Regulation of Regional Pulmonary Perfusion by Oxygen and Carbon Dioxide

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The purpose of the present study is to clarify further the effect of oxygen (O₂) and carbon dioxide (CO₂) concentration in exchange gas upon the distribution of regional perfusion in the lungs.

A balloon catheter was inserted into the orifice of the right upper lobe (RUL) bronchus of an anesthetized dog under fiberoptic bronchoscopic guidance and isolated the RUL from the remainder of the lungs. The RUL was artificially ventilated with a test gas of interest in the tidal alveolar pressure range, while the remainder of the right lung and the left lung kept a spontaneous air breathing. The test gases were pure N₂, 10% O₂ in N₂, air, 40% O₂ in N₂, 60% O₂ in N₂ and 100% O₂ for studying the effect of O₂ concentration and 2%, 5%, and 7% CO₂ in air and 10% CO₂ in N₂, in air, in 40% O₂ in N₂, in 60% O₂ in N₂, in 100% O₂ and 20% CO₂ in 60% O₂ in N₂ for studying the effect of CO₂ concentration on regional perfusion distribution in the lungs. At the end of the 7th gas exchange of the RUL, ⁹⁹mTc-albumin microsphere was injected intravenously and radioactivity was measured by a gamma camera for each lung region of interest. Normal dogs and dogs with the reimplanted or denervated right lung were studied. In all experiments perfusion percentages in the RUL and the entire right lung were compared with those obtained when the RUL was artificially ventilated with air, and perfusion ratios were calculated.

Perfusion ratios in the RUL decreased when the lobe was artificially ventilated with hypoxic gases such as N₂ or 10% O₂ in N₂, while they increased in a dose-response fashion as the O₂ concentration in the exchange gas increased, indicating the occurrence of regional hypoxic vasoconstriction and hyperoxic vasodilation, respectively. Perfusion ratios in the right lung as a whole remained nearly constant despite artificial ventilation of the RUL with various test gases of different O₂ concentrations, indicating intrapulmonary redistribution of perfusion in the right lung. There was no difference in the mode and degree in the regional vascular response of hypoxic vasoconstriction or hyperoxic vasodilation between the normal and the denervated lungs.

There was no change in perfusion ratios in the RUL when the lobe was artificially ventilated with air mixed with CO₂ of less than 7% in concentration. When the normal RUL was artificially ventilated with 10% or 20% CO₂ mixed in the above mentioned gases of different O₂ concentrations, perfusion ratios in the RUL were similar to those observed when the lobe was ventilated with gas mixtures of different O₂ concentrations without 10% or 20% CO₂. However, when the RUL of the denervated right lung was artificially ventilated with 10% or 20% CO₂ in gases with 40% or more O₂ in concentration, perfusion increased significantly as compared with those observed with gases with 40% or more O₂ in concentration without 10% or 20% CO₂, indicating the presence of hypercarbic vasodilation, while addition of 10% CO₂ to hypoxic gases didn't show any evidence of hypercarbic vasodilation.

In conclusion, regional alveolar hypoxia and hyperoxia induce regional vasoconstriction and vasodilation, respectively in both normal and denervated lungs. No nervous integrity seems to be required for this vascular response to O₂ concentration. However, in the denervated lung, regional hypercarbia of 10% and/or 20% CO₂ induces regional vasodilation in the presence of 40% or more O₂ in concentration in the exchange gas as if it worked as a potentiator of hyperoxic vasodilation. This hypercarbic vasodilation seems to keep suppressed in the normal lung, while suppression doesn't seem to work in the denervated lung. It is speculated, therefore, that there is an "O₂ sensing device" in the lung periphery which can either constrict or dilate the vascular smooth muscle by sensing changes in O₂ concentration in
the surrounding alveoli. The “O₂ sensing device” has nothing to do with a higher center such as the central nervous system. However, in the normal lung, a peripherally located “CO₂ sensing device” is under control of a higher center which inhibits the vasodilating impulse from the “CO₂ sensing device” to the vascular smooth muscle when exposed to hypercarbia in the presence of hyperoxia. In the denervated lung, this inhibition cannot work and hypercarbic vasodilation takes place.

**An Analysis of Cortical Renal Blood Flow by Means of Early Image on 99mTc-DMSA Renal Scintigram**

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It is well known that 99mTc-DMSA renal scintigram can provide a good qualified static image due to the preferential cortical accumulation of DMSA. The early dynamic image of DMSA renal scintigram reveals an initial cortical perfusion, reflecting a nuclear angiography with Tc compounds. Renal counting was initiated immediately after the administration of 2mCi of DMSA and each count was recorded in the data store play back system which was connected with the minicomputer. After ROI was set with a subtraction of renal background, renal counts were traced during 90 sec. to obtain a curve of DMSA renogram from each kidney. Then, four parameters were extracted from this curve as follows: i) $T_{max}$ of maximum peak time on this curve, ii) $C_{max}$ of counts at $T_{max}$, iii) $\tan \theta$ of an angle of the upslope part of the curve ($C_{max}/T_{max}$) and iv) $T_{plateau}$ ($T_{pl}$) of the time when a constant count was obtained on the curve after passing a peak. In this study, those four parameters were investigated in the variety of renal diseases and the implications were discussed.

In the case of renal cell carcinoma with neovascularity of the cortex, $T_{max}$ and $T_{pl}$ were prolonged, $C_{max}$ increased and $\tan \theta$ was normal. In the case of hypertensive kidneys, while $T_{max}$ and $T_{pl}$ were prolonged, $C_{max}$ was within normal limits. However, in the case of nephrosclerosis, $C_{max}$ and $\tan \theta$ decreased associated with prolonged $T_{max}$ and $T_{pl}$.

In the case of mild hydronephrosis, while $C_{max}$ and $T_{max}$ decreased, $T_{pl}$ increased and $\tan \theta$ was within normal limits. On the extension of the severity of hydronephrosis, $T_{pl}$ and $\tan \theta$ were prolonged or decreased. In the cases of glomerulonephritis and pyelonephritis with moderate lesions, while $T_{max}$ and $C_{max}$ decreased, $T_{pl}$ and $\tan \theta$ were still within normal limits. In the advanced cases of such diseases and in the case of diabetic nephropathy, all parameters were decreased.

In the cases of solitary renal cyst and congenital small kidney, while $C_{max}$ decreased, other parameters were within normal limits.

From this study, $T_{max}$ indicates initial cortical blood flow rate, $C_{max}$ initial cortical distribution volume, $T_{pl}$ effective perfusion rate and $\tan \theta$ effective perfusion area. While abnormalities in $T_{max}$ and $T_{pl}$ reveal pathologic changes in the cortical vascularity, those in $C_{max}$ and $\tan \theta$ indicate cortical parenchymal pathologies. Particularly, $\tan \theta$ may indicate a functional balance between cortical perfusion rate and area.

In conclusion, various combinations of the four parameters can allow to classify renal cortical diseases on the basis of renal hemodynamics.