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

$$PBV = V_p + F \times t_p$$
$$= F \times V_p/F + F \times \tau_p$$
$$= F \times (T_p + \tau_p)$$

where $T = V /(sec)$ 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 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_2O. 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 Vr. PBV, however, is normal (275 ml/Sq.M.) The third case is a patient with mitral steno-insufficiency. 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 tim was found in cases with increased pulmonary circulation time.

Studies on the Regional Pulmonary Function by Radioactive [131]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 [133]Xe. Six young healthy men were selected as subjects for study. Regional pulmonary blood flow (Qc) 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-

ly decreased in the lower region on the sitting position by low oxygen breathing. However, the regional pulmonary ventilation was not changed significantly in any regions.

The regional ventilation-perfusion ratio (V /Qc) was decreased from the upper region by air breathing, and increased on the contrary by low oxygen breathing.

On the supine position the regional pulmonary blood flow and bentilation in air breathing was distributed uniformly over the lung, and V /Qc ratio was also uniformly distributed. However, it showed the slight increase from the upper region to lower region because the pulmonary blood flow in the upper region showed the marked increase as much as 1.5 times of the one in the air breathing.

Regional pulmonary ventilation showed no significant change by the low oxygen breathing both on the sitting and supine position. These findings were obtained directly by the method of isotope-pulmography (^{133}Xe).

Concerning the mechanism which the regional pulmonary blood flow showed the difference in the upper and lower region for the low oxygen breathing is not clear yet, however, it seems to me that it might be due to the difference of grade of reaction of the pulmonary capillary bed for the low oxygen breathing between the upper and lower region.

Application of ^{131}I-labeled Antipyrine to Pulmonary Circulation

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Now pulmonary blood volume (PBV) in cardiopulmonary disorders has been well documented using nondiffusible tracers such as various dyes and RISA. Pulmonary extravascular water volume, however, is little studied.

Chinard in 1951 first demonstrated that two indicators with different capacities for passing through capillary walls produce time concentration curves of different shape after passing through capillary bed. Since that time, a variety of indicators and several methods of analysis of the differences in the dilution curves have been attempted and the estimation of pulmonary edema in animals and in man has been fairly performed by several workers (Ramsey, 1964; Pearce, 1965; Levine, 1965; McCredie, 1967.).

In this study, two indicators of RISA as nondiffusible tracer and ^{131}I labeled antipyrine as diffusible tracer were used to estimate pulmonary extravascular antipyrine space (PEV) by precordial counting technique.
Methods

Five healthy subjects and nine patients with cardiopulmonary diseases were studied. Six patients were with cardiac disease; 5 of them with mitral stenosis and 1 of them with congestive heart failure due to ischemic heart disease. Remaining three patients were with pulmonary disease.

Recording apparatus with two channel scintillation counters was employed. One counter was positioned in the 4th intercostal space on the left sternal line to catch dominantly right heart dilution process, and another counter was placed at cardiac apex with an angle of 30 degrees from vertical line to catch mainly left heart dilution process. Indicator dilution curves were obtained as radiocardiogram by external monitoring following sequential injections of RISA and labeled antipyrine. From each dilution curve two different types of pulmonary circulation times are obtained; one is peak to pesk time (tp) which is obtained directly as an interval from right peak to left peak,, and the other is mean to mean time (tm) which is calculated from isolation of right and left heart dilution curves with extrapolation. The difference (Δt) in pulmonary circulation times of RISA and Antipyrine was calculated. Δtp is derived from the difference in tp and Δtm from that tm. The area under rleft heart dilution curve of antipyrine was approximately equal to the area under the corresponding RISA curve, when corrected by injected counts. The pulmonary