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Does 'intestinal angina' exist? A critical study of obstructed visceral arteries

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SUMMARY

A study of the mesenteric arteries was carried out in 203 unselected autopsies. Although mild degrees of stenosis were common, the incidence of a 'critical stenosis' was rare and no correlation between degrees of stenosis and previous gastrointestinal symptoms could be found. Radiological demonstration of stenosed or blocked visceral arteries remains therefore of doubtful clinical significance.

THE gut is supplied by three arteries, the coeliac axis (CA), superior mesenteric artery (SMA) and inferior mesenteric artery (IMA). Ever since visceral angiography became possible, there has been much interest in the part which occlusion of these visceral trunks may play in the causation of symptoms, and the expectation of life. A great deal of animal research has been carried out to try and elucidate the problem, and patients have been subjected to ambitious and dangerous operations designed to correct arterial blockages to the alimentary tract. The concept of 'intestinal angina', that is to say pain experienced in relation to meals arising from an ischaemic intestine, although postulated by earlier workers, really gained credence when Dunphy (1) in 1936 showed that of 12 patients dying from necrosis of the alimentary tract, 7 had complained of food-related pain for a variable period (weeks to years) before they were overtaken by the fatal infarct. The series was small and, although much has been written since, these observations have never been exactly repeated.

The purpose of the present study has been to re-examine the incidence of atheromatous obstruction of the CA, SMA and IMA, and for the first time to relate the pathological lesion to the patient's symptoms and to his or her eventual fate.

Materials and methods

The three mesenteric vessels of 203 unselected autopsy specimens were examined either by a surgeon, a pathologist or both. Details of age, sex, cause of death and the presence or absence of previous gastrointestinal symptoms were noted. The gastrointestinal tract was removed and examined in detail. The abdominal aorta from the diaphragm to the bifurcation was then excised *en bloc*, together with the first 5–8 cm of the mesenteric arteries. The posterior wall of the aorta was opened longitudinally, and the presence of aortic atheroma noted and graded from 0 to 4 (2). The first 5 cm of the CA, SMA and IMA were then freed from surrounding tissues and, using a set of calibrated bougies, the narrowest intraluminal diameter (I) of the first 5 cm of artery was determined in millimetres. At this position the extraluminal arterial diameter (E) was measured in millimetres using callipers. The percentage stenosis was calculated from the formula

$$\frac{E-I}{E} \times 100 \text{ per cent}$$

for each of the three vessels.

Conventionally, a stenosis is assessed on an arteriogram by measuring the narrowest intraluminal diameter and expressing it as a percentage of the 'normal' proximal or distal diameter, although the presence of post-stenotic dilatations make such comparisons with distal diameter unreliable. In preliminary autopsy studies carried out before this work began it was found impossible in practice to define a 'normal' artery proximal to a stenotic lesion, as some degree of atheroma was always present.

Measurement of the external diameter of the vessel at the point where the internal diameter is narrowest, and using this formula to calculate stenosis, assumes that an artery with 0 per cent stenosis has an arterial wall thickness of 0 mm. The percentage stenoses figures quoted in this series will thus be marginally higher than those calculated from angiography. However, for the reasons stated, the method used was considered the most valid.

Results

The age range of the 203 patients studied at autopsy was 19–97 years, with 79·2 per cent falling within the 40–80 year age group. The male to female ratio was 1·2 : 1. Twenty-three per cent of the aortas examined were normal or showed fatty streaking; 60 per cent showed mild to moderate atherosclerosis and 17 per cent had severe atherosclerosis. The mean stenosis of the 203 CA examined was 21·9 per cent (s.d. ±12·4) with a range of 7–100 per cent. The mean stenosis of the SMA was 27·3 per cent (s.d. ±13·3) with a range of 7–100 per cent. The IMA mean stenosis was 30·5 per cent (s.d. ±14·0) with a range of 9–100 per cent. Of the CA examined, 3·4 per cent had a stenosis greater than 50 per cent. The remaining 90 per cent had a varying degree of stenosis but all were less than 50 per cent. Of the SMA examined, 4·9 per cent had stenotic lesions greater than 50 per cent and 95·1 per cent (the remainder) were less than 50 per cent stenosed. A total of 10·8 per cent of IMA had a greater than 50 per cent stenosis, but 89·2 per cent had stenoses of less than 50 per cent. Of the patients with arteries showing stenosis of greater than 50 per cent who had experienced gastrointestinal symptoms during life, all had other causes to explain these. The distribution of stenotic lesions either greater than or less than 50 per cent within the four age categories is shown in *Table* I. A total of 41 (20·2 per cent) of the 203 autopsies studied had recorded ante-mortem gastrointestinal symptoms, of whom 25 (12·3 per cent) died as a direct result of gastrointestinal pathology. Of these 41 cases, 6 (3 per cent) had previously undiagnosed abdominal pain for which no cause was found at post-mortem examination. One of the 6 cases had been diagnosed as abdominal angina, but at autopsy the stenosis of the CA, SMA and IMA was 13·3, 23·5 and 28·6 per cent respectively. The stenosis of the mesenteric vessels in the remaining 5 were all less than 50 per cent.

One patient had had an embolus removed from the SMA 10 years before death and had been asymptomatic

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Table I: DISTRIBUTION OF STENOTIC LESIONS EITHER > OR <50 PER CENT WITHIN THE FOUR AGE CATEGORIES

Artery	Age group (yr) <40 ($n=7$)	40–60 ($n=60$)	61–80 ($n=101$)	>80 ($n=35$)
CA stenosis (%)				
<50	100	98·3	95·9	92·9
>50	0	1·7	4·1	7·1
SMA stenosis (%)				
<50	100	96·8	95·2	96·6
>50	0	3·2	4·8	3·4
IMA stenosis (%)				
<50	85·7	93·2	87·4	84·4
>50	14·3	6·8	12·6	15·6

since. At autopsy all three mesenteric vessels were occluded, and there was a visible collateral circulation. (This was the only case where the collateral was sufficiently developed to be apparent at autopsy.) Another patient had died following a large intestinal resection for acute intestinal infarction, and at autopsy the stenosis of the CA, SMA and IMA was 13·3, 12·5 and 9·1 per cent respectively.

The remaining 33 patients had ante-mortem gastrointestinal symptoms for which a cause other than mesenteric ischaemia was found. However, in 4 patients dying from intestinal carcinoma, 1 patient had an SMA stenosis of 61·5 per cent, and 3 had IMA stenosis of greater than 50 per cent.

Four patients had two of the three mesenteric vessels greater than 50 per cent stenosed, and all 4 had no recorded ante-mortem gastrointestinal symptoms. No collateral circulation was visible.

Discussion

Theoretically, if it were possible to identify the patient with visceral artery disease whose abdominal pain presages an intestinal infarction, one could avoid this catastrophe by reconstructing the blood supply to the gut. The relationship between stenotic lesions of the visceral arteries and alimentary symptoms is therefore very important.

However, there are four additional difficulties. First, it has been claimed that stenosis and occlusions of the mesenteric arteries are common post-mortem findings in series of cases either selected or unselected for vascular disease elsewhere (2–5). The collective findings may be summarized as follows: 42 per cent of CA, 46 per cent of SMA and 65 per cent of IMA inspected showed some degree of stenosis, usually occurring within 2 cm of the aortic ostium. The average narrowing of the SMA in one small series of 75 unselected autopsy specimens was 28 per cent. Of 110 CA, 21 had a stenosis of 50 per cent or more (4). However, the number of arteries examined has been small, some patients have been selected for the presence of advanced atherosclerosis, the methods of measurement have been variable and most of the figures refer to the number of arteries stenosed rather than to the degree of stenosis. In these studies, the relation between the state of the vessels and the gastrointestinal symptoms experienced by the patients during life, has not been examined.

Secondly, it is generally believed that to warrant a diagnosis of intestinal angina, at least two out of three mesenteric arteries should be stenosed (6), but the degree of stenosis necessary to qualify has not been clarified. Although the 'two out of three' rule may be correct in the majority of patients, we have seen intestinal angina with single vessel occlusion cured by revascularization. In this series, there were four patients with two of the three mesenteric arteries stenosed greater than 50 per cent, and in all four there was no record of ante-mortem gastrointestinal symptoms.

Thirdly, the relation of surgically identifiable vascular disease to fatal gut infarction is uncertain. One-third of patients dying from acute intestinal failure demonstrates no abnormality in the SMA (7–10). Although in the remainder some degree of stenosis may be found, we know that such stenosis may be asymptomatic (1–3, 11). The reasons why the gut should die in spite of the presence of an intact vascular tree have been amply discussed elsewhere (12), and are variable and complex. The fact that this type of fatality does occur, however, casts great doubt on the rationale of surgical reconstruction of visceral arteries.

Finally, an intraluminal diameter reduction of 50 per cent is equivalent to an intraluminal cross-section area of just under 80 per cent. Although reduction in pulsatile flow and distal pressure occurs before this level is reached (13), mean blood flow is not affected until a stenosis has reduced the intraluminal cross-section area by 80 per cent (14). Hence the choice of a 50 per cent intraluminal diameter reduction as a 'critical stenosis'. Resting mean blood flow within the SMA is between 80 and 1400 ml/min (15) and this is known to rise by a factor of 50 per cent during food ingestion (16). Intestinal angina almost certainly results from an incapacity of the mesenteric vascular bed to increase its flow in response to food intake, and it might be supposed that a rigid 50 per cent intraluminal diameter stenosis close to the origin of a visceral artery might bring this about. However, the present study fails to confirm this.

Conclusion

This study is the largest so far reported and the overall figures suggest that although mild degrees of stenosis of the mesenteric arteries are common, the incidence of a 'critical stenosis' is rare (CA: 3·4 per cent; SMA: 4·9 per cent; IMA:10·8 per cent). Furthermore, no correlation between degrees of stenosis and previous gastrointestinal symptoms could be found. Radiological demonstration of stenosed or blocked visceral arteries remains, therefore, of doubtful clinical significance, and we still have no method of identifying the patient with visceral arterial disease and abdominal pain who risks developing an intestinal infarction.

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Fifty years ago (from the *British Medical Journal* 4 January 1930)

Social surgical history: 'a promiscuous principality'

TWO CASES OF ACUTE SYPHILITIC APPENDICITIS.

BY

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(*With Special Plate.*)

THE increase in visceral syphilis is one of the features of medicine in the present century, but up to now not many cases of acute syphilitic appendicitis have been reported. In searching for such records we have had the able and generous help of the librarian of the British Medical Association, but have succeeded in finding two only—those reported by Pasqualis (D. A.).[1]

In Royster's exhaustive monograph on appendicitis (Appleton, 1927) there is reference to pseudo-appendix associated with secondary syphilis. This condition is classed by Condamin and Voron (ibid.) with the form of intestinal neuralgia which Fournier had called syphilitic typhosis.

The two cases about to be described, however, are true acute inflammations.

CASE I.

A woman, aged 19, felt well until she had a miscarriage in the fifth month on the floor of a farm kitchen. During the following week she developed lower abdominal pain and tenderness with pyrexia.

On admission to hospital she did not look very ill; temperature 100°, pulse 120, respirations 26. The throat was red and covered

On opening the appendix the mucous membrane was found to be of a uniform pale pink with no evident ulceration. The

thickening was clearly due to infiltration of the subperitoneal and submucous layers.

Recovery was uneventful, and the patient was discharged on the twelfth day. The tongue became clear the day following the first injection. The Wassermann reaction was positive.

The two cases had the following corresponding features.

CASE I.	CASE II.
Wassermann reaction +.	Wassermann reaction +.
Specific throat.	Mucous patches on tongue.
Denied infection, but was unmarried at the time of the miscarriage.	Denied infection, but had an illegitimate child seven years previously.
One of 10 healthy children, and no miscarriage in the mother.	One of 5 healthy children and no miscarriage.
Mother's W.R. ++. Sister's W.R. +. (No opportunity for examining other members.)	One brother has definite facies of congenital syphilitic.
No vomiting throughout the illness.	No vomiting.
Macroscopically acute appendix with unbroken mucous membrane.	Macroscopically acute appendix with unbroken mucous membrane.