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Attempts at measuring pulmonary arterial pressure by means of Doppler echocardiography in patients with chronic lung disease

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Measurement of pulmonary arterial pressure by means of Doppler echocardiography in patients with chronic lung disease. A. Torbicki, K. Skwarski, I. Hawrylkiewicz, T. Pasierski, Z. Miskiewicz, J. Zielinski.

ABSTRACT: In 72 patients with severe chronic pulmonary or pulmonary vascular disease pulmonary arterial pressure was measured by means of right heart catheterization. Forty three patients had pulmonary hypertension, (32±11 mmHg) and 27 patients had normal pressure (14±3 mmHg). These patients were examined with continuous wave (CW) and pulsed wave (PW) Doppler echocardiography. The retrograde systolic tricuspid valve pressure gradient assessed with CW Doppler correlated with systolic pulmonary pressure (r=0.92, p<0.001, SEE 7.7 mmHg) but was measurable in only 17 of the 70 patients. The flow velocity pattern in the right ventricular outflow tract could be recorded in 68 of the 70 patients. Acceleration time (AcT) from systolic flow onset to peak velocity correlated with mean pulmonary artery pressure (r=-0.72, p<0.001, SEE 8.3 mmHg). An AcT <90 msec had an 84% positive predictive value for pulmonary hypertension. Right ventricular isovolumic relaxation time could be measured in 59 of the 70 patients and correlated with systolic pulmonary artery pressure (r=0.69, p<0.001, SEE 12.4 mmHg). No single Doppler method is at the same time easily applicable and accu5rate in prediction of pulmonary arterial pressure in patients with chron55–ic lung diseases.

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Pulmonary arterial pressure is one of the most important prognostic factors in chronic lung disease [1–5]. A noninvasive, easy to apply and reliable method of assessment of the pulmonary arterial pressure would make it possible to identify the high-risk subgroup and to follow the natural course and results of treatment of pulmonary hypertension in this population. So far, such a method has not been available [6].

Three Doppler echocardiographic methods for assessment of pulmonary arterial pressure were recently validated in cardiological patients [7]. However, patients with lung disease are more difficult to examine with ultrasound. This is due to hyperinflation of the lungs, tachypnoea, tachycardia and marked respiratory variations in the intrathoracic pressure. The present study was designed to establish, whether any of the available Doppler methods could be clinically useful to assess pulmonary arterial pressure in these patients.

Materials and methods

The study population consisted of 74 patients with chronic lung disease (CLD): 47 patients with chronic obstructive lung disease, 9 with pulmonary sarcoidosis, 8 with interstitial lung fibrosis and 10 with other forms of CLD (kyphoscoliosis, bronchiectasis, chronic recurrent pulmonary embolism, primary pulmonary hypertension). The clinical diagnosis of CLD was based on the usual clinical criteria. All patients were submitted to right heart catheterization and Doppler echocardiography in the steady state of their pulmonary disease without clinical signs of heart failure. Four patients were excluded from Doppler analysis because of complete bundle branch block or atrial fibrillation. Of the remaining 70 patients, forty three had pulmonary hypertension (PH) (mean pulmonary arterial pressure (MPAP) >20mmHg) and twenty seven patients had normal pulmonary arterial pressure (N) (table 1).

Right heart catheterization was performed with a Swan-Ganz catheter using the Seldinger technique. Standard pressure measurements were made with the use of a Siemens-Elema 746 pressure transducer coupled to a Siemens-Elema recorder (Mingograph 34) and were referred to zero level at 5 cm below the sternal angle. All measurements were performed at rest in the supine position and were averaged for at least three respiratory cycles.

The Doppler examination was carried out up to three days following the right heart catheterization. The

echocardiographic team was blind to the results of the invasive measurements. A commercially available ultrasound system was used for the Doppler examination (Hewlett Packard 77020A). Pulsed (PW) and continuous wave (CW) Doppler examinations were performed under two-dimensional echocardiographic control with a 2.5 MHz and 1.9 MHz imaging phased array transducer, respectively. All of the patients were additionally examined with a 1.9 MHz non-imaging "pencil" continuous wave transducer. Monitoring of the respiratory movements of the thorax was provided by a pulse sphygmographic transducer (HP 21050A) positioned under an elastic band wrapping the upper part of the thorax. The changes of thoracic dimensions were displayed as a curve simultaneously with Doppler and electrocardiographic (ECG) tracings. Doppler measurements of six end-expiratory evolutions were averaged for analysis.

Table 1. – Mean pulmonary arterial pressure, age and heart rate in patients with pulmonary hypertension and normal pulmonary arterial pressure

	PH MPAP ≥20 mmHg	N MPAP <20 mmHg	
n	43	27	
PAP	31.7±10.7	13.5±3.2	
Age	53.0±12.1	47.6±13.1	p=0.08
HR	88.6±17.6	81.1±13.4	p=0.06

PH: pulmonary hypertension; MPAP: mean pulmonary arterial pressure; N: normal pulmonary arterial pressure; PAP: pulmonary arterial pressure; HR: heart rate.

Three Doppler echocardiographic methods were checked for the correlations with directly measured pulmonary arterial pressure (figs 1 and 2):

1. *Measurement of the retrograde pressure gradients across right heart valves* [8, 9]. The imaging CW Doppler transducer was used to interrogate the tricuspid valve for the presence and maximal velocity of the regurgitant jet. Apical, subcostal and parasternal approaches were used. The procedure was repeated with the non-imaging CW Doppler probe in order to obtain a clear outline of the velocity spectrum of the jet. The maximal velocity thus obtained (fig. 2) was used in the simplified Bernoulli equation to calculate the retrograde systolic pressure gradient (PG) between right ventricle (RV) and right atrium (RA) as RV-RA PG = $4 \times Vmax^2$, according to the method described by Yock and Popp [9]. Recording of the velocity of the regurgitant jet across the pulmonary valve was also attempted during CW Doppler examination.

2. *Analysis of flow velocity in the right ventricular outflow tract* [10]. The sample volume of the PW Doppler was placed just below the level of the pulmonary valve in the middle of the right ventricular outflow tract (RVOT). The position of the sample volume was confirmed both with the two-dimensional image and with the typical appearance of the Doppler tracing, characterized by the lack of the opening signal from pulmonary valvular leaflets. The flow velocity pattern was then assessed as: dome-like, triangular or notched. Quantitative evaluation consisted of measurement of acceleration time (AcT) as the interval from the onset to the maximal velocity of foward flow, measurement of right ventricular pre-ejection period (RPEP), as the interval from the Q wave of the ECG to the beginning of the forward flow, and finally, measurement of the ejection time (RVET) as the interval between onset and termination of flow in the RVOT (fig. 2).

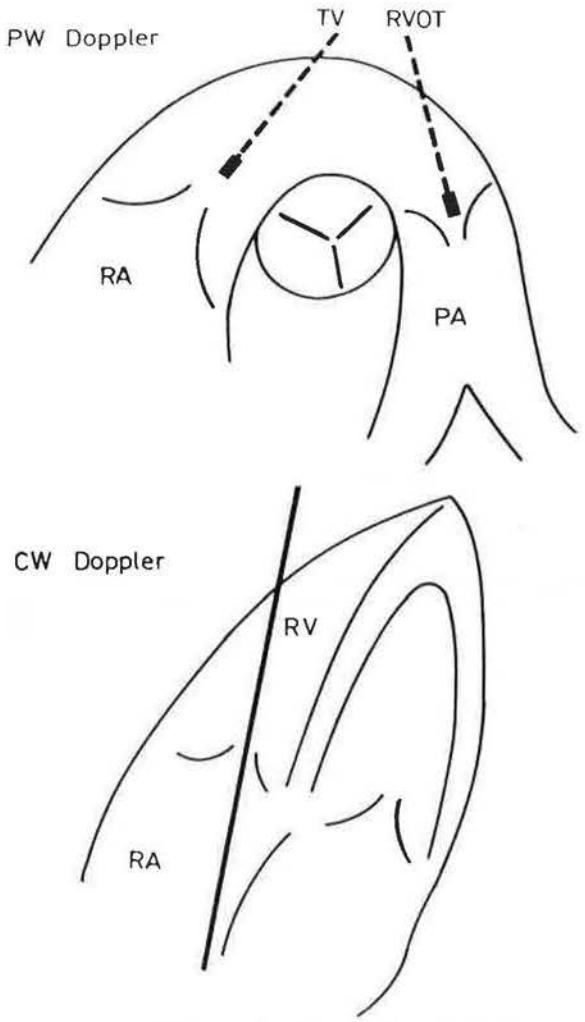

Fig. 1. – Position of the sample volume of the pulsed wave (PW) Doppler and the reference line of the continuous wave (CW) Doppler during acquisition of the Doppler data. RA: right atrium; PA: main pulmonary artery; RV: right ventricle; TV: position of the PW Doppler sample volume for the recording of the tricuspid valve events; RVOT: position for the recording of the right ventricular outflow tract events.

3. *Measurement of right ventricular isovolumic relaxation time (RVIRT)* [7, 11]. This measurement requires identification of both the closing moment of the pulmonary valve and the opening moment of the tricuspid valve (fig. 2). As simultaneous Doppler tracings from pulmonary and tricuspid valves were impossible to obtain, RVIRT was calculated indirectly from non-simultaneous Doppler tracings using the Q wave of the ECG (Q) as the

reference. Thus RVIRT=(Q-TVo)-(Q-PVc), where: Q-TVo was the interval from Q to tricuspid valve opening identified on the PW Doppler tracing of the tricuspid valve; Q-PVc was the interval from Q to pulmonary valve closure identified on the PW Doppler tracing recorded in the RVOT, and the latter was subtracted from the former. If the difference in heart cycle length between tricuspid and pulmonary valve tracings exceeded 10%, RVIRT was not calculated. Special care was taken to standardize measurements to the end-expiratory phase as respiration seemed to have marked influence on the RVIRT. Burstin's nomogram was used for the heart rate correction [11].

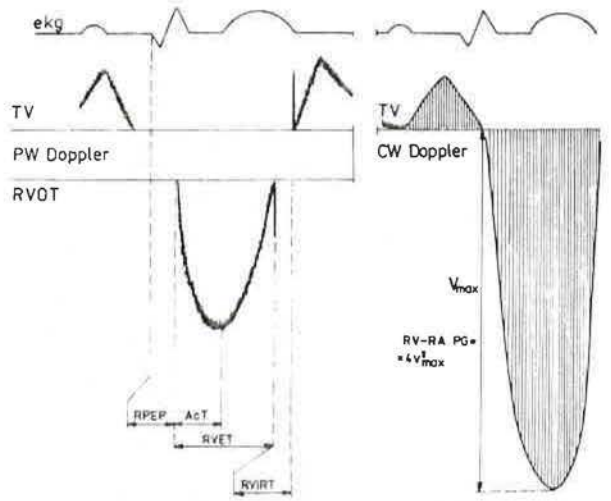

Fig. 2. – Schematic representation of the Doppler measurements used in the study. Left panel: pulsed wave (PW) Doppler examination of the tricuspid valve and right ventricular outflow tract. Right panel: measurement of tricuspid regurgitant jet velocity and calculation of the systolic gradient between right ventricle and right atrium (RV-RA PG). See text for other abbreviations.

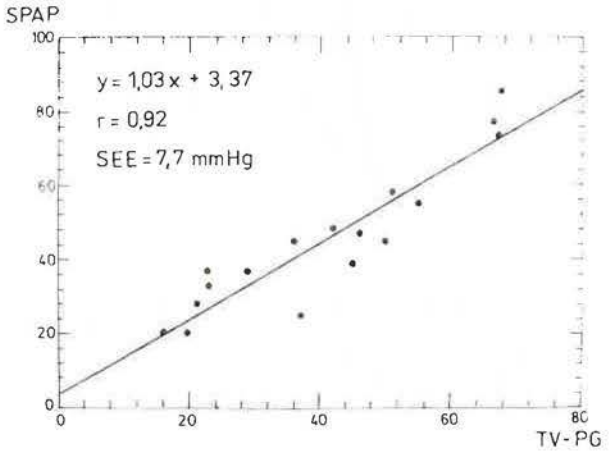

Fig. 3. – Regression of systolic pulmonary arterial pressure (SPAP) on the tricuspid retrograde pressure gradient (TV-PG).

Statistics

All values were expressed as mean±SD. Linear, exponential and multiple regression analyses were used to check the correlations between the Doppler indices and the direct pressure measurements. T-distribution values and analysis of variance were used to check the statistical significance of the difference in mean values and of the correlations. Sensitivity was calculated as TP/(TP+FN), specificity as TN/(TN+FP) and the positive diagnostic value as TP/(TP+FP), where TP represents true positive, TN true negative, FP false positive, andFN false negative.

Results

Tricuspid regurgitation was found with pulsed wave and/or continuous wave Doppler in 32 patients. However, in only 17 of the 70 patients was it possible to measure reliably the maximal velocity of the regurgitant jet. In this subgroup the calculated retrograde systolic pressure gradient between the right ventricle and the right atrium was found to correlate highly with the systolic pulmonary arterial pressure (SPAP) (table 2 and fig. 3). Pulmonary value regurgitation was found in 15 patients, but in only 7 patients could the jet velocity be clearly recorded and measured.

Table 2. – Correlations of Doppler derived measurements with pulmonary arterial systolic (SPAP) and mean pressures (MPAP)

	Feasibility %	r *vs* SPAP (SEE mmHg)	r *vs* MPAP (SEE mmHg)
TV-PG	24	0.92 (7.7)	-
PV-PG	10	-	-
AcT	97	-	-0.68 (9.2)
AcT*	97	-	-0.72 (8.3)
AcT/RR	97	-	-0.65 (9.3)
AcT/RVET	97	-	-0.69 (9.0)
PEP/AcT	96	-	0.69 (9.0)
RVIRT (B)	84	0.69 (12.4)	0.70 (8.8)

TV-PG: retrograde tricuspid valve systolic pressure gradient; PV-PG: retrograde pulmonary valve diastolic pressure gradient; AcT*: multiplicative correlation of AcT and MPAP (fig. 6); AcT: acceleration time; AcT/RR: acceleration time corrected for heart rate with Bazett formula; AcT/RVET: acceleration time corrected for right ventricular ejection time; PEP/AcT: acceleration time corrected for pre-ejection period; RVIRT (B): right ventricular isovolumetric relaxation time corrected for heart rate with Burstin's nomogram; SEE: standard error of the estimate; r: correlation coefficient. All of the correlations are significant at the level of $p<0.001$.

Flow velocity pattern in the right ventricular outflow tract could be recorded in all but two obese patients (97%) – in 23 patients from parasternal or low parasternal approach, in 34 patients from subcostal approach and in the remaining 13 patients both from subcostal and parasternal approach. Acceleration time correlated with MPAP. Correction for heart rate, ejection time (AcT/RVET) or pre-ejection period (PEP/AcT) did not improve this correlation, whereas a multiplicative model improved it slightly (table 2 and fig. 4). Standard error of the estimate (SEE) was above 8 mmHg for all

calculated regression equations. The sensitivity and specificity for the diagnosis of pulmonary hypertension calculated for the two arbitrarily chosen cut-off values are given below (table 3). The notched pattern of the flow velocity curve in the right ventricular outflow tract was a specific but not a sensitive sign of pulmonary hypertension.

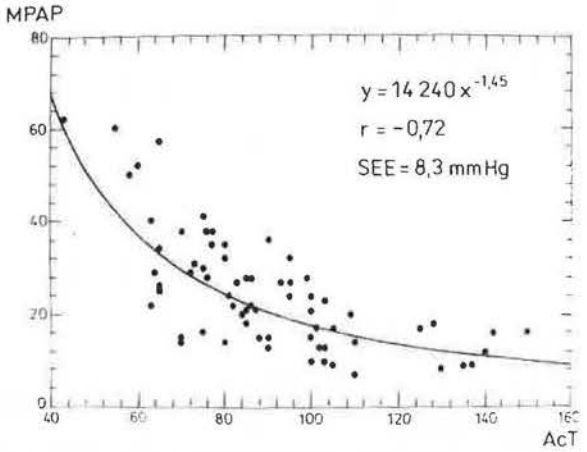

Fig. 4. – Regression of mean pulmonary arterial pressure (MPAP) on acceleration time (AcT) measured from the right ventricular outflow tract. Multiplicative model of regression: $y=ax^b$.

Table 3. – Sensitivity, specificity and positive predictive value (+) for the detection of pulmonary hypertension in patients with chronic lung disease

	Sensitivity %	Specificity %	(+) %
PEP/AcT >1.0	93	69	83
AcT <90 msec	79	78	84

PEP/AcT: acceleration time corrected for pre-ejection period; AcT: acceleration time.

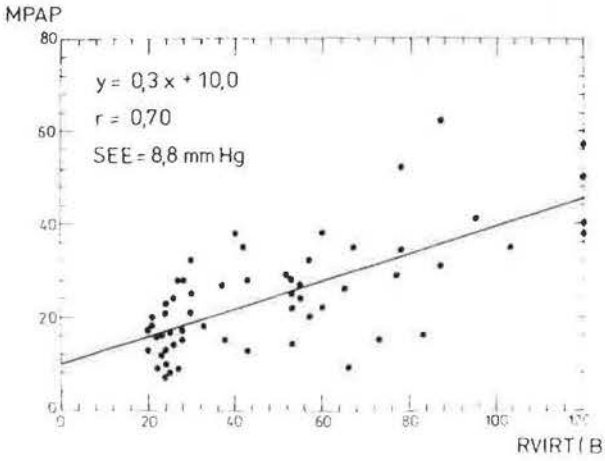

Fig. 5. – Regression of directly measured mean pulmonary arterial pressure (MPAP) on the noninvasive estimate derived from right ventricular isovolumetric relaxation time (RVIRT) corrected for heart rate according to Burstin's nomogram.

Right isovolumic relaxation time could be measured in 59 patients (84%). When corrected for heart rate with Burstin's nomogram it correlated with SPAP with an SEE of 12.4 mmHg and with MPAP with an SEE of 8.8 mmHg (fig. 5).

Discussion

Despite significant correlations between Doppler derived measurements and pulmonary arterial pressure none of the three Doppler methods studied satisfied the need for a clinically useful noninvasive predictor of pulmonary hypertension in patients with chronic lung disease [12]. We confirmed that acceleration time in the right ventricular outflow tract can be easily measured with PW Doppler despite lung hyperinflation [13–15]. In our study, however, this measurement failed to provide a precise estimate of the mean pulmonary arterial pressure. Predictions of pulmonary pressure based on right ventricular isovolumetric relaxation time were of similar accuracy, but the assessment of RVIRT itself was cumbersome.

Doppler and direct haemodynamic measurements were not performed simultaneously. However, spontaneous variations of pulmonary arterial pressure do not seem to explain the relatively wide confidence limits of the pressure predictions based on AcT and RVIRT measurements in view of the excellent correlation found concurrently between CW Doppler measurements of tricuspid jet velocity and systolic pulmonary pressure. Unfortunately, this method of right heart pressure evaluation was applicable only to the minority of the patients studied.

The low success rate of measurements of the peak velocity of tricuspid regurgitant jets in our study was partly due to strict quality criteria for CW Doppler tracings used for pressure estimation. LAABAN *et al.* [16], who reported a higher success rate (65%), found the correlation with direct pressure measurements much less relevant ($r=0.65$, $p<0.01$).

Despite these limitations, Doppler methods seem to offer more information regarding the level of pulmonary arterial pressure than other noninvasive methods used so far for this purpose in patients with chronic lung diseases [6]. Furthermore, enhancement of the CW Doppler signal with peripheral saline injections may increase the feasibility of jet velocity measurements, which are apparently the most straightfoward and accurate of available Doppler methods for intracardiac pressure predictions [17]. Also, relatively low accuracy of pressure predications based on AcT and RVIRT measurements do not necessarily imply that selected Doppler indices will not prove useful in following acute or chronic changes in pulmonary haemodynamics in an individual patient. These problems should be addressed in future studies.

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Tentative de mesure de la pression artérielle pulmonaire par échocardiographie Doppler chez les patients atteints de maladie pulmonaire chronique. A. Torbicki, K. Skwarski, I. Hawrylkiewicz, T. Pasierski, Z. Miskiewicz, J. Zielinski.

RÉSUMÉ: La pression artérielle pulmonaire a été mesurée par cathétérisme du coeur droit chez 72 patients atteints de maladie pulmonaire chronique sévère ou de maladie vasculaire pulmoniare. Quarante-trois patients avaient une hypertension pulmonaire (32±11 mmHg) et 27 une pression normale (14±3 mmHg). Ces patients ont été examinés par échocardiographie Doppler en mode continu (CW) et pulsé (PW). Le gradient de pression systolique (VD-OD) au travers de la valve tricuspide, apprécié par CW Doppler, est en corrélation avec la pression pulmonaire systolique ($r=0.92$, $p<0.001$, SEE 7.7 mmHg), mais ne peut être mesuré que chez 17 des 70 patients. La courbe de vélocité de la chambre de chasse du ventricule droit a pu être enregistrée chez 68 des 70 patients. L'intervalle de temps entre le débit et le pic de la vélocité du flux (AcT) est en corrélation avec la pression artérielle pulmonaire moyenne ($r=-0.72$, $p<0.001$, SEE 8.3 mmHg). Un AcT <90 msec a une valeur prédictive de 84% pour l'hypertension pulmonaire. Le temps de relaxation isovolumique de ventricule droit a pu être mesuré chez 59 des 70 patients et est en corrélation avec la pression artérielle pulmonaire systolique ($r=0.69$, $p<0.001$, SEE 12.4 mm Hg). Aucune méthode Doppler isolée n'est à la fois aisément applicable et précise dans la prédiction de la pression artérielle pulmonaire chez les patients atteints de maladie pulmonaire chronique.

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