Intestinal Absorption and Blood Flow

C. C. MAO AND EUGENE D. JACOBSON

Although an everted gut sac can move molecules from mucosal to serosal surfaces, the intact intestine utilizes its circulation as an integral part of the transport process, and blood flow must be considered as a potential rate-limiting step. The interaction between the circulation and intestinal absorption is a two-way street: absorption influences blood flow and the reverse holds as well. Digestion, motility, and intestinal energy consumption also affect blood flow, either by direct local stimulation or through distant release of humoral agents.

The effect of specific nutrients on splanchnic blood flow has been examined. There is general agreement that instillation or ingestion of hypertonic glucose solutions into the gut evoked an increase in both splanchnic and mesenteric blood flows (1–5). The effect of isotonic glucose was variable (2, 3).

Protein ingestion caused an increase in intestinal blood flow in humans (6) and in conscious dogs (7). Instillation of glycine in the gut loop of an anesthetized dog had the same result (2). Mesenteric blood flow was greatly augmented by dietary fat (8) but not by isotonic saline (2).

Ingestion of a mixed, usual diet increased blood flow in the conscious dog (9, 11). Once the food was in the gastrointestinal tract and digestion assumed to commence, there were further increases in mesenteric blood flow (9–12). Presentation of food induced a lesser rise in blood flow than the digestive process, and atropine opposed the postprandial hyperemia, suggesting considerable central nervous influence on the circulatory response (12).

We can summarize the preceding information by saying that the digestive process seems to demand a greater intestinal blood flow. We cannot determine, however, whether the augmented blood flow is caused by a neurohumoral reflex, the products of digestion, the metabolites involved in mucosal elaboration of digestive enzymes, or metabolites associated with intestinal absorption.

What of the reverse relationship, namely, the effect upon intestinal absorption when blood flow is varied? It is apparent that a total shutdown of perfusion would slow and eventually stop absorption of both passively and actively transported substances. There is information available concerning effects of less complete restriction of blood flow upon absorption from the gut. With severe hemorrhage, uptake of water and glucose (but not of saline) was impeded in dogs (13). Large amounts of epinephrine also impaired absorption of glucose and diminished mesenteric perfusion (14). Where more controlled reduction in mesenteric perfusion has been reported, it was found that mesenteric blood flow had to be lowered at least by 50% to diminish absorption of glucose (15, 16). Once blood flow became
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limiting factor; there was an excellent parallel between changing rates of perfusion and absorption of both actively and passively transported sugars, sodium, iron, and drugs (15–21). Marked elevation of mesenteric venous pressure also reduced absorption of glucose (22).

It is not possible to diminish mesenteric blood flow in man in a controlled fashion. Exercise in the heat does evoke a fall in splanchnic perfusion. This maneuver has been reported to decrease absorption of 3-O-methyl-d-glucose, an actively transported but not metabolized hexose, but not of d-xylose, a passively absorbed sugar (16). In another study, with differing conditions of exercise, no decrease in absorption of sugars was observed (23).

Thus, it appears that tissue perfusion can be a limiting factor in intestinal absorption when the degree of blood flow reduction is great. What is uncertain, however, is whether this vascular factor is important normally, i.e., in conditions other than mesenteric vascular disease or a laboratory experiment.

There is ample evidence showing parallel directional changes in blood flow and oxygen consumption during intestinal absorption of nutrients (2, 6), in response to vasoactive drugs (14) and with mechanical restriction of blood flow (15, 21). These findings suggest that intestinal absorption depends upon the intestinal circulation for oxygen to run metabolic activities associated with transport and that intestinal blood flow is regulated by local factors concerned with those metabolic activities. Unfortunately, there is no rigorous proof for this concept, and we are unaware of the degree to which absorption, blood flow, and oxygen consumption are normally linked. Furthermore, we are unable to identify the chemicals relating blood flow changes to absorption. Although gastrointestinal hormones (7) and central nervous activity (12) both increase mesenteric blood flow, their influence on intestinal absorption is undefined. Local metabolites associated with transport of nutrients across the gut are more likely mediators of the circulatory–absorptive relationship, but detection of such materials has defied the best efforts of physiologists in this century.

Intestinal motility is also involved in digestion and absorption of food. Moving gas and fluid may distend segments of gut. Stimuli of intestinal motor function also alter vascular smooth muscle tone. This combination of factors, which potentially or actually influence mesenteric blood flow, makes it difficult to generalize about the effect of motility upon intestinal blood flow. A summary position, based upon recent work (24–28) would be: 1) passive distension of the gut by gas or fluid will diminish local blood flow, depending upon the degree of distension; 2) increased motor activity tends to increase blood flow if the contractions are not excessively vigorous and decrease blood flow when contractions generate large pressure changes in the lumen of the gut; and 3) the neurohumoral mediators of intestinal smooth muscle activity (acetylcholine, epinephrine, et cetera) also have significant direct effects upon the mesenteric circulation, which overshadow the circulatory response to changes in motility.

A common bit of lay medical lore concerns the inadvisability of swimming postprandially. According to the story, splanchnic blood flow is greatly increased by the meal, and the exercising muscles are unable to get their needed share of the cardiac output. The denouement of this tragedy is that the muscles become ischemic, develop cramps, and the ill-informed swimmer drowns. We have noted previously that digestion and absorption of nutrients do augment mesenteric blood flow, findings that support the concept of a competition for cardiac output between the viscera and exercising muscles. However, the exercising man also increases his cardiac output. The real question, there-
Therefore, is whether digestion and absorption of food induce a redistribution of cardiac output resulting in an increased proportion of total circulation flowing to the gut.

Surprisingly, the older evidence in the literature militated against the idea that ingestion of a meal evoked a selective increase in intestinal blood flow at the expense of other regional circulations. Studies of the conscious dog and the rat indicated that the increase in mesenteric blood flow after eating was no greater than the increase in cardiac output or the increase in blood flow to other nondigestive organs (10, 11). However, these investigations employed techniques for estimation of mesenteric blood flow that no longer hold the confidence of circulatory investigators. Furthermore, newer evidence suggests that the processes of eating, digesting, and absorbing food cause a redistribution of cardiac output to the gut. In conscious dogs with implanted electromagnetic (7, 9, 12) or ultrasonic (12) flowmeters, it was demonstrated that blood flow to the gut increased after eating out of proportion to changes in cardiac output. In addition, studies of gut segments in anesthetized dogs revealed a higher blood flow in loops absorbing hypertonic glucose solutions than in empty loops of intestine (3).

It appears that the advice given to swimmers may have a basis in fact.

Another applied problem involving intestinal absorption and blood flow is the pathophysiology of the dumping syndrome. It was demonstrated in man (1) and in the dog (5) that hypertonic glucose solutions in the gut caused an increased flow of blood through the gut. This effect was associated with a decrease in blood volume as well, suggesting a movement of water from the vascular compartment into the upper gut to dilute the hyperosmolar sugar solution. In gastrectomized patients these effects on splanchnic blood flow and blood volume were more marked than in normals (1). Since the rapid entry of large volumes of fluid into the upper gut can evoke diarrhea (5, 29) and other symptoms seen in the dumping syndrome, this circulatory basis for the disorder seems attractive, if unproved.

We can summarize the relationship between intestinal blood flow and absorption with a few positive generalizations. The events immediately preceding absorption—feeding and digestion—and absorption per se seem to evoke a selective increase in blood flow to the gut. The mediators of this local vasodilation are unknown, but the gut itself appears to originate some of the stimulus for an increased blood flow during absorption of nutrients. The likely implication of the intestinal circulation in the dumping syndrome and certain forms of chronic malabsorption (30) emphasizes the pathophysiological aspects of the interaction between blood flow and intestinal transport.

REFERENCES


8. KILMORE, M. A., J. T. MCCORMICK, JR., R. I.
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