Pattern of Aorto-iliac Occlusion

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[WITH SPECIAL PLATE]

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Since the introduction of aortography by Dos Santos (1929) and Leriche's (1940) classical description of thrombosis of the aortic bifurcation it has been recognized that there are many types of aorto-iliac disease, and that the pattern of aorto-iliac occlusions is varied and complex.

In the present investigation this pattern has been studied in 100 aortograms in which occlusions responsible for producing intermittent claudication were found, patients with ischaemic rest pain or gangrene being excluded. This group is a selected sample of the patients presenting with aorto-iliac occlusion and claudication, because approximately one-third of those seen in the outpatient department were not subjected to aortography, because they either were too old or were otherwise unfit for reconstructive surgery. Most of the aortograms, therefore, were performed in patients aged under 60.

TABLE I.—Classification of Principal Aorto-iliac Lesions in 100 Aortograms According to Probable Site of Origin

Site of Origin of Lesion	Type of Lesion
Aortic bifurcation 34	Aortic occlusion:
	To renal artery 4
	To inf. mesenteric artery 6
	Aortic bifurcation stenosis:
	Unilateral 3
	Bilateral 17
	Common iliac occlusion:
Common iliac artery 31	Unilateral 1
	Bilateral (one stenosed) 3
	Common iliac stenosis:
	Unilateral 18
	Bilateral 3
	Common iliac occlusion:
	Unilateral 5
Common iliac bifurcation 28	Bilateral (one stenosed) 5
	Stenosis of common iliac bifurcation 2
	External iliac occlusion 14
	Internal iliac occlusion 3
	Combined iliac occlusions:
	Common and external 6
	External and internal 2
Common femoral artery 7	Common, external, and internal 1
	Common femoral occlusion 7

The indication for aortography in these patients was absent or diminished femoral pulsation, and the lesion responsible for claudication in seven patients was an occlusion of the common femoral artery (Table I).

The lesions found have been classed either as "principal" lesions—that is, the lesion responsible for the intermittent claudication—or as "subsidiary" lesions which were incidentally present and not producing symptoms. For example, Fig. 1 (Special Plate) illustrates a left external iliac occlusion (the principal lesion) which caused left-sided claudication, and the stenosis of the right common iliac artery due to an atherosclerotic plaque is an incidental finding—that is, subsidiary lesion.

The principal lesions are tabulated in Table I, and each has been carefully studied to determine its probable site of origin. This can readily be determined when the lesion is incomplete (Special Plate, Fig. 2), but consecutive thrombosis after occlusion can obscure the picture, and deduction regarding the site of origin of lesions has been difficult in a minority. Analysis

suggests that there are three common sites of origin for aorto-iliac occlusions—the aortic bifurcation, the common iliac artery itself, and the common iliac bifurcation.

Occlusions of the Aorta

In 34 of the 100 cases the arterial lesion appeared to originate at the aortic bifurcation (Table I).

In 20 cases there was atherosclerotic narrowing of the aortic bifurcation, which was mild in two cases and severe in 18 (Special Plate, Fig. 3). This usually produced stenosis of the orifices of both common iliac arteries, but in three cases narrowing of the common iliac origin was unilateral. In the aortogram of one female patient the lower abdominal aorta was narrowed just above the bifurcation, and this lesion has now been seen in two other female patients not in this series.

In three cases the common iliac artery was occluded on one side, with evidence of narrowing of the orifice of the other common iliac artery (Special Plate, Fig. 4). One case showed unilateral narrowing of the origin of the common iliac artery.

Lesions of the aortic bifurcation may progress at any time either because further thrombosis occurs or because a portion of platelet thrombus becomes detached and plugs the orifice of a common iliac artery. When both common iliac arteries are occluded thrombus will extend proximally as far as the outflow into the inferior mesenteric artery (six cases) (Special Plate, Fig. 5) or the renal arteries (four cases).

Aortic bifurcation lesions are therefore responsible for 34% of aorto-iliac lesions producing intermittent claudication in this series.

Occlusions of the Common Iliac Artery

Lesions of the common iliac artery (stenosis or occlusion) were the commonest form of aorto-iliac occlusion found (Table II). In compiling this table all major lesions, both principal and subsidiary, were included. In the 100 aortograms 142 lesions were found, and common iliac lesions accounted for 61 (43%) of these (22 occlusions, 34 stenosed arteries, and 5 with stenosis of the common iliac bifurcation).

TABLE II.—Classification of 142 Major Lesions (Stenosis or Occlusion) in 100 Aortograms

Site of Occlusion	No. of Occlusions
Aorta	10
Stenosis at aortic bifurcation	20
Stenosis of common iliac artery	34
Occlusion of common iliac artery	22
Stenosis of common iliac bifurcation	5
Occlusion of external iliac artery	26
Occlusion of internal iliac artery	16
Occlusion of common femoral artery	9

Occlusion of the common iliac artery may be due to lesions arising at one of three sites: (1) the aortic bifurcation (Special Plate, Fig. 3), (2) in the course of the artery itself (Special Plate, Fig. 6), or (3) the common iliac bifurcation (Special Plate, Fig. 7). It is not always possible to determine which of these three sites is the origin of a specific occlusion, but a reasonably accurate deduction can be made in most cases.

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In 31 of the 100 aortograms (Table I) the principal lesion appeared to originate in the course of the common iliac artery, and this site is therefore almost as frequent a source of occlusion as the aortic bifurcation. In 8 of these 31 cases a bilateral distribution was present (Table I). Three showed bilateral stenosis and five showed occlusion of one artery and stenosis of the other. Progression of these bilateral lesions to bilateral common iliac occlusion would lead to occlusion of the aorta itself, and it is therefore possible that some of the aortic occlusions may have arisen from primary common iliac lesions rather than from aortic bifurcation lesions as has been assumed. This suggests that lesions arising in the course of the common iliac artery may be as common as aortic bifurcation lesions.

Bilateral disease is commoner than the figures for bilateral distribution of lesions suggest, because at operation for unilateral occlusion it is common to find that atherosclerotic changes are also severe on the patent side. Symmetrical changes are usual in common iliac disease, although the rate of progression of the two sides is not synchronous.

Occlusions of External Iliac Artery

Occlusion of the external iliac artery accounted for 26 (18%) of the 142 major lesions (Table II). As the principal lesion it occurred in 14 of the 100 aortograms, and in a further nine it was associated with occlusion of other iliac arteries of the same side (Table I).

The frequency of occlusion of this artery is rather surprising, because at operation for aorto-iliac occlusion the external iliac artery is often free from severe atherosclerotic changes. Narrowing of its beginning at the common iliac bifurcation has been seen, and it is probable that occlusion is secondary to lesions of this bifurcation. Occlusion extends to the origin of the inferior epigastric artery, leaving the common femoral artery patent (Fig. 1). Occlusions of the common femoral artery are usually associated with a patent external iliac artery (Special Plate, Fig. 8), the limiting collaterals being the inferior epigastric and circumflex iliac arteries.

Occlusions of Internal Iliac Artery

Occlusion of the internal iliac artery (Fig. 7) as the principal cause of symptoms is uncommon (Table I), and it is more commonly found in combination with other iliac occlusions or as an incidental finding (Table II). Its presence is often not appreciated clinically, as the resultant symptoms of buttock claudication are overshadowed by the thigh and calf claudication produced by occlusion of the main arterial channel. Two cases have been seen in which arterial pulsation in the limb was normal but aortography confirmed that internal iliac occlusion was the cause of buttock pain.

The probable origin of internal iliac occlusions is from lesions of the common iliac bifurcation (Fig. 7). However, at operation atherosclerotic changes in the course of the internal iliac artery are often severe, and it is possible that occlusion may sometimes originate in the proximal 2 cm. of the artery rather than from the bifurcation.

Combined Iliac Occlusions

Occlusions of the common, external, and internal iliac arteries may occur singly or in combination. Because the common and external iliac arteries have no major branches adjacent to the common iliac bifurcation, occlusion will affect the whole of one artery and may occlude the other by consecutive thrombosis. The internal iliac artery breaks up into numerous branches after a short course, and is less liable to secondary occlusion.

Accordingly, the commonest combined iliac occlusion is that which involves the common and external iliac arteries (Table I).

Occlusion of all three arteries is uncommon, as a collateral circulation will develop during the initial period of stenosis and maintain patency of most of the internal iliac at least. Occlusion of all three arteries was found in one patient as a unilateral lesion (Table I).

Discussion

In this analysis an attempt has been made to define the source of aorto-iliac occlusions as well as to determine the relative frequency of each type of occlusion. Lesions of the aortic bifurcation have long been known (Leriche, 1940), as has the occurrence of lesions at other sites, but the present analysis seems to indicate that aorto-iliac occlusion arises with almost equal frequency at three sites—the aortic bifurcation, the common iliac artery, and the common iliac bifurcation.

The origination of lesions in the common iliac artery is unusual in that the lesion arises in the course of an artery which does not give origin to large branches (Fig. 2). In the lower limb atherosclerotic lesions arise at the bifurcation of major arteries—the aortic bifurcation; in relation to a fixed unyielding tendon—the adductor tendon; or in an artery subject to curve formation and giving off large branches—the popliteal artery (Watt, 1966); but the common iliac artery does not have any of these relationships.

In a study of blood-flow in a model constructed to resemble the aortic bifurcation Murphy *et al.* (1962) found that platelet thrombi were deposited at the bifurcation, and that the two tributaries were often free from deposits in their proximal 1 in. (2.5 cm.), with further linear deposits beyond this. This would approximate to the middle of the common iliac arteries, and could explain these lesions on the basis of alterations in blood-flow and thrombus deposition.

An alternative explanation is possible. The atherosclerotic aorta tends to become elongated, and in cases where the aortic bifurcation is relatively free of disease and not fixed it has been noted at operation that there is an up-and-down movement of the aorta with each pulsation. If one assumes that the aorta is relatively fixed above by the renal and superior mesenteric arteries, and by the origin of the internal iliac artery with its many branches, the elongated Y-shaped mobile segment would tend to buckle at the middle of the common iliac arteries, leading to stress in the arterial wall in the course of the vessel—that is, the site of origin of lesions. This stress could lead to intimal hyperplasia and eventually to plaque formation.

It is also possible that relative fixity of the common iliac bifurcation could protect the external iliac artery from undue

LEGENDS TO SPECIAL PLATE

FIG. 1.—Occlusion of left external iliac artery with reconstitution of common femoral artery. The internal iliac artery is large and the obturator artery is prominent. A stenosed right common iliac artery is visible at the top of the photograph.

FIG. 2.—Aortogram showing stenosis of the right common iliac artery with marked post-stenotic dilatation.

FIG. 3.—Gross irregularity of aorta at its bifurcation, with stenosis of the origin of both common iliac arteries and early aneurysmal dilatation of lower abdominal aorta.

FIG. 4.—Right common iliac artery is occluded and there was evidence of reconstitution of the external iliac artery on a later film. The left common iliac artery is stenosed at its origin, and this lesion is assumed to have originated at the aortic bifurcation.

FIG. 5.—Occlusion of the aorta up to level of inferior mesenteric artery, with reconstitution of right common iliac and left external iliac arteries. Pelvic collaterals derived from inferior mesenteric artery are well demonstrated. Some intramural extravasation has occurred at site of injection.

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FIG. 1

FIG. 2

FIG. 3

FIG. 5

FIG. 4

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FIG. 6

FIG. 8

FIG. 6.—Aortogram showing occlusion of left common iliac artery. There is no involvement of the origin of right common iliac artery by an aortic bifurcation lesion, and occlusion in this case has been assumed to arise from a lesion of the common iliac artery itself as in Figs. 1 and 2.

FIG. 8.—Occlusion of common femoral artery. This is limited proximally by the circumflex iliac artery and the inferior epigastric artery, which is seen to curve upwards and cross the outline of the patent external iliac artery.

FIG. 7.—Aortogram showing multiple lesions—plaque formation in right common iliac artery and at left common iliac bifurcation, stenosis of the origin of right internal iliac artery, and occlusion of part of left internal iliac artery.

J. G. McNULTY: RADIOLOGICAL DEMONSTRATION OF
RENAL PAPILLARY NECROSIS

FIG. 1

FIG. 1.—Oblique view of right kidney at urography. This shows caliceal clubbing and medullary cavities arising from their papillary ends in the upper and middle groups of calices.

FIG. 2.—Changes similar to those in Fig. 1 are seen in the left kidney, with a long ragged cavity arising from the middle calix, and smaller cavities arising from the upper and lower calices.

FIG. 2

movement and account for the relatively low incidence of severe atherosclerosis in the external iliac artery.

In the 100 aortograms studied the femoral and popliteal arteries were found to be patent in 56 (Table III). Femoro-popliteal patency in five patients with aortic occlusion could not be demonstrated by aortography, but can perhaps be assumed because aortic and femoro-popliteal occlusion combined would be more likely to produce gangrene than claudication. If this assumption were correct the distal patency rate would be 61%.

TABLE III.—Incidence of Proved Patency of Femoral and Popliteal Arteries in Relation to Principal Aorto-iliac Lesion in 100 Aortograms

Site of Occlusion	No. of Cases	Femoro-popliteal Patency	
		No.	%
Aorta	10	5	50
Aortic bifurcation stenosis	20	17	85
Common iliac stenosis	21	16	76
Common iliac occlusion	14	8	57
External iliac occlusion	14	4	29
Combined iliac occlusion	9	4	44
Common femoral occlusion	7	1	14
Others (common iliac bifurcation or internal iliac occlusion)	5	1	20
Total	100	56	56

The high patency rate of the distal arteries in aorto-iliac occlusion was first noted by De Bakey *et al.* (1958). Shepherd and Warren (1960) reported a femoro-popliteal patency rate of 59.6% in 52 cases operated on, and Singer (1963) found that the femoral arteries were occluded in only 25% of patients with aorto-iliac lesions. The low incidence of femoro-popliteal occlusion in patients with aorto-iliac occlusion is probably due to the protective effect of the lower distal arterial pressure (Singer, 1963).

In Table III femoro-popliteal patency is analysed with regard to the type of aorto-iliac occlusion present. This shows that the patency rate is high in aortic and common iliac occlusions

and low in external iliac, combined iliac, and common femoral occlusions. Indeed, associated femoro-popliteal occlusion seems to increase as the aorto-iliac occlusion becomes more distally placed in the arterial tree.

Summary

The pattern of occlusion was analysed in the aortograms of 100 patients with intermittent claudication due to aorto-iliac lesions.

Occlusions appeared to originate at three sites with approximately equal frequency: at the aortic bifurcation, in the course of the common iliac artery, and at the common iliac bifurcation.

Occlusion or stenosis of the common iliac artery was the commonest individual lesion, and the origin of lesions in the course of the common iliac artery is discussed in relation to the formation of platelet thrombi and the development of shearing strain at this site.

Femoro-popliteal patency was demonstrated in 56 cases, and the patency rate was found to be highest in the more proximal lesions.

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Guanoclor as an Antihypertensive Drug

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Guanoclor (Vatensol) is a new antihypertensive compound with the structural formula shown in Fig. 1. It is an adrenergic neurone-blocking agent, which also interferes with noradrenaline synthesis by inhibition of the enzyme dopamine beta-hydroxylase. Clinical use of the compound was first reported by Lawrie *et al.* (1964), who achieved satisfactory blood-pressure control in 60% of their cases with guanoclor alone, and in a further 18% with the addition of a thiazide diuretic. They also noted a significant reduction in urinary noradrenaline levels during guanoclor administration. This initial report was sufficiently encouraging to prompt the further studies reported here.

[(2-(2,6-dichlorophenoxy) ethyl) amino] guanidine sulphate

FIG. 1.—Structural formula of guanoclor.

A. Clinical Assessment

Method

Guanoclor has been used singly or in combination with a thiazide diuretic for the treatment of 35 hypertensive patients in regular attendance at the Dunedin Hospital hypertension clinic. Duration of treatment has varied from one month or under for six patients to 14 months or over for three patients, with an average duration of 6.2 months per patient. Clinic attendance was requested every few days at the start of treatment, and later as needed, to maintain stable blood-pressure control. At each clinic visit lying and standing blood-pressure and pulse rate were recorded at two-hourly intervals for a minimum of six hours, together with a standing blood-pressure reading immediately after exercise. Patients were questioned regularly about their health and any possible side-effects noted. In most cases urinalysis and blood cytology and chemistry were carried out before and during guanoclor therapy.

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