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The Pulmonary Component of the Second Sound in Right Ventricular Failure

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Sound within the pulmonary artery was measured in 24 patients to determine if right ventricular failure modifies the amplitude of the pulmonary component of the second sound (P2). The amplitude of P2 in eight patients with right ventricular failure secondary to pulmonary hypertension (2610 ± 370 dynes/cm²) did not differ from P2 in eight patients with pulmonary hypertension not accompanied by right ventricular failure (3120 ± 710 dynes/cm²). In both groups, the amplitude of P2 exceeded control subjects (520 ± 70 dynes/cm²) (P < .001 and P < .01, respectively). The maximal rate of development of the pressure gradient across the closed pulmonary valve was higher in patients with right ventricular failure (580 ± 100 mm Hg/sec) than in control subjects (150 ± 30 mm Hg/sec) (P < .001) and maximal negative dp/dt was also higher in patients with failure (750 ± 70 mm Hg/sec vs 190 ± 20 mm Hg/sec) (P < .001). The maximal rate of change of the diastolic pressure gradient correlated linearly with maximal negative dp/dt (r = .89). These observations indicate that P2 is accentuated in patients with right ventricular failure secondary to pulmonary hypertension. The accentuation results from the augmented rate of development of the diastolic pressure gradient, which reflects an augmented right ventricular negative dp/dt. Therefore, an accentuated P2 remains valid as a clinical sign of pulmonary hypertension whether or not right ventricular failure occurs.

Even though it is standard clinical practice to evaluate the intensity of the pulmonary component of the second sound (P2) during physical examination, only recently have the factors that affect its intensity been identified (1). The primary hemodynamic factor affecting intensity is the rate at which a pressure gradient develops across the valve after closure (1-4). The latter is strongly dependent upon the rate of fall of ventricular pressure (negative dp/dt) (4).

In patients with myocardial infarction or left ventricular failure, the intensity of the aortic component of the second sound is reduced (5-7). We have shown that in patients with poor left ventricular performance this reduction results from a diminished rate of change of the diastolic pressure gradient across the aortic valve which is secondary to impaired isovolumic relaxation (4). It is therefore useful to inquire how the intensity of P2 is affected by right ventricular failure in pulmonary hypertensive heart disease.

The purpose of this investigation is to determine if right ventricular failure in pulmonary hypertensive heart disease modifies the intensity of P2. We will explore the possibility of whether auscultation can give a clue to pulmonary hypertension once right ventricular failure develops, or whether the interpretation of the intensity of P2 should be modified in the presence of right ventricular failure.

Methods

Sound within the pulmonary artery was measured during diagnostic cardiac catheterization in 24 patients. Eight had normal pulmonary arterial pressure, eight had pulmonary hypertension without right ventricular failure, and eight had pulmonary hypertension with right ventricular failure.

Patients were categorized as having a normal pulmonary artery pressure if it did not exceed 30/12 mm Hg (8). Since right ventricular dysfunction has been observed in patients with right coronary disease (9, 10), they were excluded from the control group if narrowing of the right coronary artery was present.

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Right ventricular failure was defined as the presence of hepatomegaly, ascites, or dependent edema in patients with an elevated (> 9 mm Hg) right atrial pressure (8). The diagnosis of right ventricular failure, therefore, was essentially a clinical diagnosis (11-13), although measured pressures were substituted for the physical sign of systemic venous hypertension.

Patients were excluded if they had chronic obstructive pulmonary disease, since "emphysema heart" may differ from pulmonary hypertensive heart disease (14). The hemoglobin of control subjects, patients with pulmonary hypertension, and patients with pulmonary hypertension and right ventricular failure was 14.5 ± .8 gm/100 ml, 13.1 ± .7 gm/100 ml, and 13.1 ± .7 gm/100 ml, respectively.

The cardiac diagnoses of the patients in this study are shown in Table I. Patients with pulmonary hypertension (51 ± 4 years), and those with pulmonary hypertension and right ventricular failure, (57 ± 3 years), were older than the control subjects (41 ± 4 years). None of the control subjects were treated with digitalis or propranolol. Six of the patients with pulmonary hypertension who were not in right ventricular failure were treated with digitalis, and all with right ventricular failure were treated with digitalis.

None of the patients had electrocardiographic evidence of right ventricular hypertrophy. P pulmonale was present in one patient with pulmonary hypertension who was not in right ventricular failure. Complete right bundle branch block was present in one patient with right ventricular failure, and right axis deviation was present in one patient with right ventricular failure. Our criteria for making electrocardiographic interpretations have been previously described (15).

Intracardiac sound and pressure were measured with a Millar Instruments catheter-tip micromanometer and recorded on an Electronics for Medicine VR-6 photographic

TABLE I

Cardiac Diagnoses

Control Subjects		
No heart disease		6
Coronary heart disease (left anterior descending only)		1
Aortic insufficiency, mild		1
	Total	8
Pulmonary Hypertension		
Coronary heart disease		4
Mitral stenosis and/or regurgitation		2
Cardiomyopathy		1
Cause undetermined		1
	Total	8
Pulmonary Hypertension with Right Ventricular Failure		
Coronary heart disease		5
Aortic stenosis		1
Cardiomyopathy		1
Mitral stenosis and regurgitation		1
	Total	8

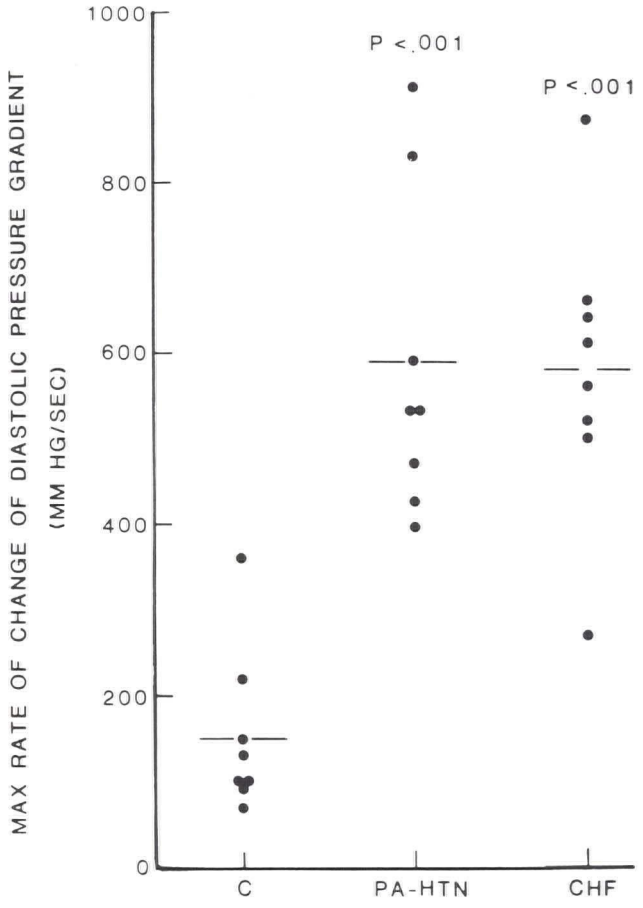

Fig. 1

Pulmonary component of the second sound in control subjects (C), patients with pulmonary hypertension (PA-HTN), and patients with pulmonary hypertension and right ventricular failure (CHF). Probabilities refer to comparisons of patients with PA-HTN to control subjects or comparisons of patients with CHF to control subjects.

TABLE II

Central Hemodynamic Measurements

		Control Subjects	PA Hypertension (No CHF)	PA Hypertension (CHF)
PA Pressure (mm Hg)	mean ± SEM	18/7 ± 1/1	64/31 ± 4/2	71/37 ± 5/2
RV End-Diastolic Pressure (mm Hg)	mean ± SEM	2 ± 0.3	7 ± 2	13 ± 2
	P		<.01	<.001
RA Mean Pressure (mm Hg)	mean ± SEM	4 ± 1	4 ± 1	15 ± 2
Heart Rate (beats/min)	mean ± SEM	83 ± 6	90 ± 5	93 ± 3
	P		NS	NS
Cardiac Index (1/min/m^2)	mean ± SEM	2.9 ± .2	2.1 ± .2	1.5 ± .1
	P		<.05	<.001
Stroke Index (ml/stroke/m^2)	mean ± SEM	38 ± 3	23 ± 3	18 ± 1
	P		<.01	<.001
Pulmonary Vascular Resistance (dyne-sec-cm^{-5})	mean ± SEM	53 ± 7	430 ± 80	490 ± 50
	P		<.01	<.001
RV Work (kg-m/min/m^2)	mean ± SEM	.40 ± .07	1.11 ± .20	.91 ± .05
	P		<.02	<.001

CHF: Congestive heart failure (right sided)
PA: Pulmonary artery
RV: Right ventricle

Probabilities refer to comparisons with control subjects.

recorder. Intracardiac sound pressure, referred to as intracardiac sound, was calibrated in dynes /cm^2. The frequency response of the system and the method of calibration of intracardiac sound have been previously described (16).

The rate at which a difference of pressure developed between the right ventricle and pulmonary artery during diastole was termed the rate of change of the diastolic pressure gradient. The rate of development of this pressure gradient across the closed pulmonary valve was calculated with the aid of a computer, as described previously (4).

Results

The amplitude of P2 in control subjects was 520 ± 70 dynes/cm^2. It was higher both in patients with pulmonary hypertension (3120 ± 710 dynes/cm^2) ($P < .01$) (unpaired t-test) and in patients with pulmonary hypertension and right ventricular failure (2610 ± 370 dynes/cm^2) ($P < .001$) (Fig. 1). The amplitude of P2 did not differ significantly between those with pulmonary hypertension who were in right ventricular failure and those who were not. Central hemodynamics are shown in Table II.

Instantaneous values of the pressure gradient that developed across the pulmonary valve after closure and throughout the duration of P2 are shown in a control patient, a patient with pulmonary hypertension, and a patient in right ventricular failure (Fig. 2). The rate of change of the diastolic pressure gradient can be judged from the slopes of these curves. The maximal rate of change of the diastolic pressure gradient across the closed pulmonary valve in control subjects was 150 ± 30 mm Hg/sec. It was higher both in patients with pulmonary hypertension (590 ± 70 mm Hg/sec) ($P < .001$) and in patients with pulmonary hypertension and right ventricular

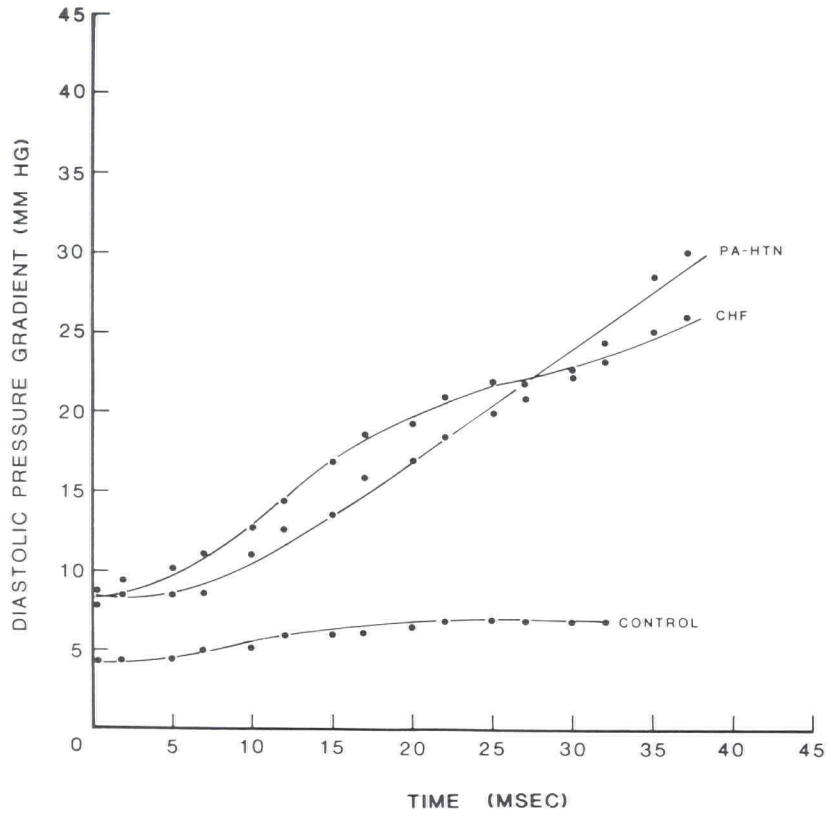

Fig. 2
Instantaneous values of the diastolic pressure gradient across the closed pulmonary valve from the beginning (time 0) to the end of P2 in a control subject, a patient with pulmonary hypertension (PA-HTN), and a patient with right ventricular failure (CHF). The maximal rate of change of the diastolic pressure gradient is the steepest slope of the respective curves.

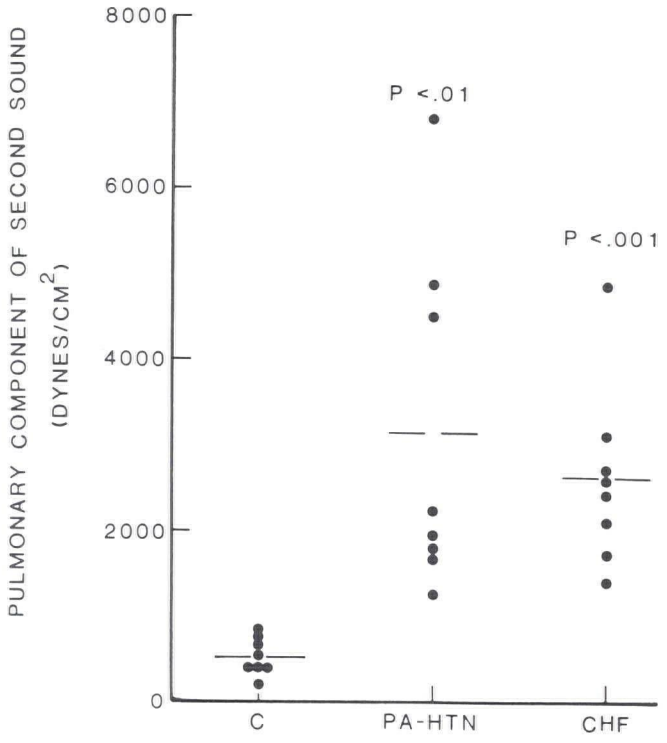

Fig. 3
The maximal rate of change of the diastolic pressure gradient across the pulmonary valve in control subjects (C), patients with pulmonary hypertension (PA-HTN), and patients with right ventricular failure (CHF). Probabilities refer to comparisons with control subjects.

failure (580 ± 100 mm Hg/sec) ($P < .001$) (Fig. 3). There was no significant difference between patients with pulmonary hypertension who were in right ventricular failure and those who were not in right vertricular failure.

The maximal rate of fall of right ventricular pressure (max neg dp/dt) in control subjects was 190 ± 20 mm Hg/sec. It was higher both in patients with pulmonary hypertension (790 ± 80 mm Hg/sec) ($P < .001$) and in patients with right ventricular failure (750 ± 70 mm Hg/sec) ($P < .001$). Max neg dp/dt showed no significant difference between patients with right ventricular failure and those not in right ventricular failure.

The amplitude of P2 correlated linearly with the maximal rate of change of the diastolic pressure gradient that developed across the closed pulmonary valve ($r = .70$). Eliminating patients with right ventricular failure did not significantly improve the correlation ($r = .71$). The maximal rate of change of the diastolic pressure gradient correlated linearly with maximal negative dp/dt ($r = .89$) (Fig. 4).

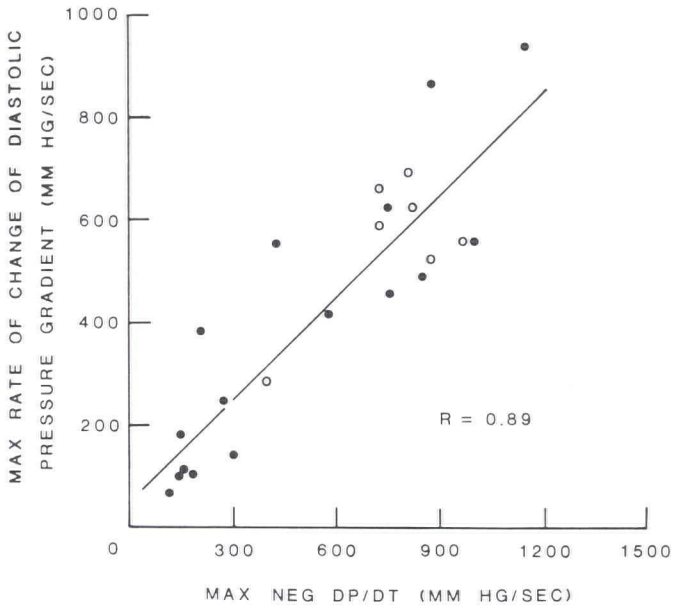

Fig. 4

Relation of maximal negative dp/dt to the maximal rate of change of the diastolic pressure gradient across the closed pulmonary valve. Open circles indicate patients with right ventricular failure. Closed circles indicate no failure.

Discussion

We recently described the hemodynamic and anatomic factors that interact to produce the accentuated P2 in patients with pulmonary hypertension (1) and in the present study showed that P2 remains accentuated in spite of the onset of right ventricular failure. This observation is consistent with the measured hemodynamic parameters that were encountered in patients with pulmonary hypertension and right ventricular failure, i.e., right ventricular maximal negative dp/dt was augmented relative to patients in whom pulmonary arterial pressure was normal. This augmentation resulted in a significant increase in the rate of change of the diastolic pressure gradient, which is a primary hemodynamic determinant of the amplitude of P2 (1-3).

The capability of the failing right ventricle to retain a faster than normal negative dp/dt parallels its capability to maintain a high positive dp/dt (17). In patients with pulmonary hypertensive heart disease and right ventricular failure we observed that maximal dp/dt exceeded values in control subjects, and ^{V}max and 1/p dp/dt remained normal (17).

The accentuated P2 in patients with right ventricular failure secondary to pulmonary hypertension reflects the increased rate of change of the diastolic pressure gradient across the closed pulmonary valve primarily as a result of the capability of the failing right ventricle to retain a faster than normal negative dp/dt. In contradistinction, failure or poor performance of the left ventricle was accompanied by a lower than normal negative dp/dt (4). This phenomenon, through its effect upon the rate of change of the pressure gradient across the aortic valve, resulted in a diminished aortic component of the second sound (4).

The amplitude of P2 in patients with right ventricular failure equaled or exceeded the amplitude of the aortic component of the second sound that we previously reported in patients with normal left ventricular function and normal aortic valves (4). The accentuation of the pulmonary component of the second sound (whether or not accompanied by right ventricular failure) reflects both an augmentation of the rate of development of the diastolic pressure gradient and the interaction of the anatomical structure of the aortic and pulmonary valves (1).

The intensity of both components of the second heart sound may be affected by a variety of interacting hemodynami and anatomic factors (2, 3, 18). Aortic stenosis and aortic insufficiency affect the amplitude of the aortic component of the second sound (16, 19). Predictably, pulmonary stenosis and pulmonary insufficiency would have a similar effect upon the amplitude of P2. Mathematical analysis of semilunar valve vibrations that produce the second sound predicted additional factors that would also affect the intensity of the second sound. Such factors include the density and viscosity of blood, the volume and geometry of the ventricle, and the coefficient of sound absorption of the ventricular wall (3). In this study, we excluded patients with emphysema heart disease (14). The increased blood viscosity, due to the higher hematocrits of patients with emphysema, would diminish the intensity of their heart sounds (20).

Conclusion

This study indicates that P2 is accentuated in patients with pulmonary hypertensive heart disease even after the development of right ventricular failure. Therefore, an accentuated P2 remains valid as a clinical sign of pulmonary hypertension, whether or not right ventricular failure occurs.

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