Title: EFFECTS OF HYPOCAPNIA AND HYPERCAPNIA ON LIVER SURFACE PO2 IN THE DOG

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Introduction: In clinical practice, mild to moderate degrees of hypocapnia are commonly seen in patients being artificially ventilated. Although the effect of hypocapnia on the cerebral circulation have been well documented, little data available in other tissues. In the present study, we investigated the effects of hypocapnia and hypercapnia on the liver surface oxygenation in the dog.

Methods. Nine mongrel dogs, weighing 11-38 kg, were anesthetized with sodium pentobarbital (30 mg/kg iv), intubated and ventilated mechanically with pure oxygen to maintain normocapnia. Femoral arterial and pulmonary arterial thermodilution catheter were inserted via a femoral cut down. The body temperature was maintained at 38°C throughout the experiment. End-tidal CO2 fraction was monitored continuously by capnograph. After laparotomy, a miniature Clark-type polarographic oxygen electrode sensor (Biomedical Sensors) was prepared and placed on the liver surface.

After a normocapnic period, the hypocapnia was induced by increasing mechanical ventilation, and fixed to hyperventilation throughout the experiment. Hypercapnia by adding carbon dioxide to the inspired gas stepwisely until the end-tidal CO2 fraction ($F_E'CO2$) reached 10 %. The following variables were measured and calculated: mean arterial pressure(MAP), cardiac output(C.O.), arterial PO2(PaO2), arterial PCO2(PaCO2), and liver surface PO2(PsO2).

The data were analyzed using the paired-t test accepting $p<0.05$ as significant.

Results. When hyperventilation was performed, PaCO2 decreased from 39 mmHg to 22 mmHg, and liver PsO2 decreased significantly ($p<0.05$) from 163 mmHg to 86 mmHg. After the inhalation of carbon dioxide, PaCO2 increased from 22 mmHg to 69 mmHg, and liver PsO2 also increased from 86 mmHg to 198 mmHg. Neither PaO2 or cardiac output showed any significant change during the experiment.

Discussion. The changes in PaCO2 (hypocapnia vs hypercapnia) were made with minimum effects of mechanical ventilation. Liver surface PsO2 correlated well with the changes in PaCO2. Hypocapnia induced by hyperventilation markedly reduced liver surface oxygen tension despite the unchanged cardiac output. Our result indicate that severe and prolonged hyperventilation may induce impaired liver oxygenation.

Figure:

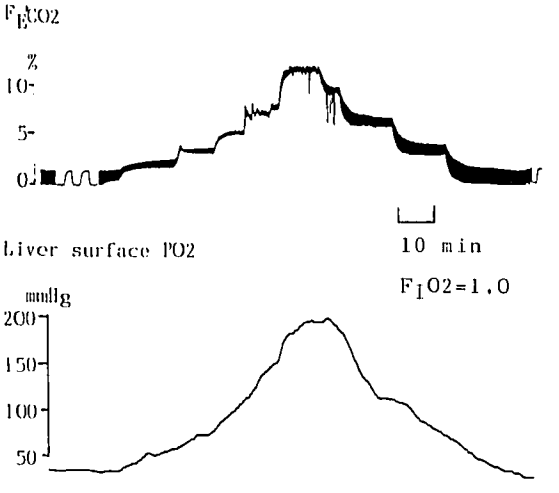

TABLE:

	Normoventilation	Hyperventilation @	CO2 inhalation	
$F_E'CO2$	5%	2%	6%	10%
MAP mmHg	140 ±13	150 ±9	160 ±9	146 ±7
C.O. l/min	2.36 ±0.31	2.11 ±0.28	2.37 ±0.35	2.23 ±0.28
pH	7.30 ±0.02	7.45 ±0.03	7.29* ±0.04	7.13* ±0.03
PaO2 mmHg	531 ±25	553 ±26	500 ±52	535 ±17
PaCO2 mmHg	39 ±2	22 ±1	48* ±5	69* ±7
Liver PsO2 mmHg	163 ±29	86 ±20	123* ±16	198* ±29

Mean ±SEM, * $p<0.05$ from @(2%$F_E'CO2$)