Gut, 1967, **8**, 203

Mesenteric arterial disease: the present position

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An organ lives on its blood supply, and the gastrointestinal tract is no exception. It is nonetheless true that until recently more interest has been shown in the function of the alimentary system as an absorptive or excretory organ than in the physiology of its own nutrition which makes these functions possible. There is at present no method of measuring blood flow to the intestine of an intact human being, because its arterial supply, unlike that of the heart or kidney, arrives through three separate vessels with a very variable pattern of internal anastomosis, and its venous return is via an internal, portal, system which cannot be catheterized. A measurement of total intestinal blood flow might not, in fact, be very useful, because without knowing how this was shared between the absorptive, the motile, and the supporting tissues of the gut, it would be difficult to correlate clinical abnormalities with any quantitated deficit in arterial input[1]. Also, we have come to realize that the symptoms and signs of bowel ischaemia are very difficult to interpret at the bedside, and there is as yet no system of laboratory tests which will reliably distinguish them from any other cause of major illness. However, with the advent of more refined biochemical tests and of selective visceral angiography, our understanding of these conditions is growing and it seems appropriate to review, very briefly, the present position.

The problem of acute, massive bowel infarction from an embolus or thrombosis is still far from solved, which is not surprising because this lesion creates a disturbance of almost unimaginable complexity, involving all the physiological sequelae of plasma volume depletion, combined with a massive metabolic acidosis and the effects of tissue death and bacterial invasion. The accident usually happens, moreover, to a patient already enfeebled by cardiovascular disease, so that it is not surprising that mortality figures have scarcely changed over the last 20 years. Although it is now possible to restore blood supply to the bowel by appropriate vascular operations, uncompensated restoration may itself hasten death. Certainly, in the experimental animal, where superior mesenteric ligation is a standard experimental procedure, it is well known that release of such a ligature results in rapid circulatory collapse (SMA shock). There are two main reasons for this collapse, namely, exsanguination into the bowel on the one hand[2] and absorption of toxic substances (cellular enzymes, histamine, free potassium ion, and Gram-negative endotoxin)[3,4,5] into the portal circulation and peritoneum, on the other. The first of these abnormalities is treatable, and it is now generally agreed that acute and total intestinal ischaemia requires massive plasma and blood volume replacement.[6] The use of low-molecular weight dextran, which is a powerful plasma expander and may also improve flow in the smaller bowel arteries, has experimental backing and has been reported as useful.[7,8] Treatment of the portal toxaemia is more difficult. The most promising experimental results have followed the use of coeliac ganglionectomy and α-adrenergic blocking agents such as phenoxybenzamine[9,10] but most clinicians would hesitate to use long-acting hypotensive agents in a desperately ill patient with failing myocardium and kidneys, even if the blood volume and central venous pressure could be controlled. (The position of the young fibrillator with a mesenteric embolus is somewhat different.) At the same time the frequency of 'agnogenic' infarction, that is, of gut necrosis in the absence of large vessel occlusion, as a complication of myocardial infarction and other hypotensive states, is becoming increasingly recognized.[11,12,13]

With regard to chronic midgut ischaemia as a cause of abdominal pain ('intestinal angina') and malabsorption, most workers who have studied the problem have been impressed with the rarity of this condition. Atheroma of the visceral arteries is common, and complete occlusion of two or

even three of the main trunks is a not unusual incidental post-mortem finding.[14,15] Experiments devised to produce bowel abnormalities in the experimental animal by chronic obliteration of the arterial supply result usually either in death from acute infarction or survival in perfect health, as judged by standard tests.[6,16] In the clinical laboratory no consistent abnormality has been found in patients with chronic intestinal ischaemia, whether they have symptoms or not. The diagnosis depends on aortography. Stimulated by the few case studies in which relief from intestinal angina has followed arterial reconstruction[17,18] many aortograms have been carried out where the symptoms seem suggestive, sometimes with negative results and sometimes revealing other conditions such as a growth of the pancreas or stomach, which in retrospect could have accounted for the clinical picture. Many aortographic studies of suspected intestinal angina conceal the number of negative examinations. The paper by Drs. Dick, Graff, Gregg, Peters, and Sarner in the present issue[19] is of great importance because it represents the first unselected survey of the intestinal vasculature in a wide range of patients with and without abdominal symptoms, and it provides us with some idea of the relation between blood flow (as estimated by the summed cross-sectional area of the arteries, which the authors certainly understand to be only roughly correlative) and symptoms. It is noteworthy that of 11 of their patients undergoing aortography because of suspected intestinal angina, six were found to have other diseases common in this age group. The remaining five had variable symptoms suggestive of mesenteric vascular disease. As the authors imply, the only consistent feature of this anatomical lesion is the variability of its symptoms! Of these five patients, the fact of chronic intestinal ischaemia was proven in two, of whom one had complete relief following arterial reconstruction. It is impossible to assess the importance of intestinal angina as a common cause of obscure abdominal pain while our only means of diagnosis is the expensive and risky method of aortography. That most patients who die of a mesenteric thrombosis have a prodromal period of angina, much as lower limb gangrene is preceded by claudication, seems certain.[20] But how many patients with ischaemic bowel pain actually come to a fatal infarction we do not at present know. Symptomless narrowing of the superior mesenteric artery can be a threat to life if the patient suffers an unrelated hypotensive episode[2] or undergoes a gut resection[21] but how often this happens is also uncertain. The answer can only come through such patient and detailed study of the intestinal vasculature in health and disease.

When we consider the effects of acute occlusion of the smaller vessels supplying the bowel we are on surer ground, because this lesion causes a structural defect which can be studied by ordinary methods, but at the same time is not usually fatal. It has long been known that arterial insufficiency to a short length of bowel produces a characteristic picture of mucosal destruction and fibrosis, but it is only recently that clinicians and pathologists have begun to separate these lesions from the ragbag of 'non-specific' enteritis and colitis. We can again learn from the cardiologists. Sudden death of a portion of myocardium is explicable only on grounds of ischaemia. The inflammatory reaction is slight because the human heart is sterile. But sudden death of part of the gastrointestinal tract is immediately followed by bacterial invasion, and for this reason, although intestinal infarcts may for all we know be as common as infarcts of cardiac muscle, they are usually interpreted as specific infections, and the unique capacity of the healthy intestinal mucosa to resist bacterial challenge is forgotten. There are of course certain organisms which have been evolutionarily selected to survive in the presence of gastric acid, bile, and lysozyme, and cause bowel illnesses which are epidemiologically recognizable, but an inflammatory reaction in the bowel, with or without bacterial invasion, that is not reliably connected with such an organism, should prompt the question of why it has occurred. Certain patterns of illness, such as Crohn's disease and proctocolitis, are definable in terms of their natural history and morphology. We accept these as diagnoses, when they are in fact descriptive definitions, and hence must be very critical of the case of 'atypical' Crohn's disease or colitis. Of the various factors which can affect mucosal resistance to bacteria, an obvious one is ischaemia, and it is now clear that many bowel lesions which have baffled us in the past are in fact examples of the infected infarct, the end-product of which, clinically and pathologically, reflects the interplay of variations in blood supply and intraluminal flora.[22] These infarcts are, predictably,

more common in the colon than in the small bowel, because of the pattern of arterial supply and bacterial population, and the syndrome of 'ischaemic colitis' is well established[23] while ischaemic destruction of small intestinal segments is less well understood except in the purely mechanical case of strangulation. It is ironic that the introduction of the enteric-coated potassium tablet should have drawn attention to what may be a common form of illness in our atherosclerotic society[24,25,26]. Of great interest is the fact that the clinical and pathological picture of infarction can occur without a major artery being demonstrably blocked, particularly in such conditions as rheumatoid and polyarteritis, which is a reminder of how little is known of the physiological control of the small vessels of the bowel. This is one of the growing points of gastroenterology and will preoccupy us for some time to come.

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