Pulmonary Pulmonary Hypertension

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At the beginning of this presentation, the pulmonary hypertension was classified into three groups as follows:

- 1) Pulmonary pulmonary hypertension
- 2) Pulmonary hypertension in congenital cardiovascular disease
- 3) Pulmonary hypertension due to the left heart disease

This (3) pulmonary hypertension due to the left heart disorder corresponds to the passive pulmonary hypertension (Wood, P.) and is accompanied by the elevation of the pulmonary venous pressure. Pulmonary hypertension due to left to right shunt in congenital cardiovascular disease (2) is equivalent to the hyperkinetic pulmonary hypertension. And pulmonary pulmonary hypertension (1) is caused by the disease of the lung and/or thorax and has possibility to develop the pulmonary heart disease defined by WHO.

Normal pulmonary blood pressures

Pulmonary blood pressures should be discussed with respect to a common reference or zero point. The upper limit of normal pulmonary artery mean pressures measured with the reference point which has been settled at the midpoint between angle of Louis and the back in our laboratory, was 16 mm.Hg. The mean pulmonary artery wedge pressure in Japanese normal adult was found to be from 5 to 9 mm.Hg.

Causal factors of pulmonary pulmonary hypertension

Pathogenesis of the pulmonary pulmonary hypertension will be divided into the one caused by functional disorders of "hypoxia—hypercapnea—polycythemia" and the other by structural changes of the pulmonary vasculature. In order to differentiate the participation of these causal factors in the development of the pulmonary pulmonary hypertension, analytical observation was performed in 200 patients with chronic pulmonary disease or disordered respiratory functions who had high pulmonary artery pressures or vascular resistances.

1. Hypoxia or hypoxemia

In these patients there was no close correlation between the arterial oxygen saturation (Sao_2) and the pulmonary artery mean pressures (PAm) as shown in Fig. 1. However, almost all cases with Sao_2 below 87% had pulmonary hypertension of

Fig. 1. Relation between pulmonary artery mean pressures and arterial oxygen saturation in patients with chronic pulmonary and/or thorax diseases.

PAm above 16 mm.Hg, and the majority of pulmonary hypertensive patients showed Sao_2 in a range of from 95 to 82%. Even in the group of chronic pulmonary emphysema or hypoventilation syndrome, the relation between Sao_2 and PAm was not so close. These data suggest that causal factor of the pulmonary hypertension will be not simple.

Inhalation of 14.5% oxygen: This inhalation for 20 minutes reduced Sao_2 to the value between 90 and 80%. In 5 normal subjects Sao_2 reduced to 83% on an average, and PAm increased from 13 mm.Hg to 16 mm.Hg on an average. In 18 patients with chronic pulmonary disease Sao_2 reduced from 94% to 84% on an average and PAm elevated from 16 mm.Hg to 19 mm.Hg on an average. Pulmonary arterial wedge pressures (WP) and cardiac output did not show significant changes, so that calculated pulmonary vascular resistances (PVRI) increased significantly. The increase of PAm was relatively uniform and the mechanism of the pressure elevation was considered to be due to a pulmonary arteriolar vasoconstriction.

2. Hypercapnea

Between the arterial carbon dioxide tension ($Paco_2$) and PAm no close correlation was found as shown in Fig. 2, but all cases with $Paco_2$ above 53 or 55 mm.Hg had pulmonary hypertension of PAm above 20 mm.Hg and were emphysema patients. Moreover, in many cases with $Paco_2$ above 47 mm.Hg PAm were over 17 mm.Hg.

Inhalation of 4.5% carbon dioxide: This inhalation for 20 minutes increased $Paco_2$ to the value between 47 and 60 mm.Hg. In 18 normal subjects PAm did not change and $Paco_2$ increased to 47 mm.Hg on an average. In 14 patients with chronic pulmonary emphysema PAm rose from 19 mm.Hg to 24 mm.Hg on an average, and

Fig. 2. Relation between pulmonary artery mean pressures and arterial carbon dioxide tensions in patients with chronic pulmonary and/or thorax diseases.

$Paco_2$ increased from 45 mm.Hg to 55 mm.Hg on an average. In 9 patients with the other pulmonary disease PAm elevated from 18 mm.Hg to 21 mm.Hg on an average and $Paco_2$ increased from 43 mm.Hg to 53 mm.Hg on an average. Calculated PVRI was increased in all pulmonary patients.

By contrast 25 cardiac patients showed evident rise of PAm and WP too, but PVRI were reduced. This experiment revealed that the elevation of the pulmonary artery pressure occurred in only the group of pulmonary patients and many of such cases showed lowered sensitivity and irritability of the respiratory center.

As described above, the increase of PAm through these acutely induced hypoxia and hypercapnea were within only several mm.Hg.

Effects of hyperoxia upon the pulmonary hypertension

i) 40% oxygen inhalation: This inhalation for 20 minutes decreased PAm as follows. In 15 emphysema patients PAm fell from 24 mm.Hg to 22 mm.Hg on an average, and in 14 patients with the other pulmonary disease PAm were lowered from 24 mm.Hg to 21 mm.Hg. And PVRI reduced in all cases.

ii) 100% oxygen inhalation: This inhalation decreased PAm a little bit more than by 40% oxygen. Five normal subjects showed PAm decreased from 14 mm.Hg to 11 mm.Hg on an average. In 8 emphysema patients PAm fell from 26 mm.Hg to 22 mm.Hg on an average, and in 8 cases of the other pulmonary disease PAm decreased from 22 mm.Hg to 19 mm.Hg too.

Both oxygen inhalation raised Sao_2 up to 100% in almost all cases, and PVRI decreased significantly. Emphysema patients who inhaled 100% oxygen showed

Paco₂ increased from 44 mm.Hg to 46 mm.Hg on an average, but the patients with moderately high Paco₂ had the inhalation of 40% oxygen only.

By inhalation of 40 or 100% oxygen the reduction of PAm was within several mm.Hg, and pulmonary hypertension of PAm above 25 mm.Hg did not fall to the normal pulmonary blood pressures and most cases with PAm above 20 mm.Hg also did not return to the normal.

iii) Polycythemia: In cases of pulmonary pulmonary hypertension polycythemia was encountered relatively rarely in practice.

Effects of reserpine upon the pulmonary hypertension

Reserpine 1 or 2 mg. was administered intramuscularly and after 90 minutes changes of the pulmonary circulation and respiratory functions were observed. In 9 patients of chronic pulmonary disease, among whom 7 cases had pulmonary hypertension, PAm fell from 21 mm.Hg to 18 mm.Hg on an average. There were no significant changes in WP and cardiac output, so that PVRI reduced significantly. The brachial artery blood pressures (BAm) also decreased slightly. It was interesting that the injection of reserpine showed significant decrease of Sao₂ in all cases, and especially in chronic pulmonary patients the reduction was -8% on an average. The pulmonary hypertensive cases with PAm below 20 mm.Hg showed the fall to

Fig. 3. Relation between pulmonary artery mean pressures and electrocardiographic right ventricular hypertrophy, hematocrit, edema, liver swelling and duration of illness in patients with chronic pulmonary and/or thorax diseases.

the normal pulmonary blood pressure, but cases with PAm above 20 mm.Hg did not return to the normal pressure.

From pulmonary pulmonary hypertension to chronic cor pulmonale

Electrocardiographic right ventricular hypertrophy was found, as shown in Fig. 3, in all cases of pulmonary pulmonary hypertension with PAm above 35 mm.Hg, in a high rate in the cases above 30 mm.Hg, in the cases above 25 mm.Hg too, and came across in cases with pressures above 20 mm.Hg. Patients with edema or liver swelling showed PAm above 20 mm.Hg.

Summary

Through these experimental observations it was considered that in the patients with pulmonary pulmonary hypertension of PAm above 25 mm.Hg irreversible changes of the pulmonary vasculature such as structural damage, avascularity and fibrosis or semi-permanent contracture of the pulmonary arterioles besides functional disorders of "hypoxia—hypercapnea—polycythemia" would be participated as the causal factors. The fact that a rough correlation presents between hypercapnea and pulmonary hypertension suggests that structural changes of the lung promote the pulmonary hypertension and aggravate the hypoventilation on the other hand. The semi-permanent arteriolar contracture would have a possibility to be loosened by some strong medicaments in future.

On exertion, the pulmonary pulmonary hypertension shows a marked elevation of PAm, and increments of cardiac output per 100 cc/min. increase of oxygen consumption fell occasionally below 600 or 800 cc/min. and sometimes were negative.