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Oxygen and CO₂ content in the splanchnic and nonsplanchnic blood of dogs with portacaval transposition

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It has often been stated that the oxygen content of the portal venous blood is higher than that found in the inferior vena cava. In the present studies this concept has been tested, and found to be invalid in unanesthetized, resting dogs subjected to prior portacaval transposition.

METHODS

Thirteen mongrel dogs, weighing 10 to 20 kilograms each, received portacaval transposition³ under total body hypothermia.⁷ During convalescence, they were trained to lie quietly in a lateral position. From 11 to 106 days postoperatively, the dogs were fasted at intervals of 12 to 18 hours, and catheters were inserted under local anesthesia⁸ into the suprarenal inferior vena cava, the proximal portal vein, the left hepatic vein, and a peripheral artery. The position of the venous catheters was fluoroscopically controlled. After a resting state was achieved, time-integrated blood samples were simultaneously drawn, and subsequently analyzed by the manometric Van Slyke method for oxygen content. Nine of the dogs also had Van Slyke CO₂ determinations performed. Hematocrits were routinely done. At autopsy, the patency of the venous anastomoses was proved.

RESULTS

The results of the gas analyses are shown in Table I. Although there was some variation in results, the over-all pattern was consistent. The arterial oxygen concentration was greatest, as would be expected. The oxygen concentration in the hepatic venous blood was lowest. The oxygen saturation of the suprarenal vena cava was higher than that of the portal vein in 12 of 13 animals. The only exception was in Dog 10, who had a significant hemorrhage at the time of arterial catheterization. The difference between the vena caval blood and the portal venous blood was significant ($p < 0.005$). It was also noted that in all but two of the animals, the CO₂ content of the portal blood was either equal to or higher than that in the vena caval samples.

DISCUSSION

There seems little doubt that in patients with cirrhosis of the liver and portal hypertension the oxygen content is higher in the splanchnic veins than in the systemic veins.^{1, 5, 6} Most authors have suggested that this is due to arteriovenous shunting within the nonhepatic splanchnic system.

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The origin of the belief that the same situation exists in the healthy dog is less clear. The classical work of Blalock and Mason² has often been quoted in support of this idea, but in fact, these authors did not measure, and did not claim to have measured the oxygen concentration of the vena caval system. Instead, they studied the oxygen gradient across the liver. The vena caval samples analyzed by them were from a temporarily isolated segment into which only the hepatic veins drained.

Only two pertinent studies about dogs are available, those of Womack and Peters⁹ and Hardin, Shumaker, and Sheng Su.⁴ The former investigations were carried out under anesthesia, and were part of a protocol which involved considerable manipulation of the portal structures, as well as intermittent occlusion of the portal vein. Under these circumstances, the oxygen concentration in the splanchnic blood was consistently higher than that in the inferior vena cava. Similarly, the dogs studied by Hardin and associates⁴ were examined one day after operative placement of catheters. The portal venous concentration averaged 76 percent, as compared to 49 percent in the femoral vein. The unusually desaturated state of the latter samples raises some doubt about the physiologic normalcy of the animals at the time of measurement. In addition, the site of peripheral sampling does not necessarily reflect the oxygen content in the suprarenal inferior vena cava.

Under the conditions of our study, the blood in the portal vein actually was more desaturated than that in the portion of the inferior vena cava, which had received venous effluent from the hindquarters, lower trunk, and kidneys. Thus, a liver revascularized by means of portacaval transposition has a resting venous inflow of at least as high oxygen saturation, and probably higher than that of the diverted distal portal vein. These findings stress the necessity of repeating earlier studies of the portal and systemic oxygen saturation in unaltered dogs under more satisfactory conditions of testing than those employed in the past. It is possible that portacaval transposition alters the physiology of oxygen transport in the splanchnic bed, thus accounting for the results herein reported. Alternatively, however, it is conceivable that a lower systemic oxygen content as opposed to the portal oxygen content would not exist in normal animals, providing they were tested after full recovery from operation.

SUMMARY

The oxygen and CO₂ content of unanesthetized fasting dogs was studied from 2 to 14 weeks after portacaval transposition. The splanchnic venous oxygen content was generally somewhat less than the oxygen content in the suprarenal inferior vena cava. The CO₂ content tended to be greater. These findings challenge the validity of the common assumption that portal venous blood has a higher oxygen content than that of mixed systemic venous blood obtained from the inferior vena cava.

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References

1. Berman EJ, Habegger ED. Vascular crisis in atrophic cirrhosis of the liver. *Wisconsin M J* 1952;51:1175. [PubMed: 13015912]
2. Blalock A, Mason MF. Observations on the blood flow and gaseous metabolism of the liver in unanesthetized dogs. *Am J Physiol* 1936;117:328.
3. Child CG III, Barr D, Holswade GR, Harrison CS. Liver regeneration following portacaval transposition in dogs. *Ann Surg* 1953;138:600. [PubMed: 13092790]

4. Hardin RA, Shumaker HB Jr, Sheng Su C. Studies on portal venous oxygen saturation. Arch Surg 1963;87:831. [PubMed: 14058849]
5. Liebowitz, HR. Bleeding esophageal varices: Portal hypertension. Springfield, Ill: Charles C Thomas, Publisher; 1959. p. 321-323.
6. Smythe CN, Fitzpatrick HF, Blakemore AH. Studies of portal venous oxygen content in unanesthetized man. J Clin Invest 1951;39:674.
7. Starzl TE, Butz GW Jr, Munger DH. A technique for portacaval transposition (addendum). J Surg Res 1961;1:218.
8. Starzl TE, Lazarus RE, Schlachter L, Thornton FH, Wendel RM, Stearn B, Scanlon WA. A multiple catheter technique for studies of hepatic metabolism and blood flow in dogs with portacaval transposition. Surgery 1962;52:654. [PubMed: 13916396]
9. Womack NA, Peters RM. An investigation of the relationship between portal venous pressure and inferior vena caval and portal venous oxygen saturations. Ann Surg 1957;146:691. [PubMed: 13470759]

Table I

Oxygen and carbon dioxide in volumes percent

Dog No.	Day postop.	Hematocrit (%)	Arterial		Portal vein		Vena cava		Hepatic vein	
			CO ₂	O ₂	CO ₂	O ₂	CO ₂	O ₂	CO ₂	O ₂
1	106	41	—	14.62	—	10.15	—	11.76	—	7.10
2	125	44	32.84	16.74	40.75	8.29	34.52	13.16	37.87	9.98
3	99	33	—	—	42.26	9.48	45.20	8.64	43.95	5.95
4	96	36	33.88	12.39	37.36	10.15	36.13	10.48	37.37	7.00
5*	14	28	24.63	14.61	25.54	12.20	28.34	10.47	29.99	7.14
6	14	41	28.68	15.25	34.15	9.41	31.62	11.90	32.97	8.95
7	14	45	33.93	16.21	36.24	11.97	36.31	14.09	36.98	11.67
8	14	35	31.87	13.97	36.71	7.96	33.86	11.83	35.15	8.26
9	15	44	33.89	16.73	41.08	13.44	36.07	15.58	35.79	12.25
10	11	45	34.76	14.82	40.18	8.48	38.20	12.20	38.66	8.76
11	12	46	29.06	17.16	33.30	13.97	32.40	16.20	34.00	13.70
12	12	45	30.50	15.57	35.44	9.73	34.66	14.25	34.89	11.62
13	12	44	34.00	15.89	38.63	10.28	37.09	12.70	36.96	10.89
Average		40.5	31.64	15.33	36.80	10.42	35.36	12.55	36.21	9.48
Standard deviation		5.7	3.1	1.4	4.5	1.9	4.1	2.1	3.4	2.4

* Significant hemorrhage during vessel catheterization.