Disappearing Foot Pulses: *
Case Cured by Iliac Endarterectomy with Comments on Flow Mechanisms

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VARIOUS explanations have been advanced for the fact that arterial pulses may be present on one occasion and not another. It is a fact that, assuming an adequate and constant aortic blood pressure, the pulses in the feet particularly can vary in force from time to time. The reason for this may be fairly clear following arterial surgery, when the arteries may be in spasm, if no thrombosis or peripheral embolization of atherosclerotic material has occurred and the anastomosis was technically satisfactory. Arteriospasm may also be secondary to thrombophlebitis. It is also true that foot pulses completely absent preoperatively, due to lower aortic occlusive disease, may not reappear until from 24 to 72 hours after endarterectomy, resection or bypass; following this the pulsations may become temporarily so marked as to be visible from a distance, only to subside presently to an essentially normal amplitude. We have believed that this early absence of palpable pulses was due to a diminished blood-carrying capacity of contracted arteries long accustomed to a low head of pressure, perhaps abetted by a degree of vasoconstriction. The excessively forceful pulses present on the second or third postoperative day might be explained, it seemed to us, on the basis of overdistention of vessels unaccustomed to a normal head of arterial pressure and thus having subnormal tone. The return of normal tone would presumably coincide with the return to normal force of the foot pulses.[7]

And yet, the above circumstances do not apply precisely to the patient whose foot pulses are present intermittently before any operation has been performed. If this phenomenon occurs with the patient at bed rest and supine, one must assume changes in vasomotor tone or volume of blood flow. But if the pulse disappears after exercise, only to become readily palpable again following a few moments of rest, a different type of mechanism would presumably be involved.

It is of course well known that foot pulses, and doubtless the radial pulses too under proper circumstances, may disappear temporarily following exercise. However, the clinical reports that have come to our attention have varied regarding the explanation for these phenomena.[3, 5, 7, 10] The case to be presented was well documented as to the anatomic and pressure relationships, and the hypotheses that we shall offer appear to us to be reasonable ones.

Case Report

A 58-year-old white farmer was referred to the University Hospital with the complaint of intermittent claudication in the left calf for two years following vigorous exercise. Several months prior to admission he had developed marked cyanosis of the little toe and, more recently, mild cyanosis of the great toe. He had discovered that on rapid

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walking the cyanosis of the little toe gave way to a pale gray color and that the toe became numb.

Physical examination revealed a thin, wiry man who had no pertinent positive findings other than those referable to the left leg and its blood supply. The carotid, radial, abdominal aortic and right leg pulses were all within normal limits. The electrocardiogram exhibited no abnormalities.

The left leg, however, presented phenomena of much interest. The left femoral pulse was slightly weaker than the right, and there was a soft bruit and a faint thrill over the area of the left iliac artery. The left fifth toe was indeed cyanotic, the left great toe only slightly so. Both the left dorsalis pedis and posterior tibial pulses were present, though they were definitely weak. At this point the patient was asked to walk the length of the hall rapidly until pain developed in the left calf. This he obligingly did, then and a number of times subsequently for new groups of residents, internes, and students. At the time the intermittent claudication developed and he returned to his bed, the little toe was blanched white, resembling the pale "dead" finger of Raynaud's disease, and it was numb. The foot pulses were completely absent for about 60 seconds following exercise, after which they rather rapidly reappeared, and the pallor of the fifth toe gave way to the previous cyanotic state. As noted, this demonstration was repeated a number of times with different observers. The oscillometric readings in the calf reflected substantial though subnormal pulsations prior to exercise, but no oscillations were present during the period immediately following exercise when the foot pulses were temporarily absent.

An *aortogram* disclosed partial occlusion of the left common iliac artery (Fig. 1), but no occlusion of the left femoral or popliteal arteries.

At *operation* on November 15, 1961, the left iliac artery was exposed through a midline abdominal incision, and pressures were taken both above and below the point of partial obstruction. The pressure above was 120/102 mm. Hg and that below 102/62 mm. Hg (Fig. 2), as measured intra-arterially with a strain gauge. While this difference might not appear to be significant, it is to be appreciated that the internal diameter of an artery must be reduced by approximately one half before a definite pressure gradient is produced. Thus even a 20 mm. Hg difference in mean arterial pressure reflects a marked degree of arterial stenosis.

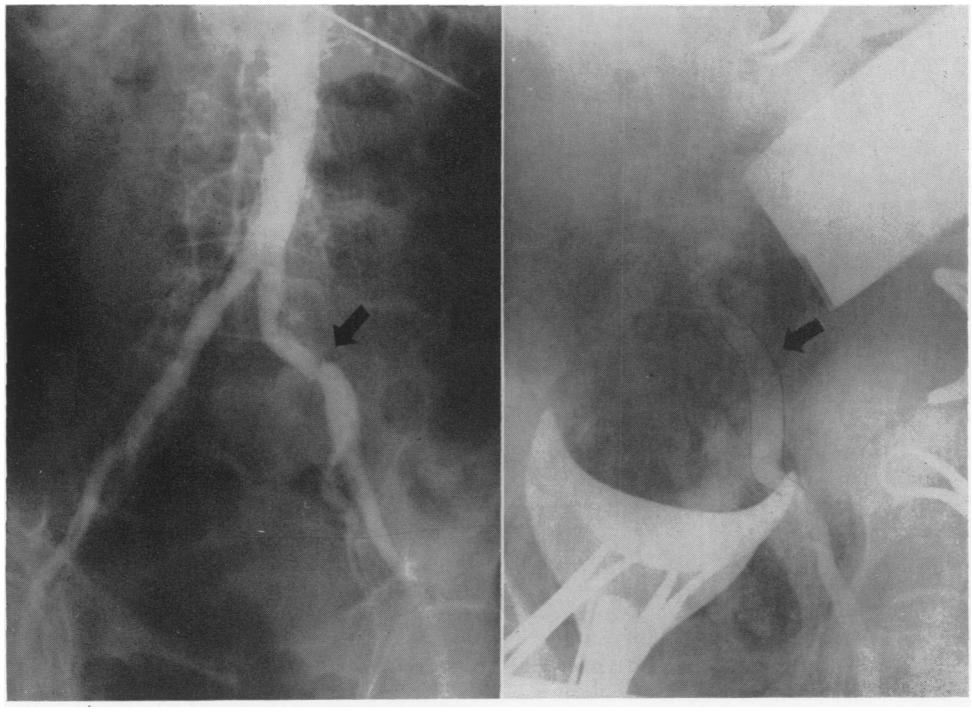

FIG. 1. *Arteriograms. Left.* Aortogram demonstrating marked stenosis of left common iliac artery with post-stenotic dilatation. *Right.* Post-endarterectomy arteriogram demonstrating restoration of normal intraluminal diameter.

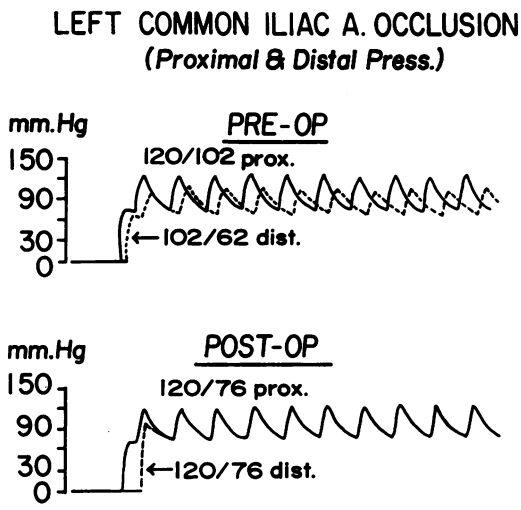

FIG. 2. *Pressure curves. Above.* The mean pressure difference of only from 20 to 30 mm. Hg indicated that the intraluminal diameter of the artery had probably been reduced by at least one half. At times clinically significant pressure differences cannot be detected by palpation alone. *Below.* Following endarterectomy the pressure curves above and below the site of previous stenosis could be superimposed. Pressure measurements at operation are very helpful, if not essential, in diagnosis and in evaluating the effectiveness of corrective operative maneuvers.

There was a palpable thrill at the point of stenosis, and post-stenotic dilatation was even more apparent than had been anticipated from the arteriogram (Fig. 1). The vessel was incised longitudinally for a distance of 2.0 cm., and an endarterectomy was performed, followed by simple closure with 4–0 silk. The intraluminal diameter of the vessel at the point of stenosis had been only about 0.3 cm., as compared with at least 1.0 cm. above and more than 1.5 cm. below at the level of the post-stenotic dilatation following endarterectomy. A repeat arteriogram revealed a lumen of normal diameter (Fig. 1) and the pressures above and below the site of endarterectomy were equal (120/76 mm. Hg, Fig. 2). The pressures in the distal aorta and the right common iliac arteries were also 120/76 mm. Hg, and no thrill was palpable in either of the renal arteries or in the superior mesenteric artery. A left lumbar sympathectomy was performed and the abdomen was closed.

The *postoperative course* was uneventful. The cyanosis of the left fifth toe had been replaced by normal color, and vigorous walking would no longer produce pallor of this digit. Similarly, the foot pulses were constantly present, even after vigorous exercise, and the intermittent claudication of the left calf had been abolished.

Discussion

It is of interest to speculate upon the mechanism by which the pulses in the left foot were made to disappear by exercise preoperatively. The explanation which appealed to us most strongly was that of an almost fixed volume of flow delivered to a vascular bed of varying size. Specifically, it was presumed and subsequently established at operation that the lumen of the left common iliac artery was rigid and markedly narrowed at the level of the atherosclerotic obstruction. Thus, when on vigorous exercise vasodilatation and perhaps the opening of arteriovenous shunts in the thigh muscles and skin occurred, the already reduced head of arterial pressure in the popliteal and tibial arteries was further diminished to a point at which the foot pulses were no longer palpable. But following a minute of complete rest, the muscle demands were abruptly reduced, the capillary bed returned to the smaller resting level, and additional flow was again made available to the more distal portion of the extremity. This hypothesis would appear to be strengthened by the fact that aortic blood pressure presumably rose during exercise.

In 1941, Leary and Allen [5] examined the possible mechanisms by which intermittent claudication develops on walking, and they made special note of those patients whose foot pulses disappeared on exercise; this circumstance had been previously noted by Thomas,[8] in 1922, and by Pearl,[6] in 1937. Leary and Allen concluded that the intermittent claudication in four patients they described was due to reduced blood flow to the extremities produced by arteriospasm. They considered this spasm inexplicable, however, noting that the usual response to exercise is arterial dilatation. Their studies of course antedated widespread aortography, though they did ascribe a bruit over the abdomen at the site of the aortic bifurcation as reflecting arterial disease, possibly

a dissecting aneurysm. In contrast to their explanation, Veal,[9] in 1936, published arteriographic evidence to support his contention that the pain of intermittent claudication was due not to arterial spasm but to organic occlusive disease. In reporting a case of disappearing foot pulses on exercise, cured by femoral endarterectomy, Jones [4] noted that the temporary obliteration of foot pulses following exercise was unaffected by paravertebral block that produced a sustained skin temperature rise in the toes. However, he still concluded that the disappearance of foot pulses, produced by running in place, was the result of arterial spasm; and that following endarterectomy a rise of arterial pressure distal to the previous point of partial femoral occlusion (from 124 to approximately 146 mm. Hg) was sufficient to prevent spasm following exercise postoperatively.

We offer an alternative and, we believe, a more probable explanation for the disappearance of foot pulses following exercise. Our point of view coincides with that of Edwards, Cohen and Kaplan.[3]

Since the point of partial atherosclerotic occlusion is rigid, one may anticipate a more or less fixed volume of blood flow through this stenotic orifice, assuming that the arterial pressure proximal to the obstruction remains essentially unchanged. If following exercise the vascular bed distal to the level of occlusion dilates, as most workers agree that it does, the fixed volume of blood delivered into the enlarged distal vascular bed would not be adequate to sustain the level of intra-arterial pressure which previously existed just below the level of atherosclerotic occlusion. With the pressure in the smaller distal vessels reduced, the degree of spasm normally supplied by the elastic fibers (tonus) might act further to reduce the size of the vessel. The law of Laplace was invoked by Burton [2] to emphasize that in small arteries, with elastic fibers, a critical level of intra-arterial pressure is required to prevent the virtual closing of these vessels. Thus any slight departure from the point of equilibrium given by Laplace's law might result in either complete closure or bursting of the vessel. It was further noted that if the pressure falls below a certain critical value, determined by the unstretched radius of the vessel and vasomotor tone, complete closure and cessation of flow must result.

Additional argument in favor of the increased bed—reduced pressure explanation for the disappearance of foot pulses in our patient has been forthcoming in the report of Blaisdell and Gauder.[1] They showed experimentally that partial occlusion of the common iliac artery in the dog resulted in reduced pressure amplitude in the ipsilateral common femoral artery; but that a normal pressure curve was restored in the common femoral artery if a second clamp was placed across the superficial femoral artery just distal to the origin of the profunda femoris artery. This distal clamp of course converted an open system into an essentially closed one, invoking the pressure postulates of Pascal's Law, i.e., that the pressure exerted anywhere on a confined liquid is exerted equally in all directions. The effect of the second or distal clamp was to reduce the vascular bed which the reduced volume of flow through the stenosed iliac artery had to fill. Incidentally, the purpose of their study was to demonstrate that the presence of normal pressure in the common femoral artery above a more distal point of occlusion does not necessarily exclude occlusive arterial disease above the level of the inguinal ligament; that successful endarterectomy or bypass in the leg may not restore adequate blood flow to the extremity if more proximal partial occlusion, previously unsuspected because of a normal pressure in the common femoral produced by distal femoral occlusion, is not also corrected.

The clinical studies of Edwards and his

associates [3] also supported the concept of a disparity between the volume of blood flow and the size of the distal vascular bed, and they refuted the suggestion that spasm plays a major role in the disappearance of foot pulses following exercise. Using pressure measurements, these workers demonstrated to their satisfaction that the disappearance of foot pulses on exercise was due to the shift of blood away from the foot and toes during exercise. Thus they advised caution, as had others on the basis of similar studies,[10] in the use of exercise for the ischemic foot.

Summary and Conclusions

The intermittent presence of arterial pulses, particularly in an extremity, may be due to a variety of factors, including atherosclerotic occlusive disease, spasm or embolism.

A case is reported in which the pulses in the left foot regularly disappeared for a brief period following vigorous exercise. During this period the left fifth toe became white from ischemia and numb, and no oscillometric readings were present at the calf level. All abnormal findings were abolished by left iliac endarterectomy. Mechanisms of arterial flow are discussed.

It is concluded that the foot pulses disappeared when the fixed volume of flow through the stenotic iliac artery could no longer distend the distal arterial bed which had been dilated by exercise.

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