

Quantitative Assessment of Pulmonary Extravascular Water Volume with THO

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Double indicator dilution technique suggested by Chinard & Enns (1951) made it possible to measure the extravascular water content of lungs in vivo.

Pulmonary extravascular water volume (PEV) was measured in our laboratory applying the modified technique of double indicator dilution using RISA and THO system by Ramsey et al. Current study was done to clarify the relationship of PEV with pulmonary hemodynamics and with arterial blood gas level in the acquired heart diseases.

Methods: Four normal subjects and twenty-four patients with acquired heart disease were studied. About 0.6 ml of physiological saline containing 100 μ Ci of THO and 20 μ Ci of 131 I-RISA was rapidly injected into the median cubital vein by tourniquet method. Blood was sampled continuously on rotating turntable from the brachial artery by vacuum suction pump.

131 I activity was counted in well-type scintillation counter and 3 H (Tritium) in Nuclear Chicago liquid scintillation spectrometer with 10 per cent of counting efficiency.

Calculation: Time-concentration curves for both RISA and THO were plotted on semi-logarithmic paper. Each curve without recirculation was obtained. Cardiac output and mean circulation time were calculated in usual manner. The difference between dis-

tribution volume of THO and RISA was used as PEV.

Results: Patients with episodes of cardiac decompensation, even though they had no symptoms at the time of study, had apparently higher value for PEV. Some patients of neither episode nor clinical signs showed also higher value. There was no apparent relationship between PEV and cardiac output, but some cases with high PEV showed low cardiac output indicating heart failure. The study of the relationship between PEV and pulmonary blood volume (PBV) showed no obvious correlation, and some patients with high PEV had normal PBV. PEV correlated with R-L time obtained by precordial monitoring. There was no apparent relationship between PEV and mean pulmonary arterial pressure (PPAm). Though some cases with high PPAm had high PEV, other cases with high PPAm had normal PEV. Group with normal PPAm showed normal PEV.

There were the approximately similar relations between PEV and mean left atrial pressure, and mean pulmonary arterial wedge pressure which reflecting pulmonary venous side. There was a close inverse correlation between arterial oxygen tension (PaO_2) and PEV, while no correlation was found between arterial CO_2 tension and PEV.

Lung Density Measurement by γ -Ray Thickness Gauge

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Lung densities of chronic obstructive lung disease were measured, which had been little reported up to now.

The purpose of this study is to observe the differences of the lung densities which may be influenced by parenchymal destruction, over-

inflation and interstitial fibrosis of lung.

Method and materials; γ -ray thickness gauge was used, that was composed of ^{137}Cs γ -ray source, moving table and scintillation counter. With this apparatus, chest thickness (water equivalent thickness) was measured on the right medio-clavicular line using scanning method.

The lung density was calculated as follows;

$$\text{Lung density (g/cm}^3\text{)} = \frac{\text{chest thickness(water equivalent)} - \text{thorax soft tissue thickness}}{\text{geometrical lung thickness}}$$

Denominator of this formula indicates lung parenchymal thickness. Soft tissue thickness is measured using X-ray film.

103 subjects were studied, those included 15 controls, 41 chronic pulmonary emphysema, 33 chronic bronchitis and 13 bronchial asthma.

Results; The lung densities of control group are $0,33 \pm 0,04 \text{ g/cm}^3$, chronic pulmonary

emphysema group $0,08 - 0,28 \text{ g/cm}^3$, but in this group, only one case indicated exceptionally high density, $0,43 \text{ g/cm}^3$, chronic bronchitis group $0,17 - 0,42 \text{ g/cm}^3$ and bronchial asthma group indicate almost same results as bronchitis.

The low density cases, whose densities were less than $0,20 \text{ g/cm}^3$, were observed 32 cases of 41 emphysema patients, and 21 cases of 33 chronic bronchitis patients. In general, on emphysema lung density was considerably low, and on chronic bronchitis and asthma the density between the control group and the emphysema group.

With this method, I think that quantitative observation of lung tissue destruction can be made on various lung diseases.

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