

Circulatory Changes on Exercise in Patients with Chronic Cor Pulmonale in Regard to Dyspnea and Prognosis

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(Received May 27, 1971)

Circulatory abnormalities in patients with chronic cor pulmonale are mainly pulmonary circulatory disturbance, especially pulmonary arterial hypertension (pulmonary hypertension) and right ventricular hypertrophy and/or right heart failure. In addition abnormal pulmonary functions of basic lung disease will accelerate the circulatory dysfunction.

In this presentation, hemodynamic changes on exercise test and correlation between the circulatory abnormalities and occurrence of dyspnea and prognosis in patients with chronic cor pulmonale, especially due to pulmonary emphysema, were clinically investigated.

SUBJECTS AND METHODS

Clinical course and prognosis were observed in 103 patients with chronic pulmonary diseases, mainly emphysema cases, 81 males and 22 females, aged 34 to 77. In 65 cases of them hemodynamic studies were performed by means of right heart catheterization. The periods of the follow-up were from 1.5 to 10 years after the first examination and 4.4 years on an average.⁴⁾⁵⁾

RESULTS

1) Circulatory responses to exercise test in the patients with chronic lung disease, especially with chronic cor pulmonale.

A moderate bicycle type leg exercise for 6 or 9 minutes was performed in a supine position, and the last one minute oxygen uptake in this test was 349

This paper was read before symposium "cor pulmonale" at the Second Asian-Pacific Congress on Diseases of the Chest, Taipei, Nov. 11-14, 1971.

cc/min/M² on an average. Cardiopulmonary functions reached a steady state in 5 minutes of the exercise, and the values of cardiac output were obtained by Fick's principle²⁾³⁾.

Fig. 1 shows changes of the pulmonary blood pressures and cardiac output on the exercise test in pulmonary patients³⁾. In each case increase of the pulmonary artery mean pressure and/or right ventricular end-diastolic pressure and increase of the cardiac output on the exercise were significant. These pulmonary circulatory responses to the exercise were characteristic in chronic lung disease, especially chronic cor pulmonale patients due to pulmonary emphysema.

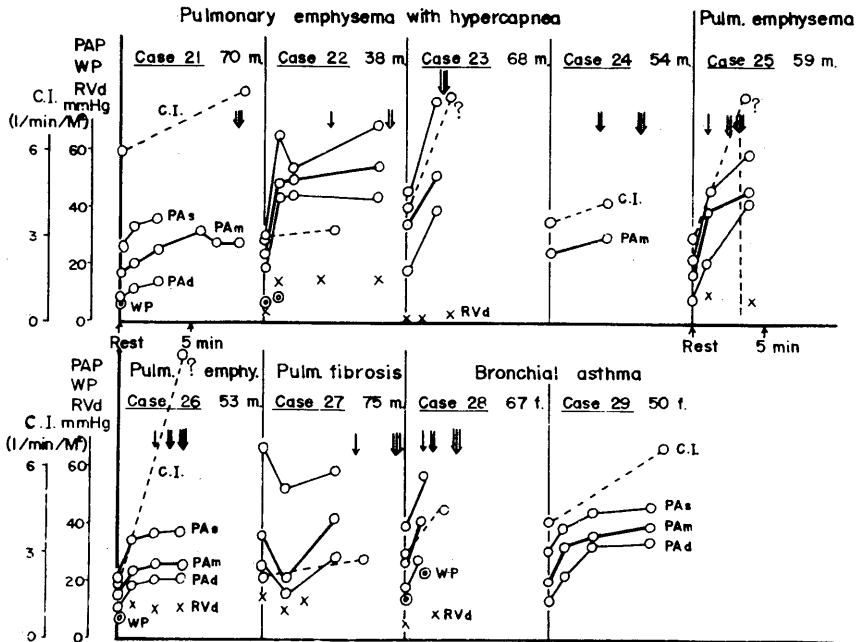


Fig. 1. The grade of dyspnea and the changes of pulmonary hemodynamics on moderate exercise in patients with pulmonary diseases.

Increment of the cardiac output per 100 cc increase in oxygen consumption on the exercise (Δ CO) was calculated¹⁾³⁾. The Δ CO was over 800 cc/min in all normal subjects and under 800 cc/min in many patients with cardiopulmonary disease, especially with chronic cor pulmonale. The patients were divided into two groups, Group 1 is the group with normal Δ CO, Group 2 is the group with lesser Δ CO below 800 cc/min¹⁾³⁾. Cases of Group 1 had the average increase of the cardiac output of 56 % on the exercise and in Group 2 it was only 27 %.

As shown in Fig. 2, Group 1 had an average elevation of pulmonary artery mean pressure (PA_m) of 9 mmHg, and in Group 2 it was 10 mmHg. Many of

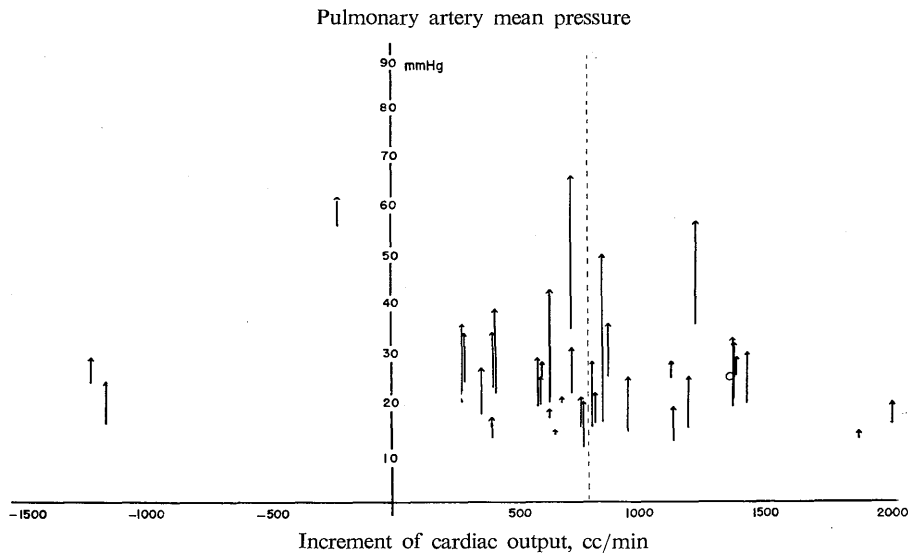


Fig. 2. Relation of pulmonary artery mean pressure to increment of cardiac output in patients with chronic pulmonary disease.

cases who had high resting PAm, especially chronic cor pulmonale patients, were situated in Group 2 which showed lesser ΔCO on the exercise, and they had relatively large elevations of PAm¹⁾.

There were no significant changes of the right ventricular end-diastolic pressure (RVd) on the exercise in normal subjects. In pulmonary patients, however, especially emphysema cases RVd elevated as shown in Fig. 3¹⁾. It was 3 mmHg on an average for Group 1, 4.4 mmHg for Group 2. Many cases with lesser ΔCO had high RVd at rest and relatively marked elevation of RVd on the exercise³⁾.

As described above, pulmonary patients, especially emphysema cases with chronic cor pulmonale shown by pulmonary hypertension, right ventricular hypertrophy and/or right heart failure had abnormal hemodynamic responses to the exercise test. These abnormalities consisted of elevation of the pulmonary artery pressure, lesser increment of the cardiac output per 100 cc increase in oxygen consumption and elevation of the right ventricular end-diastolic pressure on the exercise. These abnormal hemodynamic responses to the exercise will be much helpful for the detection of chronic cor pulmonale in cases of early or obscure cor pulmonale.

2) Relations between occurrence and severity of dyspnea and the pulmonary circulatory disturbances on the exercise.

The cardinal symptoms of the patients with chronic cor pulmonale are shortness of breath and palpitation upon exercise. The pulmonary circulatory responses to

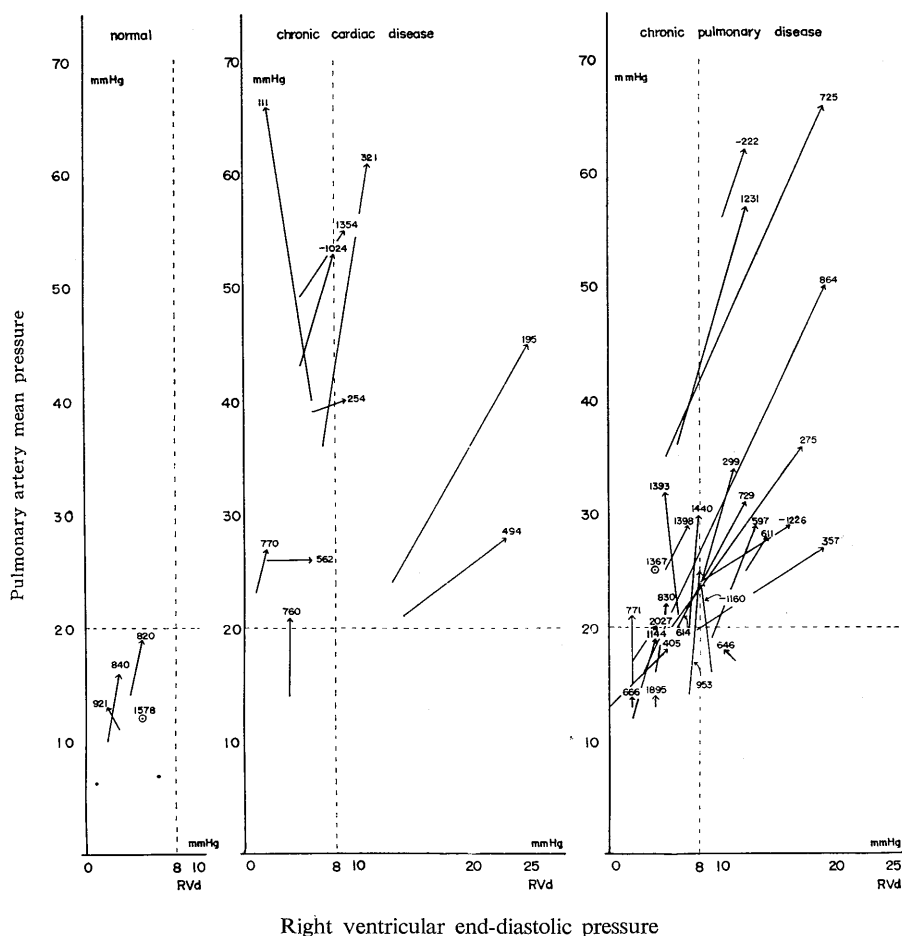


Fig. 3. Changes of pulmonary artery mean pressure and right ventricular end-diastolic pressure on exercise in normal subjects, patients with chronic cardiac disease and patients with chronic pulmonary disease.

the exercise test were specific in those patients as described above. These facts compelled us to take an interest in a study of relationships between the occurrence and severity of dyspnea and the pulmonary circulatory responses.

In normal subjects the sensation of dyspnea was not complained of during this exercise, which was continued 8 to 9 minutes. Change of PAM was a few mmHg, and change of pulmonary artery wedge pressure (WP) was parallel to the change of PAM, but RVD did not change²⁾³⁾. Many patients with pulmonary diseases complained of dyspnea during the exercise. In Fig. 1, 4 and 5 the mark of thin arrow means the time point when the patient recognized the sensation of slight dyspnea. The mark of thick arrow means the time point when the patient recog-

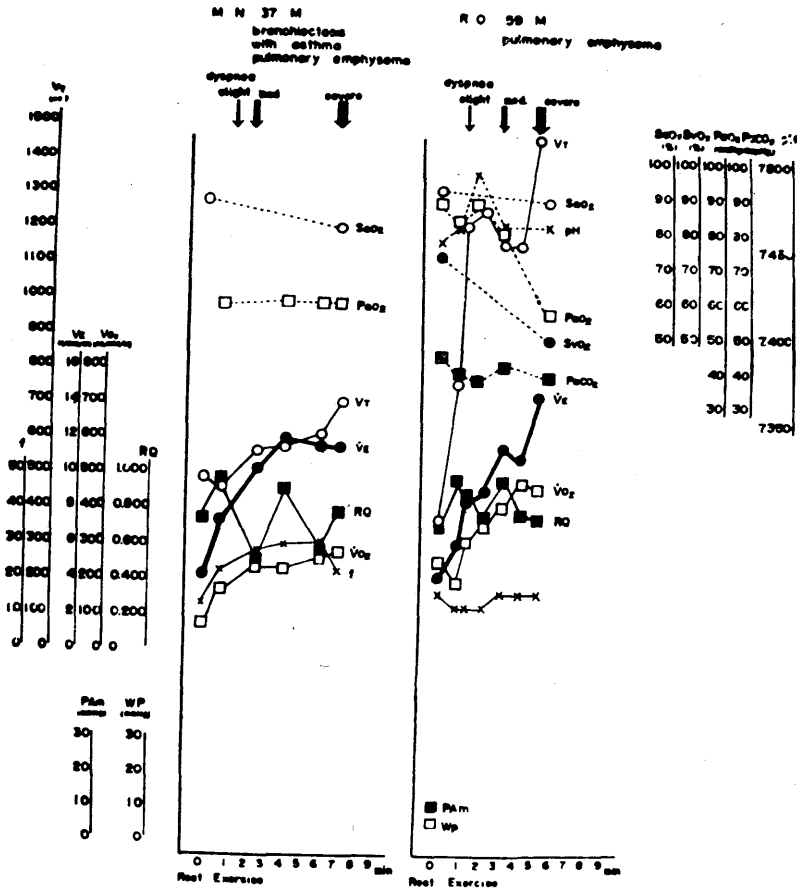


Fig. 4. Sensation of dyspnea and changes of respiratory-circulatory findings on exercise in emphysema patients without hypercapnea

nized the sensation of intolerably severe dyspnea and interrupted the exercise. The mark of middle sized arrow means the time point when the patient had the sensation of dyspnea at about midpoint between those two.

As shown in Fig. 4, on emphysema patients without hypercapnea, at a early stage of exercise, the 1st or 2nd minute, the sensation of slight dyspnea was complained of and was succeeded by moderate dyspnea and next by severe dyspnea in a relatively short period. Arterial blood carbon dioxide tension ($PaCO_2$) was about 40 mmHg as shown in the figure. On the contrary, as shown in Fig. 5, on emphysema patients with hypercapnea of $PaCO_2$ over 50 mmHg at rest, associated with chronic cor pulmonale, the slight dyspnea was not complained of, and at a relatively late period of exercise, the 7 or 8th minute, severe dyspnea occurred suddenly, and the exercise was interrupted²⁾³⁾.

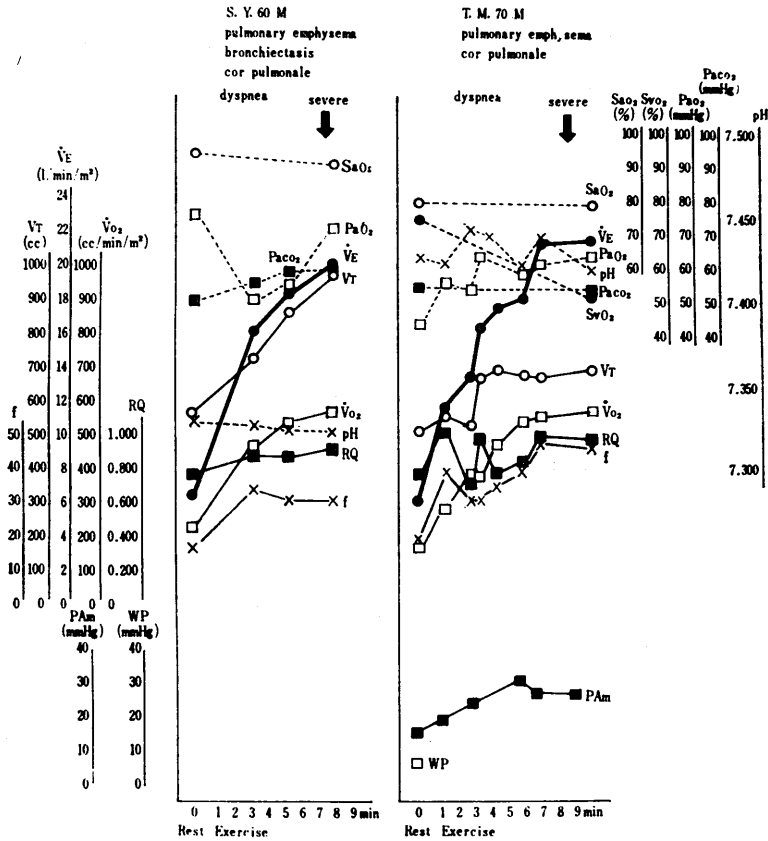


Fig. 5. Sensation of dyspnea and changes of respiratory-circulatory findings on exercise in emphysema patients with hypercapnea and associated with chronic cor pulmonale

Oxygen uptake at the mouth reached nearly the highest value at the 2nd to 5th minute, and then stayed in the highest value. Minute ventilation reached nearly the highest value at the 2nd minute, and then stayed on the same volume or showed a tendency of slight increase.

Mixed venous carbon dioxide tension increased almost 10 mmHg in pulmonary diseases, but reached nearly a steady state at the 5th minute. PaCO₂, however, remained in the same level as at rest. Heart rate also reached a steady state at the 5th minute. Oxygen saturation of mixed venous blood decreased, but did not reached under 30 %. Changes of these blood gases, in cases that could be measured in succession, changes of ventilation and heart rate reached a steady state at the 5th minute¹⁾³⁾.

3) Clinical course and prognosis of patients with chronic cor pulmonale.

Clinical course and prognosis were observed on 103 patients with chronic

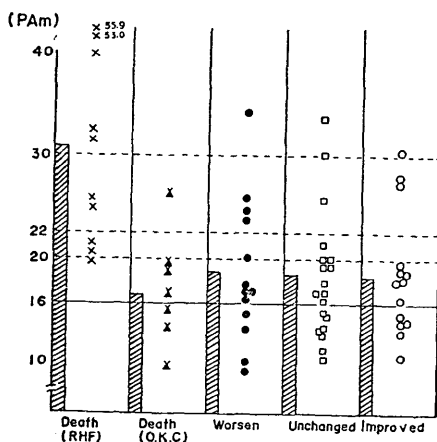


Fig. 6. Values of pulmonary artery mean pressure on each groups divided by their clinical process in patients with chronic pulmonary emphysema. ■ shows average value of pressure in each group.

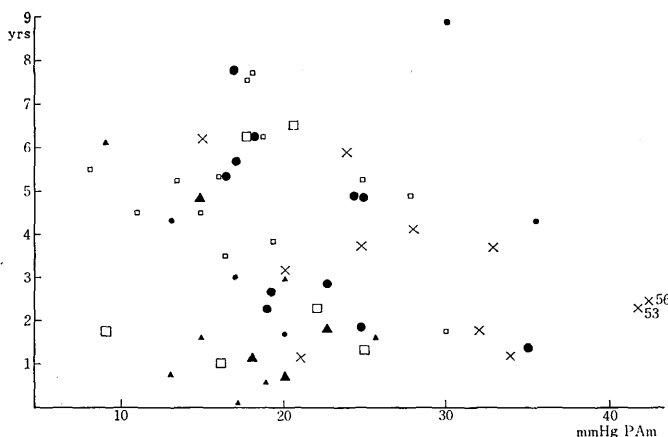


Fig. 7. Relation between pulmonary artery mean pressure(PAm) and duration until last checking-up or until death in connection with clinical course. × : dead, ● : worsen

emphysema, and on 65 cases of them right heart catheterization was performed.

Thirty cases died during periods of the observation. Among 30 cases of "dead", 8 patients died from right heart failure, 5 died from probable right heart failure and the other 17 cases died from other causes as infiltration, malignancy and et cetera. PAm measured at the first examination was shown in Fig. 6, in grouping by changes of the clinical course⁴⁾⁵⁾. PAm in "dead" cases from right heart failure was elevated in a range from 19 to 59 mmHg (31.5 mmHg on an average) at the first examination (Fig. 6), and survival time of the patients who had PAm above 22 mmHg was in a range from 1.2 to 6.2 years (3.3 years on an average)

as shown in Fig. 7⁵). PAm in "dead" from other causes was less than 20 mmHg but one with 26 mmHg (17 mmHg on an average). In "worsen" cases it was 18.9 mmHg on an average, 18.3 mmHg in "unchanged" and 17.9 mmHg on an average in "improved" cases (Fig. 6).

From viewpoint of the pulmonary hypertension, among 8 chronic cor pulmonale cases with PAm over 30 mmHg 5 cases died from right heart failure, 1 got worsen and 2 cases remained unchanged, but none showed any improvement. Eighteen cases with PAm over 22 mmHg varied in the clinical course as follows: "worsen" in 4 cases, "unchanged" in 3, "improved" in 3, "dead" from right heart failure in 7 and "dead" from other cause in only 1 case⁴⁾⁵⁾ (Fig. 7).

SUMMARY AND CONCLUSION

Patients with chronic cor pulmonale showed some characteristic hemodynamic responses to the exercise which were elevation of the pulmonary artery pressure, lesser increment of the cardiac output per 100 cc increase in oxygen consumption and elevation of the right ventricular end-diastolic pressure.

Cases of chronic cor pulmonale, especially due to pulmonary emphysema, with hypercapnea who also have a lesser increment of the cardiac output, complained of severe disabling dyspnea abruptly at a relatively late stage of the exercise, though increase of the pulmonary artery pressure was observed at the same manner as the other pulmonary patients. It would be reasonable to presume that not only sensitivity to carbon dioxide tension but also sensitivity to neural stimuli originated by pulmonary vasculature and other factors increase in patients with hypocapnea and decrease in emphysema patients with hypercapnea and chronic cor pulmonale.

Pulmonary hemodynamic abnormality, especially pulmonary hypertension, is an important factor in predicting the prognosis of chronic lung disease, especially of patients with chronic cor pulmonale.

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