

vertically on left side of sternum in 4th intercostal space, and the other was collimated to cardiac apex with  $30^\circ$  angle obliquely. 50  $\mu\text{Ci}$  of  $^{131}\text{I}$  as RISA was then rapidly injected into an antecubital vein. At the moment of injection, the recording was begun. Two different shapes of radiocardiogram well obtained; pulmonary circulation time was derived by the method as previously described by Nakazima and associates (presented at the 6th Annual Meeting of Japanese Society of Nuclear Medicine). Cardiac index was calculated using the technique by McIntyre et al. Pulmonary blood volume was calculated from cardiac index multiplied by pulmonary circulation time. Circulating blood volume was obtained by division of injected RISA counts by sample blood counts at 5 minutes. After breathing quietly 12 per cent oxygen for 10 minutes, radiocardiogram was again recorded in the same manner and hemodynamic variables are calculated as described above. The values of cardiac index calculated from left heart detection method was evidenced to be approximately the same as that from right heart detection method.

#### Results

Cardiac index increased in almost all cases; especially in 3 normal subjects it increased markedly. Pulmonary circulation time

generally decreased, but in some cases it did not change significantly and in only one it was prolonged. Circulating blood volume and pulmonary blood volume increased slightly. In majority of cases that cardiac index increased, slightly. In majority of cases that cardiac index increased, pulmonary circulation time decreased accordingly. No significant correlation was found between circulating blood volume and pulmonary blood volume.

#### Discussion

The effects of acute hypoxia on pulmonary hemodynamics revealed generally increase in cardiac index, shortening in pulmonary circulation time, and pulmonary blood volume was slightly increased or unchanged. Increase in cardiac index in normal subjects was larger than that in cardiopulmonary patients. This suggested that the reaction of cardiovascular system during hypoxia may decrease in cardiopulmonary patients as compared with normal subjects. Pulmonary circulation time obtained by precordial counting method might include some components of circulation times in both right and left heart, so pulmonary blood volume in this report would include the elements of cardiac blood volume. Alterations of true pulmonary blood volume could not be examined. In this investigation hypoxic effects on pulmonary blood volume was not discussed.

### Study on the Measurement of Pulmonary Blood Volume (PBV) with Radio-Cardiogram (RCG).

#### — An analysis of RCG by its analog computer simulation —

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In 1957, Lammerant reported that PBV might be calculated from the peak-to-peak time of RCG, multiplied by cardiac output. It has been reported by a number of investigators that the peak-to-peak time does not represent the true mean pulmonary circulation time. An analog computer analysis of RCG simulated by an appropriate mathematical

model has been developed by Kuwahara et al, which makes to be able to quantify cardiac output and equivalent volumes of right and left hearts, pulmonary and body blood vessels. PBV can be calculated from the following equation by using parameter values of the mathematical model,  $F$ ,  $V_p$ ,  $\tau_p$  and  $T_p$ , where  $F$  (ml/sec) is a mean blood flow rate,  $V_p$  (ml)

represents equivalent volume of pulmonary blood vessel and  $\tau_p$ (sec) represent transportation lag in pulmonary system;

$$\begin{aligned} \text{PBV} &= V_p + F \times t_p \\ &= F \times V_p / F + F \times \tau_p \\ &= F \times (T_p + \tau_p) \end{aligned}$$

where  $T = V / F$  (sec) is a time constant of pulmonary blood vessel.  $T + \tau$  represent a mean pulmonary circulation time.

The peak-to-peak time is shortened or lengthened by changing the parameters  $V$ ,  $V_1$  and when PBV is constant; where  $V$  represents an equivalent volume of right heart, and is the time required for injection of RISA. As  $V$  becomes gradually larger, the peak of RCG for right heart shifts to the peak for left, that is, the peak-to-peak time becomes short. As  $V_1$  becomes larger, the peak of RCG for left heart shifts to the left; that is, the peak-to-peak time becomes long. Four cases are represented: The first case is normal control. The cardiac output measured by Fick method shows 8270 ml/min, the stroke volume is 90 ml/beat and the computed value for cardiac output and stroke volume is 7850 ml/min and 84 ml/beat, respectively.  $V$  is 111 ml/sy.M.  $V_1$ , 102 ml/Sy.M., PBV, 263 ml/Sq.M. The second case is a patient with primary myocardial disease. The venous pressure is 208 mm H<sub>2</sub>O. The cardiac output is

decreased (2850 ml/min).  $V$  and  $V_1$  is 190 ml/Sq.M. and 477 ml/Sq.M., respectively. This fact means that  $V_1$  is 2.5 times as large as  $V$ . PBV, however, is normal (275 ml/Sq.M.) The third case is a patient with mitral stenosis. This case has no distinctive peak in RCG. The cardiac output is 2630 ml/min by Fick method, and this value is in good agreement with a result by the computer (2730 ml/min). Stroke volume is decreased (36 ml/beat).  $V$  (542 ml) and  $V_1$  (542 ml) are extremely increased. PBV is normal (259 ml/Sq.M.) The fourth case is a patient with atrial septal defect. Up to now in patient with intracardiac shunt, PBV could not be calculated from FCG. In this case, however, this analog computer gives pulmonary blood flow and volume. By Fick method the pulmonary blood flow is 9442 ml/min systemic blood flow is 4408 ml/min, and L. to R. shunt of pulmonary blood flow is 53%. By our analog computer, pulmonary blood flow is 11430 ml/min, systemic blood flow is 3650 ml/min, L. to R. shunt of pulmonary blood flow is 68%. PBV is 397 ml/Sq.M. (upper limit of normal). Generally speaking, some difference between the peak-to-peak time and computer derived pulmonary circulation time was found in cases with increased pulmonary circulation time.

## Studies on the Regional Pulmonary Function by Radioactive <sup>131</sup>Xenon

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Effect of low oxygen breathing for the regional blood flow and regional ventilation were studied by isotope-pulmography using the radioactive <sup>133</sup>Xe. Six young healthy men were selected as subjects for study. Regional pulmonary blood flow ( $Q_c$ ) and regional ventilation ( $V$ ) in six regions, that is, upper, middle and lower region of both lungs were measured by six scintillation counters both on the sitting and supine position. Overall pulmonary blood flow and ventilation were also measured.

Low oxygen breathing, 10-11% oxygen, were given for 10-15 minutes and the plateau of

arterial oxygen saturation were obtained by ear oximeter. Regional pulmonary blood flow was measured before low oxygen breathing, during low oxygen breathing and after 15 minutes of air breathing and regional pulmonary ventilation was measured before and during low oxygen breathing.

Overall pulmonary blood flow was increased by low oxygen breathing both on the sitting and supine position. Overall pulmonary ventilation also increased by low oxygen breathing except one case.

Regional pulmonary blood flow was markedly increased in the upper region but moderate-