ASSESSMENT OF PULMONARY ARTERY PRESSURE BY PULSED DOPPLER ECHOCARDIOGRAPHY IN PATIENTS WITH CHRONIC PULMONARY DISEASES

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Doppler echocardiography was recently developed for obtaining a flow velocity profile at any point in the cardiac chamber. A pulsed Doppler technique combined with cross sectional echocardiography was used to examine the flow velocity pattern in the right ventricular outflow tract in 32 patients with chronic pulmonary disease (CPD) and in 15 healthy subjects as controls. All patients underwent cardiac catheterization. Pulmonary flow velocity profiles in the right ventricular outflow tract were recorded simultaneously with electrocardiograms and pre-ejection periods (PEP), right ventricular ejection time (RVET), acceleration time (AT), and calculated PEP/RVET, AT/RVET were determined. All normal subjects had "dome-like" flow velocity patterns with a peak flow at the midsystole. In patients with CPD, there was a significant correlation between $\log_{10}$ MPAP (mean pulmonary arterial pressure) and AT/RVET, the coefficient being $r = -0.623$, between PVR (pulmonary vascular resistance) and AT/RVET ($r = -0.52$), and between SVI (stroke volume index) and PEP ($r = -0.645$).

Complications of pulmonary hypertension in CPD were relatively mild, but the pulsed Doppler technique was found useful for evaluating pulmonary arterial pressure and other right heart hemodynamics of CPD patients.

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IN patients with chronic pulmonary diseases (CPD), structural remodeling of the pulmonary arteries and hypoxic pulmonary vasoconstriction contribute to pulmonary hypertension. Pulmonary hypertension is a major factor affecting the prognosis of CPD patients. To assess pulmonary hypertension, chest X-rays¹ and electrocardiograms were used. Although the accurate evaluation of right heart hemodynamics requires radionuclide techniques and cardiac catheterization, these are expensive and invasive. Echocar-

diography is a noninvasive technique to assess pulmonary hypertension by tracing abnormal motion of the pulmonary valve^{2–4}. Doppler echocardiography was recently developed to obtain flow velocity profiles at any point in the cardiac chamber. This technique has often been used to determine pulmonary arterial pressure^{5–10}. In most published papers, the subjects had high pulmonary arterial pressure, owing to congenital heart disease, valvular disease or primary pulmonary hypertension. Pulmonary hypertension in CPD patients is relatively mild compared with these diseases.

An attempt was made to predict pulmonary arterial pressure and right heart

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TABLE I DATA ON RESPIRATORY FUNCTION OF PATIENTS*

Data	Control	CPD Patients
No. of patients	15	32
%VC	106.9±18.6	71.1±21.8
FEV _{1.0} %	75.2±12.5	58.6±18.6
PaO ₂ , mmHg	83.6±12.5	73.8±15.4
PaCO ₂ , mmHg	35.3±5.9	40.1±5.1

* Values are means±SD.

hemodynamics of CPD patients by pulsed Doppler echocardiography.

MATERIALS AND METHODS

Patient selection

The control group consisted of 15 healthy subjects (9 men and 6 women). The mean age was 56±11 (range, 27 to 69 years). All were suspected to have ischemic heart disease, but none showed abnormal findings on cardiac catheterization.

The study group consisted of 32 patients (19 men and 13 women), with a mean age of 56±15 (range 16 to 76). Twenty patients had pulmonary emphysema, 8 had chronic bronchitis, 3 had diffuse panbronchiolitis and 1 had bronchiectasis.

They were diagnosed as having CPD by history, physical examination, standard pulmonary function tests (Table I), blood gases, bronchography and selective alveolobronchography. Patients with pulmonary stenosis, tricuspid insufficiency, other valvular diseases, heart failure or ischemic heart disease were excluded from the study. The existence of significant tricuspid insufficiency was at opponent on color Doppler echocardiography.

Cardiac catheterization

A Swan-Ganz catheter (Edwards laboratories) was inserted into the femoral vein by the method of Seldinger. Cardiac output was measured by thermodilution using a Gould cardiac index computer P1435. The stroke volume index (SVI) and pulmonary vascular resistance (PVR) were calculated as follows:

$$\text{SVI} = \text{CO} / \text{HR} / \text{BSA}$$

$$\text{PVR} = [(\text{MPAP} - \text{MPAWP}) / \text{CO}] \times 1332 \text{ dyne}$$

·sec·cm⁻⁵

CO: cardiac output

HR: heart rate

BSA: body surface area

MPAP: mean pulmonary arterial pressure

MPAWP: mean pulmonary arterial wedge pressure

Pulsed Doppler echocardiography

Aloka SSD-860 pulsed Doppler echocardiography units with a 3.5-MHz transducer were used. Pulmonary flow velocity profiles were recorded in the right ventricular out-flow tract under the guidance of color Doppler echo images. Doppler signals were recorded simultaneously with an electrocardiogram on a strip chart recorder at a paper speed of 100 mm/sec. The studies were performed within 24 h of cardiac catheterization. All patients were examined in the left lateral decubitus position and tracings were recorded in the end-expiratory breath-holding state. Flow velocity profiles were obtained for all patients. The clinical conditions were stable and essentially the same for the Doppler examination and for cardiac catheterization.

Analysis of Doppler flow velocity profiles (Fig. 1)

Acceleration time (AT) was measured from the onset of ejection (F₀) to peak velocity (fm); right ventricular ejection time (RVET), from the onset to the end of flow (fe); and pre-ejection period (PEP), from Q-wave of ECG to F₀. All these parameters were averaged over 5 consecutive cardiac cycles and corrected by R-R intervals.

RESULTS

1) Comparison of right heart hemodynamics between the control and study groups (Fig. 2).

The mean values of MPAP, HR and PVR differed significantly between the 2 groups. The respective values for the 2 groups were: MPAP: 11±3 and 18±8; HR: 66±8 and 76±13; PVR: 73±40 and 180±129. There was no significant difference, however, in SVI. The values were 52±8 and 49±12.

2) The relation between Doppler indices and right heart hemodynamics in the study group.

Fig.1. Flow velocity profiles in right ventricular outflow tract

Fig.2. Right heart hemodynamics in 2 group.

CONT: Normal subject, CPD: Chronic pulmonary disease, MPAP: indicates mean pulmonary arterial pressure, HR: heart rate, S.V.I.: stroke volume index, PVR: pulmonary vascular resistance.

There was a significant correlation between AT/RVET and MPAP, the correlation coefficient being $r = -0.623$. An improved correlation was not found between AT/RVET and natural log of MPAP, the coefficient being $r = -0.628$ (Fig. 3 and Fig. 4). There was a significant correlation be-

tween PVR and AT/RVET, the coefficient being $r = -0.502$ (Fig. 5).

SVI and Doppler indices were compared. SVI was negatively correlated with PEP ($r = -0.645$) and PEP/RVET ($r = -0.610$) (Fig. 6). Natural log of SVI did not significantly change the correlation ($r = -0.626$

Fig.3. Left: acceleration time (AT) vs mean pulmonary arterial pressure (MPAP).
Right: AT, right ventricular ejection time (RVET) ratio vs MPAP.

Fig.4. Left: acceleration time (AT) vs $\log_{10}$ (mean pulmonary arterial pressure) (log MPAP).
Right: AT, right ventricular ejection time (RVET) ratio vs log MPAP.

Fig.5. Left: acceleration time (AT) vs pulmonary vascular resistance (PVR).
Right: At, right ventricular ejection time (RVET) ratio vs PVR.

Fig. 6. Left: pre-ejection period (PEP) vs stroke volume index (S.V.I.).
Right: PEP, right ventricular ejection time (RVET) ratio vs S.V.I.

Fig. 7. Left: pre-ejection period (PEP) vs log₁₀ (stroke volume index) (log S.V.I.).

for PEP, and $r = -0.599$ for PEP/RVET) (Fig. 7).

DISCUSSION

Disturbance of the pulmonary circulation in CPD adversely affects morbidity and mortality. Therefore, determination of the presence and severity of pulmonary hypertension and other abnormalities in hemodynamics is very important. Factors that influence pulmonary arterial pressure include cardiac output, pulmonary vascular resistance and pulmonary venous pressure. Until now, catheterization has been conducted to obtain these data. In recent years, however, great advances have been made in ultrasound diagnostic techniques, especially Doppler echocardiography, mak-

ing possible the non-invasive evaluation of cardiac diseases.

There have been many reports of the use of pulsed Doppler echocardiography⁵⁻¹⁰ to determine pulmonary arterial pressure. Most subjects examined have had high pulmonary arterial pressure ($10 \leq \text{MPAP} \leq 50 \sim 90 \text{ mmHg}$)⁵⁻⁹ due to congenital heart disease, valvular disease and primary pulmonary hypertension.

Pulmonary hypertension in CPD patients is relatively mild, but it may severely affect the prognosis in CPD. The pulsed Doppler technique was useful for evaluating relatively mild pulmonary hypertension and other right heart hemodynamics.

In this study, it was difficult to obtain a clear view of 2-dimensional echocardiography because of the increased pulmonary

residual volume in CPD patients. All patients were examined in the left lateral decubitus position so that a clear view could be obtained. A flow velocity profile was also obtained. The most important factors affecting this profile were heart rate and stroke volume. To minimize their influence, we recorded at least 5 consecutive beats when patients were in the end expiratory breath-holding state. Each index was corrected by R-R intervals and the data were averaged. Cardiac catheterization and Doppler examinations were performed within 24 h. The 2 procedures were performed under similar conditions.

Patients with pulmonary hypertension had definite flow velocity patterns, that is, fast acceleration and slow deceleration. To measure acceleration time (AT), MPAP was determined noninvasively by the pulsed Doppler technique. Although complications of pulmonary hypertension in CPD are relatively mild, this technique was able to successfully determine MPAP values.

Published correlation coefficients are $r = -0.90$ ($\log_{10}$ MPAP vs AT/RVET) as reported by Kitabatake et al⁵ and $r = -0.87$ (MPAP vs AT) by Debestani et al⁹. As compared with the published figures, the correlation coefficient found in this study was not so good. Two reasons for this are: 1) MPAP in CPD patients is relatively mild, 2) CPD patients may have dyspnea. Apnea affects the pulmonary arterial pressure and flow velocity patterns. Systolic pulmonary arterial pressure can be estimated from the peak velocity of the tricuspid regurgitant jet, and diastolic pulmonary arterial pressure from the pulmonary regurgitant jet¹¹. However, determination of the correct peak velocity requires angle correction, and in a relatively low pressure range, the regurgitant jet would be too small to evaluate. We think that our method is better for evaluation of pulmonary arterial pressure, despite the lower correlation coefficient.

Reflected pulse waves reaching the pulmonary valve early when the pulmonary artery is sclerosed might explain changes in flow velocity pattern. But other factors might influence pulmonary arterial pressure, e. g. remodeling of the pulmonary vascular tree and hypoxic pulmonary vasoconstriction. Further study is needed to determine

exactly which factors affect flow velocity patterns.

Methods for measuring SVI, such as the thermodilution technique and cardiac scintigraphy are available, but they are invasive and expensive. The pulsed Doppler technique is thus a very useful method for the easy and noninvasive evaluation of SVI.

PEP or PEP/AT has been reported to show a close correlation to MPAP.^{3,6,7} As for left heart hemodynamics as evaluated by mechanocardiogram, 3 factors prolong PEP: 1) decrease in contractility, 2) aberrant conduction of ventricle and 3) decrease in the preload.¹²⁻¹⁶ This study showed PEP to be closely related to SVI, which was affected by both contractility and preload. The results of this study supported left heart hemodynamics.

In conclusion, pulsed Doppler echocardiography is a valuable method for the noninvasive evaluation of right ventricular after load and SVI in patients with chronic pulmonary disease.

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