hidden in the vibrations from A_2 .

Echocardiography can be used to measure the

Table 1 Non-invasive estimation of pulmonary artery systolic pressure (PAP) before and after operation

Case no.	Age	Diagnosis	Systolic PAP (mmHg)		Time after operation
			Preoperative	Postoperative	
1	4	ASD	65	<30 <30	10 d, 3 mth
2	58	MR	72	<30	3 wk
3	3	VSD	60	<30 <30	3 and 6 mth
4	52	MS, MR	86	<30	2 wk
5	53	MS, AR	75	<30	6 mth
6	64	MR	75	60 35	3 and 5 mth
7	1½	VSD	90	60 65	2 and 4 mth

ASD, atrial septal defect; MR, mitral regurgitation; MS, mitral stenosis; VSD, ventricular septal defect; AR, aortic regurgitation

interval, but with the Doppler method it is easier to localise and record pulmonary valve and possibly tricuspid valve movements. Both tricuspid and pulmonary flow and valve movements were recorded in all patients in this study. The method can also be applied in newborn and even premature infants. The smallest one studied so far weighed 1200 g. Only in small children with Fallot's tetralogy have pulmonary flow and pulmonary valve movements been difficult to record.

The results indicate that the method is useful for the non-invasive estimation of systolic pressure in the pulmonary artery. There were no false positives or false negatives, but only borderline pressure was estimated in one patient with significantly raised pressure. False negative results may occur in the presence of significant tricuspid regurgitation, but such patients were not included in this study. Extrapolation of the nomogram above the published heart rate seems reliable. The ability to record instant changes in pulmonary artery pressure was shown by simultaneous recording in two patients with increased respiratory variation in pressure. Repeatability was good and in 12 patients with mitral valve disease estimation of systolic pulmonary artery pressure both at rest and during exercise proved possible. Though the estimated pressure during exercise was not verified by simultaneous pressure recording it appears acceptable as there was a corresponding increase in the mitral valve gradient.

Correlation was uniformly good between directly measured and estimated pressures except in three patients. In two of these where the examinations were not performed on the same day conditions were such that the pulmonary artery pressure was probably higher at the time of ultrasound than at pressure recording. In the third patient (marked c in Fig. 5) estimated pressure was much lower than

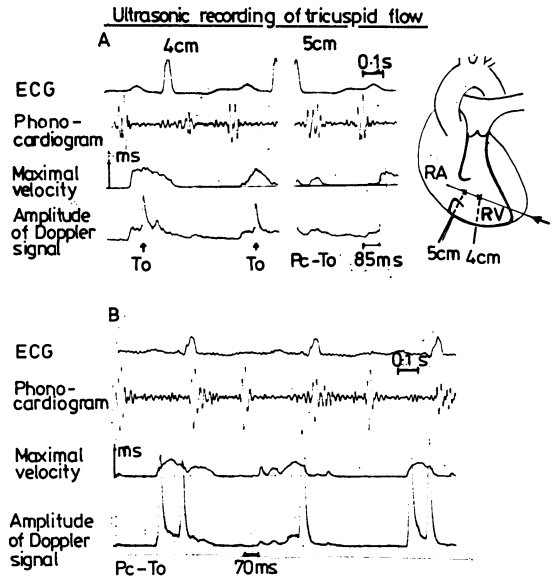


Fig. 11 Possible errors in recording tricuspid valve opening. (A) With pulmonary regurgitation, flow towards the transducer may be recorded early in diastole in the right ventricle (4 cm). The later start of tricuspid flow in this patient is seen by noting the later appearance of tricuspid valve opening (T_0) in the amplitude curve, as well as by recording closer to the tricuspid orifice (5 cm) where pulmonary regurgitation is less likely to be recorded. (B) In heart failure with very low cardiac output main opening of the tricuspid valve may not occur until atrial contraction (1st and 3rd diastole), so one may have to look for the much lesser early diastolic flow (2nd diastole).

Table 2 Non-invasive ultrasonic recording

Case No.	Age (y)	Mean pressure drop across mitral valve (mmHg)		Pulmonary artery systolic pressure (mmHg)	
		Rest	Exercise	Rest	Exercise
1	48	3	10	<30	<30
2	59	4	9	<30	<30
3	36	6	16	<30	<30
4	44	4	12	<30	30
5	51	4	22	<30	30
6	61	8	18	<30	35
7	67	4	13	<30	40
8	45	7	22	<30	40
9	61	8	16	30	45
10	57	10	14	30	45
11	42	7	20	30	45
12	53	13	34	55	77

that found at catheterisation. The reason for this is not clear. Pulmonary valve closure and tricuspid valve opening were clearly recorded, right atrial pressure was not raised, and tricuspid regurgitation was not present. She had, however, tight mitral stenosis, where the pressure drop across the valve and consequently pulmonary artery pressure may vary considerably with variations in cardiac output. The two measurements were unfortunately not done simultaneously, and some of the discrepancy may have been caused by differences in pressure on the two occasions. An earlier non-invasive estimation with the same method of pulmonary artery pressure closer to that obtained at catheterisation might support this (50/60 mmHg). This patient illustrates the necessity of simultaneous measurements when evaluating the method in patients where pressure is likely to vary considerably. It might be questioned if the method can be equally applied in all kinds of

heart disease. However, many differing heart disorders of varying severity were seen in this study, and good correlation was found between estimated and recorded pressure.

The method assumes that the rate of fall of right ventricular pressure is the same regardless of the height of the pressure. Curtiss *et al.*,⁶ however, have shown that the rate of rise of right ventricular pressure increases with an increase in systolic pressure. Our own observations indicate that rate of fall of right ventricular pressure during relaxation also increases with an increase in systolic pressure, and may be two to three times that found with normal pressures. This would decrease significantly the expected lengthening of the P_c-T_0 interval at higher pressures. At a systolic pressure of 60 mmHg the interval might decrease so much that the pressure could be underestimated by 15–20 mm Hg. Why, then, does this method work as well as it does? A possible explanation is the so-called "hang-out" interval described by Shaver *et al.*⁷ They showed that closure of the pulmonary valve was normally delayed from 30 to 80 ms after the end of right ventricular systole. The delay varied with diastolic pressure in the pulmonary artery and decreased when the pressure increased. This would lengthen the P_c-T_0 interval and might diminish or cancel the effect of increased rate of pressure fall.

A similar finding was noted by Cumming,⁸ who described an early P_2 in patients with persisting high pulmonary artery pressure, and a delayed P_2 in patients with low pulmonary artery pressure after banding of the pulmonary artery. Further investigation is necessary to study if and by how much the relaxation rate and the P_c-T_0 interval are influenced by the height of pressure, by changes in sympathetic tone, or by drugs.

The other requirement if this method is to be reliable is a normal right atrial pressure; if increased, the estimated pulmonary artery pressure may be too low. A raised right atrial pressure should be easily found by recording jugular venous pressure; tricuspid regurgitation will be noted by Doppler.

However, even in those patients estimation of a significantly raised pulmonary artery pressure is useful even if the degree of underestimation is not clear. It may be that the height of the jugular venous pressure or the degree of tricuspid regurgitation can be allowed for in the estimation but this needs further study.

The study was supported by the Norwegian Council on Cardiovascular Disease.

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