lateralis; certainly in this viviparous fish it would be reasonable to assume that the function of a fetal hemoglobin would be the same as that accepted for the fetal hemoglobins of mammals (*9*).

CLYDE MANWELL*
Department of Biological Sciences, Stanford University, Stanford, California, and *Marine Field Laboratories of the University of Washington, Friday Harbor*

References and Notes

1. F. McCutcheon, *J. Cellular Comp. Physiol.* 8, 63 (1936); A. Riggs, *J. Gen. Physiol.* 35, 23 (1951).
2. C. P. O'Connell, *Calif. Fisheries Bull. No. 93* (1953), p. 1.
3. This research was performed on the tenure of a National Science Foundation predoctoral fellowship. The assistance of J. P. Baumberger (Stanford University) and of A. W. Martin, Paul Sund, and other members of the Marine Field Laboratories of the University of Washington is gratefully acknowledged.
4. R. Brinkman and J. H. P. Jonxis, *J. Physiol. (London)* 88, 162 (1936).
5. K. Betke, *Der Menschliche Rote Blutfarbstoff bei Fetus und Reifem Organismus* (Springer, Berlin, 1954).
6. C. F. Pantin, *Notes on Microscopical Technique for Zoologists* (Cambridge Univ. Press, Cambridge, 1948).
7. The method of Brinkman and Jonxis, as employed by J. H. P. Jonxis and T. H. J. Huisman [*Blood* 11, 1009 (1956)], was followed.
8. H. S. Baar and E. M. Hickmans, *J. Physiol. (London)* 100, 3P (1941); A. Rossi-Fanelli, G. F. Azzone, B. Mondovi, *Arch. Biochem. and Biophys.* 58, 119 (1955).
9. J. Barcroft, *Researches on Pre-natal Life* (Thomas, Springfield, Ill., 1947), vol. 1.
10. F. G. Hall, *J. Physiol. (London)* 83, 222 (1935).
11. F. McCutcheon, *J. Cellular Comp. Physiol.* 29, 333 (1947).
12. A. C. Redfield, *Quart. Rev. Biol.* 8, 31 (1933).

* Present address: Marine Biology, Scripps Institution of Oceanography, La Jolla, Calif.

19 August 1957

Effect of Digestion on Distribution of Blood Flow in the Rat

The belief that splanchnic blood flow increases at the expense of flow in other organs during digestion was challenged by Herrick *et al.* (*1*) in 1934. By use of thermostromuhrs, dogs were found to show similar increases in carotid, femoral, and superior mesenteric arterial blood flow postprandially. Abramson and Fierst (*2*) found that the blood flow to the human hand, forearm, and leg tended to increase after eating. A meal increases the splanchnic blood flow of a human subject (*3*). However, the cardiac output also increases (*4*). When the experimental values are adjusted for surface area, it is found that the absolute postprandial increase in splanchnic blood flow in a 1.73 m^2 man is about 710 ml/min. A 24 percent increase in the cardiac output of such a man (*5*) represents 1300 to 1400 ml/min. From this it may be concluded that the nonsplanchnic blood flow is increased by digestion.

The subject has been reinvestigated (*6*) with the aid of a newly developed method (*7*). The method is based on the observation that all organs other than the brain have, during the first minute after a single intravenous injection of $K^{42}Cl$, substantially the same extraction ratios for K^{42}. The fractional distribution of K^{42} among the organs during the first minute therefore corresponds to the fractional distribution of the cardiac output. The anomalous behavior of the brain has been shown to be of minor consequence in the measurements of the blood flow to other organs. Values obtained by this method describe the fractions of the cardiac output directed to each organ. A knowledge of the cardiac output permits the calculation of the blood flow to each organ.

One hundred and seventeen rats were used. Control animals were starved for 24 to 72 hours but were permitted to drink water *ad libitum*. "Fed" animals were allowed to eat and drink *ad libitum* up to the time of the experiment. The gastrointestinal tract of the "fed" animals always contained 10 to 15 g of food at autopsy. The animals were anesthetized with Nembutal (40 mg/kg intraperitoneally). The cardiac output was determined by dye dilution, with Evans blue as the indicator; the blood was sampled at a rate of 90 collections per minute (*8*). Other similarly treated animals of the same stock were used for the fractional distribution studies with K^{42}; the details of the method have been described previously (*7*).

The cardiac output of 17 control animals averaged 172 ± 38 ml/kg min. Eleven fed animals had a cardiac output of 223 ± 59 ml/kg min. Determinations of fractional distribution were made on 49 control and 40 fed animals. The fractions found for each organ were multiplied by the cardiac output value in animals of the same group (adjusted for body weight) to give blood flow values to the various organs.

Table 1 shows the blood flow values obtained in the organs of the two groups. For simplicity all values have been adjusted for the body weight and are presented as the blood flow to the organs of a 250-g rat.

It is clear from these results that, during digestion, there is a uniform increase in the blood flow to all organs of the rat. The splanchnic organs do not gain their increased blood supply at the expense of the blood supply to other organs; on the contrary, all organs benefit from the increased cardiac output associated with digestion.

These results, though obtained in anesthetized rats, are similar to those reported in conscious dogs and men; they do not conflict with any reported findings. In the absence of contrary evidence, it is suggested that the prevailing concept that digestion results in diversion of blood flow from other organs to the digestive tract be critically re-examined.

Table 1. Blood flow values in fasting and fed rats (all values have been adjusted to 250-g rats; blood flow is given in milliliters per minute).

Organ	Blood flow	
	Fasted	Fed
Liver (arterial)	3.2	4.3
Gut and spleen	7.1	9.4
Myocardium	1.1	1.3
Skin	3.2	4.2
Kidneys	6.6	8.7
Carcass	21.8	27.9

EDWARD J. REININGER
LEO A. SAPIRSTEIN
Department of Physiology, Ohio State University, Columbus

References and Notes

1. J. F. Herrick *et al.*, *Am. J. Physiol.* 108, 621 (1934).
2. D. I. Abramson and S. M. Fierst, *ibid.* 133, 686 (1941).
3. J. L. Brandt *et al.*, *J. Clin. Invest.* 34, 1017 (1955).
4. R. M. Paine and N. W. Shock, *Circulation* 1, 1026 (1950); B. Berman, J. R. Braunstein, J. McQuire, *ibid.* 1, 1017 (1950); A. Grollman, *Am. J. Physiol.* 89, 366 (1929).
5. This figure is based on a 3.12-lit cardiac index. The splanchnic blood flow values are taken from Brandt *et al.* (*3*). The 24 percent increase in cardiac output after eating is the mean of values previously reported (*4*).
6. This work was supported by a grant-in-aid from the Central Ohio Heart Association and the American Heart Association. A portion of the work was supported by a contract between the U.S. Air Force School of Aviation Medicine, Randolph Field, Tex. and the Ohio State University Research Foundation. The assistance of Francesco Arcidiacono is gratefully acknowledged.
7. L. A. Sapirstein, *Circulation Research* 4, 689 (1956); *Am. J. Physiol.*, in press.
8. E. J. Reininger, Ph.D. dissertation, Ohio State University, 1957. The technique used was slightly modified from that originally described by R. Bullard [*Federation Proc.* 15, 28 (1956)].

19 August 1957

Activation of Enzymatic Hydrolysis of Benzoylcholine by Tryptamine

During an investigation of the anticholinesterase activity of indole derivatives (*1*) it was found that tryptamine accelerates the enzymatic hydrolysis of benzoylcholine by plasma cholinesterase (*2*). It has also been reported that analgesics (*3*) and other compounds (*4*) activate plasma cholinesterase, and in certain cases, red cell cholinesterase (*5*). Some authors attributed this activation to an interference wth the partial inhibition of the enzyme (E) by the excess