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 (O_2) and carbon dioxide (CO_2) 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 N_2, 10% O_2 in N_2, air, 40% O_2 in N_2, 60% O_2 in N_2 and 100% O_2 for studying the effect of O_2 concentration and 2%, 5% and 7% CO_2 in air and 10% CO_2 in N_2, in air, in 40% O_2 in N_2, in 60% O_2 in N_2, in 100% O_2 and 20% CO_2 in 60% O_2 in N_2 for studying the effect of CO_2 concentration on regional perfusion distribution in the lungs. At the end of the 7th gas exchange of the RUL, ^{99m}Tc-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 N_2 or 10% O_2 in N_2, while they increased in a dose-response fashion as the O_2 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 O_2 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 CO_2 of less than 7% in concentration. When the normal RUL was artificially ventilated with 10% or 20% CO_2 mixed in the above mentioned gases of different O_2 concentrations, perfusion ratios in the RUL were similar to those observed when the lobe was ventilated with gas mixtures of different O_2 concentrations without 10% or 20% CO_2. However, when the RUL of the denervated right lung was artificially ventilated with 10% or 20% CO_2 in gases with 40% or more O_2 in concentration, perfusion increased significantly as compared with those observed with gases with 40% or more O_2 in concentration without 10% or 20% CO_2, indicating the presence of hypercarbic vasodilation, while addition of 10% CO_2 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 O_2 concentration. However, in the denervated lung, regional hypercarbia of 10% and/or 20% CO_2 induces regional vasodilation in the presence of 40% or more O_2 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 "O_2 sensing device" in the lung periphery which can either constrict or dilate the vascular smooth muscle by sensing changes in O_2 concentration in

the surrounding alveoli. The "O_2 sensing device" has nothing to do with a higher center such as the central nervous system. However, in the normal lung, a peripherally located "CO_2 sensing device" is under control of a higher center which inhibits the vasodilating impulse from the "CO_2 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 ^{99m}Tc-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 back ground, 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 Tmax, iii) tan θ of an angle of the upslope part of the curve (C_{max}/T_{max}) and iv) Tplateau (Tpl) 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 Tpl were prolonged, Cmax increased and tan θ was normal. In the case of hypertensive kidneys, while T_{max} and Tpl were prolonged, C_{max} was within normal limits. However, in the case of nephrosclerosis, C_{max} and tan θ decreased associated with prolonged T_{max} and Tpl.

In the case of mild hydronephrosis, while C_{max} and T_{max} decreased, Tpl increased and tan θ was within normal limits. On the extension of the severity of hydronephrosis, Tpl and tan θ were prolonged or decreased. In the cases of glomerulonephritis and pyelonephritis with moderate lesions, while T_{max} and C_{max} decreased, Tpl and tan θ 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, Tpl effective perfusion rate and tan θ effective perfusion area. While abnormalities in Tmax and Tpl reveal pathologic changes in the cortical vascularity, those in C_{max} and tan θ indicate cortical parenchymal pathologies. Particularly, tan θ 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.