unilateral lung under fluoroscopy. All patients were studied by chest radiograms, spirometry, maximum expiratory flow volume curves and perfusion lung scanning before, immediately, 4, 24, 48, and 72 hours after bronchography. Either $^{99m}$Tc-albumin microsphere or $^{99m}$Tc-MAA was used for perfusion lung scanning except for at 4 hours after bronchography when $^{131}$I-MAA was used. Perfusion partition to the right and left lungs was calculated by integrating the cps curves of both lungs simultaneously obtained at the time of scanning. Functional loss of perfusion of the ipsilateral lung was calculated by using Birath's formula (1957).

Perfusion reduction of the ipsilateral lung was most significant immediately after bronchography. Functional loss was from 15 to 73% (average 42±6.5%). Perfusion recovery was steady as time elapsed after bronchography; namely, functional loss was 23±9.8% at 4, 10±6.9% at 24, and 4±4.9% at 72 hours. Nine of the 11 patients showed a perfusion recovery at 24 hours. There was little change in vital capacity, maximal mid-expiratory flow rate and timed vital capacity before and after bronchography. Disappearance of 60% urokin from the lung was also rapid, leaving the so-called millimeter patterns after 4 hours. Peak flow rate ($V_{\text{peak}}$) and flow rate at 75% of vital capacity ($V_{75}$) decreased most significantly at 4 hours, but flow rates at 50% ($V_{50}$) and 25% ($V_{25}$) of vital capacity were most depressed at 24 hours and gradually recovered thereafter. $V_{\text{peak}}$ and $V_{75}$ were almost parallel with perfusion changes of the bronchographed lung but $V_{50}$ and $V_{25}$ lagged behind perfusion changes.

Perfusion reduction seems to result from a decrease in alveolar ventilation due to airway narrowing and obstruction by contrast medium and occurs immediately following bronchography, whereas $V_{50}$ and $V_{25}$ seem to reflect narrowing and obstruction of the small airways which are not well appreciated on perfusion lung scans.

**Relationship between Pulmonary Regional Ventilation and Perfusion in Chronic Obstructive Lung Disease**

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In order to estimate the relationship between regional ventilation and perfusion of the lung, 18 patients of which 5 with chronic obstructive lung disease (COLD), 6 with the other lung disease than COLD (Non-COLD group) and 7 without lung disease as a control were examined using a scintillation camera with a diverging collimator led to minicomputer system.

With the patients in supine position, the perfusion image in the lung was obtained from the counts of radioactivity of $^{133}$Xe which was
dissolved in saline solution and injected intravenously before the procedures. The ventilation image on the lung, on the other hand, was obtained from the counts of radioactivity of inhaled $^{133}$Xe gas. Scintiphotograph was taken during breath holding for 10 to 20 sec. after a single deep breath.

The regional radioactivities on the ventilation and perfusion in terms of the counts in each 55 ($=11 \times 5$) matrices converged from 1375 ($=55 \times 25$) matrices in each unilateral lung which was postulated to represent a regional lung field were recorded on the computer system.

Alveolar ventilation volume ($\dot{V}_{alv}$) was calculated as follows:

$$\dot{V}_{alv}(ml/m^2) = \frac{\text{minute ventilation volume}}{\text{dead space}} \times \frac{\text{respiratory frequency}}{\text{BSA (m}^2\text{)}}$$

Regional ventilation volume ($V$) was got by following calculation:

$$V = \dot{V}_{alv} \times \frac{\text{individual count in each regions}}{\text{total count in entire lung field}}$$

Cardiac output was obtained by $^{99m}$Tc-albumin technique and regional perfusion ($Q$) were calculated in the same way as regional ventilation. Furthermore, regional ventilation-perfusion ratio ($V/Q$) was calculated from $V$ and $Q$. Finally, the representation of distribution with mean and standard deviation, image and histogram display of the regional $V/Q$ were obtained by the computer system.

As results, the distribution pattern of regional $V/Q$ was diffusely uniform in control group, but in COLD group was changed markedly. Histogram display of regional $V/Q$ had a high and sharp peak in controls, while low or several indented peaks and long lower slopes with irregularity were seen in COLD group.

### Aerosol Deposition in the Central Airways

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Aerosol deposition in the human respiratory tract was studied by theoretical and experimental methods. Theoretical analysis was obtained by applying Weibel's model and deposition equations under laminar flow. Aerosol particle was considered to be dry, spherical and monodisperse aerosol. The calculation was done during one cycle of respiration. According to our calculation, the flow speed of inhaled air in the central airways was high because total volumes of each bronchial generation were not so increased in these area. Therefore, impaction was the major mechanism of deposition in central airways when larger particles were inhaled. More deep inspiration did not cause peripheral penetration of these particles.

This was because filtration of central airways to larger particles became more effective by taking deep inspiration with same cycle. Segmental bronchi had specific features in aerosol deposition. Larger particles deposited most in