THE REGIONAL DISTRIBUTION OF \( Q \) AND \( V_{A}/V \) MEASURED WITH KR-81m

The regional distribution of \( Q \) and \( V_{A}/V \) in the lungs was obtained from the continuous infusion of KR-81m. Measurements were made in twelve patients with lung disease, who were instructed to breath normally after 10 seconds-breathe-holding. There was a good agreement between KR-81m lung counts (Ce) during 10 second-breath-holding and Tc-99m MA A lung counts. \( r = 0.097 \)

During a steady state, which occurs as early as after several breaths at tidal volume, the arrival of KR-81m in the alveolar compartments to the removal of KR-81m by ventilation \( (C_m V_{A}/V) \) and radioactive decay of KR-81m \( (C_m(1-e^{-\lambda t})) \), where \( C_m \) is KR-81m lung counts during a steady state, \( \lambda \) is the radioactive decay constant. In practice, KR-81m counts in the pulmonary artery \( (Ca) \) should be subtracted from Ce and Cm. Ca can be obtained from a early image during KR-81m continuous infusion. 

\( V_{A}/V \) corrected by Ca was a good agreement with \( V_{A}/V_{EC} \) in twelve patients with lung disease, \( r = 0.922 \) therefore our method is available for a quantitative analysis of regional lung function.

REGIONAL PULMONARY PERFUSION, ALVEOLAR OXYGEN TENSION AND PHARMACOLOGICAL EFFECTS

Regional pulmonary arterial perfusion is regulated by regional alveolar oxygen tension. Alveolar hypoxia induces regional hypoxic vasoconstriction and alveolar hypoxemia, hyperoxic recruitment of the pulmonary vascular beds and/or potentially vasodilation. The following is a summary of our drug study which we conducted to examine whether the drugs used could alter pulmonary vascular responses to alveolar hypoxia and hyperoxia. Each drug was administered systemically by drip infusion. The right upper lobe of an adult mongrel dog was isolated in vivo by a balloon catheter and artificial ventilation was done by using nitrogen, air and 60% oxygen to induce alveolar hypoxia, normoxia and hyperoxia, respectively, while the rest of the lung kept a spontaneous air respiration. Amniphylline \( (0.25 mg/kg/min) \) induced further regional hypoxic vasoconstriction but acted as a vasodilator under alveolar hypoxia. Isoproterenol \( (0.1 mg/kg/min) \) and noradrenaline \( (1 \mu g/kg/min) \) abolished regional hypoxic vasoconstriction but their actions were blocked by pretreatment with propranolol and phenoxybenzamine, respectively. In the reimplanted lung, however, propranolol did not counteract isoproterenol. Propranolol per se (1 mg/kg) and dopamine \( (30 \mu g/kg/min) \) did not change vascular responses to alveolar oxygen tension. Prostaglandin \( PFX \) reduced regional perfusion under alveolar hyperoxia. Allegedly selective \( \beta \)-adrenoreceptor stimulators, salbutamol \( (0.1 mg/kg/min) \) and procarotol \( (5 mg/kg/min) \) did not ameliorate vascular responses to alveolar hypoxia.

PERFUSION AND VENTILATION SCINTIGRAPHY IN LUNG DISEASES

Clinical usefulness of perfusion scintigraphy by Tc-99m-W.A.A. combined with ventilation scintigraphy by Xe-133 gas was emphasized in 68 cases (Table) of lung diseases. Images of the case of lung cancer (hilus) anomaly of pulmonary artery, follow up after lung operation and Swyer-James were highly evaluated in this series.

<table>
<thead>
<tr>
<th>Lung ca.</th>
<th>chr. bronchitis</th>
<th>lung tuberculosis</th>
<th>sarcoidosis</th>
<th>pneumonia</th>
<th>pneumothorax</th>
<th>bulia</th>
<th>anomaly of PA</th>
<th>aortitis</th>
<th>emphysema</th>
<th>pneumoconiosis</th>
<th>relaxatio of diafragm</th>
<th>Swyer-James Synd.</th>
<th>misc.</th>
</tr>
</thead>
<tbody>
<tr>
<td>16</td>
<td>11</td>
<td>4</td>
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<td>3</td>
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</tr>
</tbody>
</table>

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REGIONAL PULMONARY PERFUSION, ALVEOLAR OXYGEN TENSION AND PHARMACOLOGICAL EFFECTS

Regional pulmonary arterial perfusion is regulated by regional alveolar oxygen tension. Alveolar hypoxia induces regional hypoxic vasoconstriction and alveolar hypoxemia. Hyperoxic recruitment of the pulmonary vascular beds and/or potentially vasodilation. Each drug was administered systemically by drip infusion. The right upper lobe of an adult mongrel dog was isolated in vivo by a balloon catheter and artificial ventilation was done by using nitrogen, air and 60% oxygen to induce alveolar hypoxia, normoxia and hyperoxia, respectively, while the rest of the lung kept a spontaneous air respiration. Amniphylline \( (0.25 mg/kg/min) \) induced further regional hypoxic vasoconstriction but acted as a vasodilator under alveolar hypoxia. Isoproterenol \( (0.1 mg/kg/min) \) and noradrenaline \( (1 \mu g/kg/min) \) abolished regional hypoxic vasoconstriction but their actions were blocked by pretreatment with propranolol and phenoxybenzamine, respectively. In the reimplanted lung, however, propranolol did not counteract isoproterenol. Propranolol per se \( (1 mg/kg) \) and dopamine \( (30 \mu g/kg/min) \) did not change vascular responses to alveolar oxygen tension. Prostaglandin \( PFX \) reduced regional perfusion under alveolar hyperoxia. Allegedly selective \( \beta \)-adrenoreceptor stimulators, salbutamol \( (0.1 mg/kg/min) \) and procarotol \( (5 mg/kg/min) \) did not ameliorate vascular responses to alveolar hypoxia.