## [1471.199] NIRS Technology Detects a Decrease in Intestinal Blood Flow Caused by Hypovolemia

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BACKGROUND: A non-invasive method for monitoring changes in tissue perfusion might improve care for ELBW infants. Tissue  $O_2$  consumption = K x  $[SaO_2 - SvO_2]$  x BF, where K is the amount of  $O_2$  bound to hemoglobin, BF is tissue blood flow, and  $[SaO_2 - SvO_2]$  is the oxygen saturation gradient between arterial and venous blood. If oxygen consumption is constant, a decrease in BF will cause an increase in  $[SaO_2 - SvO_2]$ . SaO<sub>2</sub> can be monitored by pulse oximetry. Since tissue  $O_2$  saturation (NIRS-StO<sub>2</sub>) measured by NIRS technology reflects a mixture of SaO<sub>2</sub> and SvO<sub>2</sub>, SvO<sub>2</sub> can be calculated from SaO<sub>2</sub> and NIRS-StO<sub>2</sub>. OBJECTIVE: To determine the effect of hypovolemia (**HV**) on portal venous blood flow (PVBF), SaO<sub>2</sub>, SpvO<sub>2</sub> (portal venous) and intestinal NIRS-StO<sub>2</sub>.

DESIGN/METHODS: PVBF was measured using the dye dilution method in rats with aortic, portal venous (PV) and superior mesenteric artery (SMA) catheters. Evan's blue dye (EBD) was infused into the SMA. Aortic and PV blood were analyzed for EBD. PVBF = Infusion rate of EBD / EBD (PV-A). These studies were repeated under **HV** (removal of 20% of blood volume). In a second group of rats, a CASMED NIRS probe was placed on the abdomen. While monitoring NIRS-StO<sub>2</sub>, aortic and PV blood were analyzed for SO<sub>2</sub> using co-oximetry. These studies were repeated under **HV**. Calculations: assuming that intestinal O<sub>2</sub> consumption was not affected by **HV**, PVBF<sub>cont</sub> x [SaO<sub>2</sub>-SpvO<sub>2</sub>]<sub>cont</sub> = PVBF<sub>HV</sub> x [SaO2-SpvO2]<sub>HV</sub>. PVBF<sub>cont</sub> / PVBF<sub>HV</sub> = [SaO<sub>2</sub>-SpvO<sub>2</sub>]<sub>HV</sub> / [SaO<sub>2</sub>-SpvO<sub>2</sub>]<sub>cont</sub>. Preliminary studies indicate that intestinal NIRS-StO<sub>2</sub> reflects 57% portal venous and 43% arterial blood. Using this relationship, **calculated SpvO**<sub>2</sub> (**CSpvO**<sub>2</sub>) = (NIRS-StO<sub>2</sub> - 0.43 x SaO<sub>2</sub>) / 0.57.

RESULTS: The PVBF<sub>cont</sub> / PVBF<sub>HV</sub> measured by dye dilution was (**2.1**(0.5) n=3). Values = mean(SEM)

	SaO2	SpvO2	CSpvO2	SaO2-SpvO2	SaO2-CSpvO2
Control	91(1)	67(5)	63(6)	24(4)	28(5)
Hypovolemia	93(1)	36(5)	40(4)	57(6)	53(4)

## The effect of hypovolemia on SO2 (percent)

values = mean (SEM), n=-4

Using changes in  $[SaO_2-SpvO_2]$  or  $[SaO_2-CSpvO_2]$ , the PVBF<sub>cont</sub> / PVBF<sub>HV</sub> was (**2.5**(0.3) n=4) and (**2.1** (0.4) n=4), respectively.

CONCLUSIONS: **HV** decreased PVBF by 50% determined by both the dye dilution and  $[SaO_2-SpvO_2]$  method. The NIRS **CSpvO**<sub>2</sub> was similar to measured SpvO<sub>2</sub>. This is the first direct evidence that changes in  $[SaO_2-CSpvO_2]$  are indirectly proportional to intestinal blood flow. We speculate that continuous monitoring of  $[SaO_2-CSpvO_2]$  by pulse oximetry and NIRS technology can be used to detect clinically significant changes in tissue perfusion. Funded by: CASMED

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