Reports and Letters

Splanchnic Blood Flow after Hemorrhage

It is commonly taught (*1*) that circulatory adjustments after hemorrhage involve splanchnic and cutaneous vasoconstriction with a consequent diversion of the blood flow to those portions of the body that are involved in the somatic response to crisis. Evidence to the contrary (*2*) has been little noted. So far as the splanchnic area is concerned, small hemorrhages (10 ml/kg) have been found to exert only a minor effect on the blood flow (*3*). The cardiac output, however, likewise showed only small change. It has been stated that larger hemorrhages cause transient reductions in the hepatic blood flow, but no measurements of cardiac output were reported (*4*), so that the extent of circulatory embarrassment with these hemorrhages cannot be assessed.

We have found that hemorrhages of 25 ml/kg in the dog anesthetized with sodium pentobarbital regularly result in a diminution of the cardiac output to 60 percent or less of its control value. The effect of such hemorrhages on the splanchnic blood flow was therefore investigated (*5*).

After induction of anesthesia with sodium pentobarbital (30 mg/kg intravenously) and placement of a hepatic venous catheter, the cardiac output was measured by the dye dilution technique (*6*) employing 10 mg Rose Bengal (*7*). Immediately after the sampling of blood for the output measurement, an infusion of Rose Bengal containing 2.0 mg/ml in saline was begun at 0.5 ml/min. Samples of femoral arterial and hepatic venous blood were taken at 10, 15, and 20 minutes after the beginning of the infusion; the precautions outlined elsewhere were carefully observed in the hepatic venous sampling (*8*). The splanchnic blood flow was calculated from the dye clearance (infusion rate/equilibrium concentration in the blood) divided by the arterial-hepatic venous extraction ratio. Concentration equilibrium was attained in all experiments reported.

The animal was then bled through the jugular vein, using a No. 14 needle until 25 ml/kg of blood had been removed. The hemorrhage ordinarily required 10 to 15 minutes. Preliminary heparinization of the animal (20 mg/intravenously) facilitated the bleeding. All animals showed manifestations of sympathetic activity and some degree of air hunger, but all survived the hemorrhage.

One hour after the completion of the hemorrhage, the cardiac output and splanchnic blood flow were again determined by the methods given here. The results obtained in a series of six animals are presented in Table 1.

The cardiac output of these animals before hemorrhage ranged from 133 to 240 ml/kg per minute, with an average of 190. The splanchnic blood flow ranged from 16 to 51 ml/kg per minute with an average of 32. Both the averages and the variability are consistent with our previous experience and that of others (*7–9*). After hemorrhage the cardiac output ranged from 39 to 115 ml/kg per minute with an average of 77. The splanchnic blood flow after hemorrhage ranged from 14 to 37 ml/kg per minute with an average of 24. The estimated splanchnic blood flow (EHBF) showed an absolute rise in four dogs and a fall in two, but in only one case (dog 2) did it show a fall comparable to that in the cardiac output. The splanchnic fraction of the cardiac output, originally averaging 0.17 rose to 0.34. In dog 4 this value was 0.77, indicating that more than three-fourths of the cardiac output perfused the splanchnic bed.

It would appear from these experiments that failing circulation supports the splanchnic bed at the expense of the remainder of the body rather than the converse. This must not be construed to indicate that the splanchnic *reservoir* has not discharged. It is quite clear that a portion of the body may discharge its contained blood while maintaining its perfusion rate.

It remains to be seen whether these findings represent an unusual response of the anesthetized animal. In the absence of experimental evidence to the contrary in the conscious animal, it may be suggested that the usual teaching that the splanchnic blood flow is disproportionately reduced in circulatory embarrassment must be subjected to critical reevaluation.

LEO A. SAPIRSTEIN
NANCY M. BUCKLEY
ERIC OGDEN
Department of Physiology, Ohio State University, Columbus

Table 1. Cardiac output and splanchnic blood flow in anesthetized dogs before and after hemorrhage.

Dog	Wt. (kg)	Cardiac output (lit/min)	Extraction ratio	Blood clearance (ml/min)	EHBF (ml/min)	EHBF/cardiac output
			Before hemorrhage			
1	14.8	2.6	0.41	98	234	0.09
2	17.3	3.5	0.16	131	833	0.24
3	20.0	4.8	0.37	375	1028	0.21
4	12.8	1.7	0.26	80	314	0.19
5	14.8	3.0	0.39	133	341	0.12
6	16.9		0.32	159	502	
			After hemorrhage			
1	14.8	1.1	0.40	97	247	0.22
2	17.3	1.3	0.38	105	239	0.18
3	20.0	1.4	0.37	129	391	0.28
4	12.8	0.5	0.18	67	382	0.77
5	14.8	1.7	0.36	129	424	0.25
6	16.9		0.27	152	627	

References and Notes

1. C. H. Best and N. B. Taylor, *Physiological Basis of Medical Practice* (Williams and Wilkins, Baltimore, ed. 6, 1955), p. 302; B. A. Houssay *et al.*, *Human Physiology* (McGraw-Hill, New York, ed. 2, 1955), p. 55.
2. A. Blalock and S. Levy, *Am. J. Physiol.* **118**, 734 (1937).
3. A. Y. Werner, D. M. MacCanon, S. M. Horvath, *ibid.* 170, 624 (1952).
4. H. O. Heinemann, C. M. Smythe, P. A. Marks, *ibid.* 174, 352 (1953).
5. Supported by the Central Ohio Heart Association and the American Heart Association. A portion of this work was performed as part of a contract between the United States Air Force School of Aviation Medicine, Randolph Field, Tex., and the Ohio State University Research Foundation.
6. J. W. Moore *et al.*, *Am. J. Physiol.* **89**, 331 (1929).
7. D. G. Vidt *et al.*, *ibid.* 181, 337 (1955).
8. L. A. Sapirstein and A. M. Simpson, *ibid.* **182**, 337 (1955).
9. W. G. B. Casselman and A. M. Rappoport, *J. Physiol. London* **124**, 173 (1954).

16 July 1955