POSTER SESSION III—CIRCULATION

A133

Title: EFFECTS OF CARBON DIOXIDE (HYPOCAPNIA AND HYPERCAPNIA) ON TISSUE BLOOD FLOW AND OXYGENATION OF LIVER, KIDNEY AND SKELETAL MUSCLE IN THE DOG.

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Introduction. With the exception of cerebral and coronary circulations, little data is available regarding the effects of carbon dioxide on vital organ perfusion and oxygenation. We investigated the effects of carbon dioxide on the splanchic visceral organs (liver and kidney) as well as skeletal muscle in the anesthetized dogs.

Methods. Thirty two adult mongrel dogs, weighing 13.3 kg, were anesthetized with sodium pentobarbital (30mg/kg, iv), intubated and ventilated mechanically with 100% oxygen to maintain normocapnia. End-tidal CO2 fraction (FECO2) was monitored continuously by capnograph. Femoral arterial catheter and pulmonary artery thermodyiation catheter were inserted via right femoral cut down. After laparotomy, miniature Clark-type polarographic oxygen electrodes (Biomedical Sensors) were placed on the surfaces of liver, kidney, and the rectus femoris muscle. Electromagnetic blood flow probes (Mihon-koden) were also applied to hepatic artery (HA), portal vein (PV), and femoral artery (FA). After a stable normocapnic ventilation, the hypcapnia was produced by increasing respiratory rate, and this mechanical hyperventilation was kept fixed throughout the ensuing experiments. In order to induce hypercapnia exogenous carbon dioxide was added to the inspired gas stepwisely until FECO2 reached 10%. In each steps, the following variables were measured: cardiac output (CO), arterial PO2 (PaO2), arterial PCO2 (PaCO2), tissue surface PO2 (liver PaO2, kidney PsO2, muscle PsO2), and blood flows (HABF, PVBF, PABF). The data were analyzed using the paired t-test accepting the p<0.05 as significant.

Results. Table 1. and Figure 1. summarize the results. Hyperventilation resulted in a significant decrease in HABF, PVBF, liver PaO2 and kidney PsO2 in parallel with the decreased PaCO2, but these parameters increased dose dependently when the carbon dioxide was added to the inspired gas (hypercapnic hyperventilation). On the contrary, splanchic and skeletal muscle PsO2 increased by hypcapnia and decreased during hypercapnia. Neither PaO2 or CO showed any significant change during entire experiment.

Discussion. Arterial CO2 tension appears to exert significant effects on both splanchic and skeletal muscle perfusion as well as corresponding changes in tissue oxygenations. It would seem that as arterial CO2 tension rises a significant redistribution of blood flow occurs favoring the vital organs at the expense of muscle perfusion. While the hypcapnic state is associated with an increasing muscle bed perfusion with lowered splanchic blood flow and oxygenation. It is possible that injudicious and prolonged hypocapnic hyperventilation may seriously compromise splanchic organ perfusion and oxygenation.