ment for multistage amplification, capable of drawing a large return very rapidly from a minimal input, seems so applicable to the general problem of excitation, whether in receptors, nerve, muscle, or indeed eggs, that it would seem worthwhile to look for it in all these structures.

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References and Notes

1. S. Hecht, S. Shlaer, M. H. Pirenne, *J. Gen. Physiol.* **25**, 819 (1941–42).
2. G. Wald, P. K. Brown, I. R. Gibbons, *J. Opt. Soc. Amer.* **53**, 20 (1963).
3. J. H. Webb, *ibid.* **29**, 309 (1939).
4. G. Wald, in *Conference on Nerve Impulse*, 4th, D. Nachmansohn, Ed. (Macy, New York, 1954), pp. 48–49.
5. G. Wald, in *Enzymes: Units of Biological Structure and Function*, O. Gaebler, Ed. (Academic Press, New York, 1956), p. 355.
6. R. G. Macfarlane, *Nature* **202**, 498 (1964); compare E. W. Davie and O. D. Ratnoff, *Science* **145**, 1310 (1964).
7. L. Lorand, *Biochem. J.* **52**, 200 (1952); K. Laki and J. A. Gladner, *Physiol. Rev.* **44**, 127 (1964).
8. R. A. Fisher, *Statistical Methods for Research Workers* (Oliver and Boyd, London, 1925), pp. 57–65; H. L. Rietz, *Mathematical Statistics* (Open Court, Chicago, Ill., 1927), pp. 39–45.
9. K. Pearson, Ed., *Tables for Statisticians and Biometricians* (Biometric Lab., University College, London, ed. 2, 1965), part 1, p. 113.
10. W. H. Seegers, *Prothrombin* (Harvard Univ. Press, Cambridge, Mass., 1962).
11. D. Mazia and T. Hayashi, *Arch. Biochem. Biophys.* **43**, 424 (1952).

16 August 1965

Blood-Flow Relation between Hepatic Artery and Portal Vein

Abstract. *Blood flows in the hepatic artery and portal vein have been measured with a square-wave, electromagnetic flow meter. Hepatic arterial flow increased when portal venous flow was decreased, but, when hepatic arterial flow was decreased, portal venous flow also decreased. The relation between the two blood systems can be explained as the simple mechanical effect of interposing a slower-flowing stream in the path of a faster-flowing stream.*

The dual blood supply of the liver has prompted much speculation about the relations between the blood flows of the hepatic artery and the portal vein. The two principal current theories are (i) that the hepatic arterial flow and the portal venous flow do not influence each other (*1*) and (ii) that a reciprocal and compensatory relation exists between changes in flow in the two systems (*2*). The development of electromagnetic flow meters has made possible the direct measurement of blood flows in the hepatic artery and portal vein. A definite relation between the two blood supplies has been established under experimental conditions (blood vessels exposed but not cannulated).

Healthy mongrel dogs were anesthetized lightly with sodium pentobarbital (30 mg/kg of body weight), intubated, and placed on a respirator. The arterial blood pressure was monitored during the experiment through a needle in the femoral artery connected to a Sanborn pressure transducer, model 267B. Hepatic arterial and portal venous blood flows were measured with a square-wave electromagnetic flow meter (*3*). The portal venous flow was measured at the point just before the vein branched to enter the liver and the hepatic arterial flow, at a point distal to any gastroduodenal branches.

Attempts to decrease the portal vein flow by direct or partial occlusion were followed by a decrease in the systemic arterial pressure as blood pooled in the intestines. A significant decrease of the blood flow in the portal vein was produced, without causing a decrease in systemic blood pressure, by occluding the superior mesenteric artery. This decrease in portal venous blood flow was associated with an increase in the hepatic arterial blood flow, while release of the occlusion of the superior mesenteric artery resulted in restoration of normal flows in both the portal vein and the hepatic artery.

After these blood-flow measurements had been made, a side-to-side portacaval shunt was formed. The portal venous blood flow between the shunt and the liver was measured with the shunt first open and then closed. The hepatic arterial blood flow was also measured with the shunt open and closed. The remarkable decrease in the blood flow through the portal vein was again accompanied by an increase of the flow in the hepatic artery. Closing the shunt restored the portal flow through the liver and decreased the hepatic artery flow to its previous levels. The relation between the blood flows in the portal vein and hepatic artery is shown in Fig. 1. The reduction in portal venous blood flow is compared to the increase in hepatic arterial flow, and the amount of this increase appears to be directly related to the amount of decrease in portal venous flow.

Occlusion of the hepatic artery, which decreases the arterial flow to the liver, is associated not with an increase but with a decrease in the portal venous flow. Figure 2 shows the effect on the blood flow in the portal vein of a decrease in the hepatic arterial flow. A decrease in the arterial flow was accompanied by decreased portal venous flow.

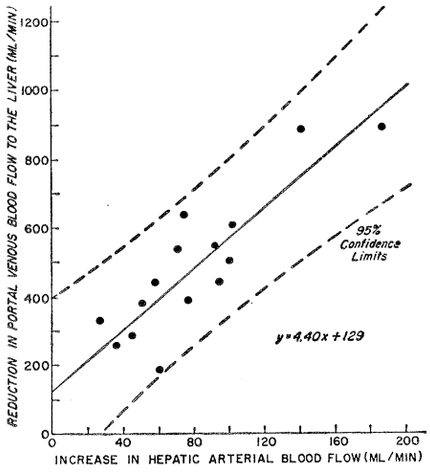

Fig. 1. The effect of reduction in portal venous blood flow to the liver on blood flow in the hepatic artery. The reduction in portal venous flow was produced by occlusion of the superior mesenteric artery or by formation of a side-to-side portacaval shunt. The increase of blood flow in the hepatic artery is directly related to the decrease of the flow in the portal vein.

Two similar experiments were performed in dogs that had been given reserpine (5 mg/kg of body weight) 24 hours before the experiment to eliminate a possible effect of epinephrine. The systemic pressure in these dogs was lower than that in the untreated dogs, and the systolic pressure stayed at about 100 mm-Hg throughout the experiment. In these animals the re-

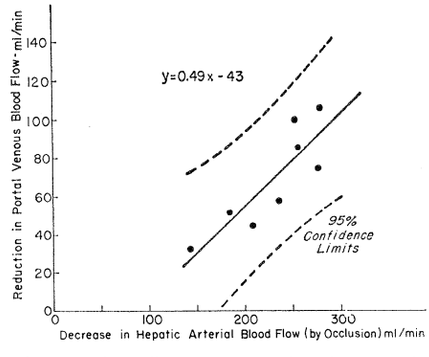

Fig. 2. The effect of reduction in hepatic arterial blood flow to the liver on blood flow in the portal vein. A decrease in hepatic arterial flow results in a decrease in portal venous flow. The amount of decrease of the blood flow in the portal vein is directly proportional to the increase of the flow in the hepatic artery.

lations between the portal venous and hepatic arterial blood flows did not differ from those of the dogs that had not received reserpine.

The portal venous and hepatic arterial systems are not related by the usual capillary venular connections. At least three forms of communication between these two systems have been described (*4*): (i) connections between portal and arterial sinusoids are noted in the periphery of the liver lobule; (ii) direct anastomoses exist between corresponding divisions of the portal vein and hepatic artery; and (iii) arterial branches frequently end in the terminal branches of the portal vein just before these branches empty into sinusoids. It has been estimated that at least 30 percent of the hepatic arterial blood is shunted into the portal venous system before the arterial blood reaches the sinusoids (*5*). Wakim observed that the portal blood flow is much faster distal to anastomatic communications with branches of the hepatic artery (*4*).

The hepatic artery–portal vein blood flow relations which we observed can be explained as simply the mechanical effect of the interposition of a slower-flowing stream in the path of a faster-flowing stream. The rate of flow in the slower system will be increased and the rate of flow in the faster system will be proportionately decreased. Decreasing the amount of slow flow is equivalent to removal of an impedance, and the rate of flow in the faster system should increase. Conversely, if the flow impetus of the faster system is decreased, the flow rate in the slower system should decrease. The relation between the systems is thus one of work mechanics, with a transfer of energy from the arterial to the venous system made possible by direct anastomoses of the two systems in the liver.

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References and Notes

1. R. E. Condon, L. M. Nyhus, N. S. Chapman, H. N. Harkins, *Gastroenterology* **43**, 547 (1962); J. L. Bollman, M. Khattab, R. Thors, J. H. Grindlay, *Arch. Surg.* **66**, 562 (1953).
2. H. Schweigk, *Arch. Exp. Pathol. Pharmakol.* **168**, 693 (1932); S. Soskin *et al.*, *Am. J. Physiol.* **124**, 558 (1938).
3. Square-wave electromagnetic flow meter, model 202, from Carolina Medical Electronics, Inc.
4. K. G. Wakim, *Am. J. Med.* **16**, 256 (1954).
5. N. Rabinovici and J. Vardi, *Surg. Gynec. Obst.* **120**, 38 (1965).

2 August 1965

Physiological Predetermination: Imbibition, Respiration, and Growth of Lima Bean Seeds

Abstract. *Temperatures 15°C or lower during the first hour of imbibition immediately inhibited respiration in lima beans, with proportional inhibition of subsequent growth of seedlings. Correlations between initial respiration rates and growth rates of seedlings were found in different lots of seeds.*

Almost 50 years ago Kidd and West (*1*) reported that conditions during the early stages of germination could "pre-determine" subsequent growth of plants. More recent experiments (*2*) have shown that in lima beans (*Phaseolus lunatus*, L., var. Early Thorogreen) temperatures of 15°C or lower during the first hours of germination may severely inhibit later growth of seedlings. Susceptibility of lima beans to chilling injury is predetermined during seed development; seeds that bleach during maturation are more sensitive than normal seeds. In corn (*Zea mays*), measurements (*3*) of respiration rates 1 to 3 hours after the start of imbibition detected injury by heat or freezing and could be used to predict growth of seedlings. It is important both to find practical ways to predict plant behavior and to explain the mechanism of predetermination. We now report data showing that respiration is associated with chilling injury of lima beans, that the extent of the injury can be estimated from measurements of respiration, and that respiration rates are also related to subsequent growth of seedlings even in the absence of injurious treatments.

Chilling treatments that inhibited respiration also inhibited growth of seedlings (Table 1). Imbibitions at 15°C inhibited respiration and seedling growth, and the more severe treatment at 5°C inhibited respiration more markedly, eventually killing the seeds. It was shown (*2*) that bleached seeds are more susceptible to chilling injury than normal green seeds, and that susceptibility is limited to the first few hours of imbibition. After measurement of respiration, seeds were planted on moist paper towels and incubated in darkness at 25°C. Respiration rates 4 hours after the beginning of imbibition were correlated, at the 5-percent level of significance, with axis lengths of seedlings after 5 days of germination (Fig. 1). Comparison of initial respiration rates with fresh weights of seedlings of different lots of green and bleached seeds, at the 1-percent level of significance (Fig. 2, *A* and *B*, respectively), indicates a relation, between respiration rates at the start of germination and subsequent growth of seedlings, similar to that reported for corn (*3*). The correlation indicates that measurements of respiration can lead to a rapid estimate of the extent of injury caused by chilling treatment.

In one lot of seeds, respiration of bleached seeds was less than that of green seeds during the temperature-sensitive period, but not subsequently. These results, and observations that anaerobic conditions enhance the chilling injury (*2*), suggest that respiration rates during imbibition may be a factor in determining susceptibility to chilling injury. Relations between levels of respiratory energy, measured as concentrations of adenosinetriphos-

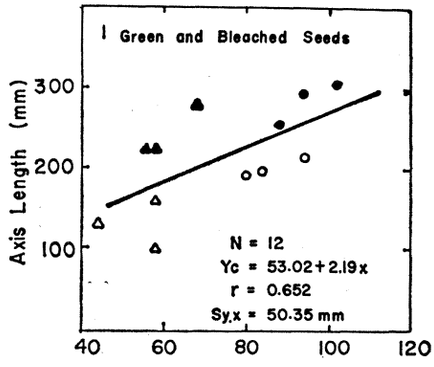

Fig. 1. Comparison of respiration of green and bleached seeds of lima beans 4 hours after initial imbibition with axis length 5 days later. ●, green seeds, imbibition at 25°C; ▲, green seeds, imbibition at 5°C; ○, bleached seeds, imbibition at 25°C; △, bleached seeds, imbibition at 5°C.

Table 1. Influence of imbibition temperatures on respiration and growth of seedlings in green and bleached seeds of lima bean.

Temp., first hour (°C)	Respiration, O_2, each (μl/hr)*		Growth (mm)	
	2-hr	6-hr	Roots	Shoots
		Green		
25†		95	169	115
15†		60	131	111
25‡	34	91	106	99
5‡	4	52	0	0
		Bleached		
25†		87	122	79
15†		54	71	59
25‡	25	80	60	48
5‡	5	46	0	0

* Respiration measured at 25°C. †, ‡ Seedling growth measured 5 (†) and 4 (‡) days after the start of imbibition.

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