

Anterior Tibial Muscle Compartment Claudication with Incomplete Arterial Occlusion *

A Case Report

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THE PAIN of claudication in skeletal muscle and of angina in the myocardium is due to the reduction of the supply of oxygen to the muscle or an increase in the demand for oxygen by the muscle or both. There is a vasodilator reflex that accompanies increased muscular activity and usually compensates for this increased demand for oxygen by the contracting muscle. It has been difficult to understand a mechanism for the production of claudication secondary to vasospasm alone because of this reflex vasodilation.²⁻⁴ Claudication has therefore been thought to be indicative of underlying arterial disease with a narrowing of the arterial inner diameter; a reduction in the peripheral pressure, and from these a reduction in all important blood flow to the periphery. This case emphasizes the prime importance that peripheral blood pressure plays in the maintenance of arterial blood flow through small arteries. The diagnosis of claudication was confirmed by a functional test in which the usual vaso-regulatory mechanisms did not maintain adequate blood flow with increased muscle activity. In fact a marked spasm was produced by the test. The therapy undertaken was a direct clinical application in support of Burton's theory of the critical closing pressure in relation to peripheral blood flow.

Case Report

The patient, a 43-year-old taxicab driver, has been followed in various clinics since the age of 17. He was seen in the Emergency Department following three months of typical intermittent claudication of the left anterior tibial muscles. The pain was preceded by a tightening of these muscles and was relieved after three to four minutes of rest. While it was usually limited to the anterior tibial muscle group he had, on occasion, experienced both left and right calf claudication. The severity of the pain had been increasing.

On examination he was a healthy anxious man with vital signs within normal limits; a blood pressure of 140/75 and an 80 pulse. The palpable peripheral pulses were all present though slightly reduced in the left foot. Oscillometry revealed a reduction in the amplitude of the pulsations in the left leg.

Preop.	Oscillometry (pressure 120)		Pulses	
	R	L	R	L
Upper thigh	3	2	Femoral	3+ 3+
Lower thigh	3.5	1	Popliteal	3+ 3+
Calf	3	0.5	Dor. ped.	3+ 2+
Ankle	2	0.3	Post. tib.	3+ 2+

On repeated testing he would develop tightening of the anterior tibial muscles of the left leg after running in place (at a rate of 3 steps per second) for 45 to 65 seconds. This would then be followed by severe claudication, marked pallor and loss of both dorsalis pedis and posterior tibial pulses. This pallor and pain would clear in one to six minutes. Smoking a cigarette had no effect on the rapidity of onset or on the severity of the pain, nor did a paravertebral block of the first and second lumbar sympathetic nerve ganglia. There was a marked rise in the skin temperature of toes

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FIG. 1. Pre- and postoperative arteriograms. Note the three vessel run off in the postoperative film.

accompanied by a diffuse flush and warming, after the block.

Toe.....	I	II	III	IV	V	
Pre-block	23	23	23	23	23	
Post-block	29	31	31.5	32	31	Degrees centigrade

Arteriogram showed an incomplete superficial femoral block at the adductor hiatus and common femoral spasm at the site of the needle puncture.

Under general anesthesia the adductor canal was opened and an endarterectomy was performed with a Cannon stripping loop. Before the endarterectomy the mean intraarterial blood pressure above the block, measured by direct cannulation was 146 mm. of mercury. Below the block it was measured by the same method at 124 mm. of mercury. Post-operatively he was treated with intravenous Sodium Heparin for 48 hours and with appropriate Dicumarol for 14 days to maintain a near 20% pro-

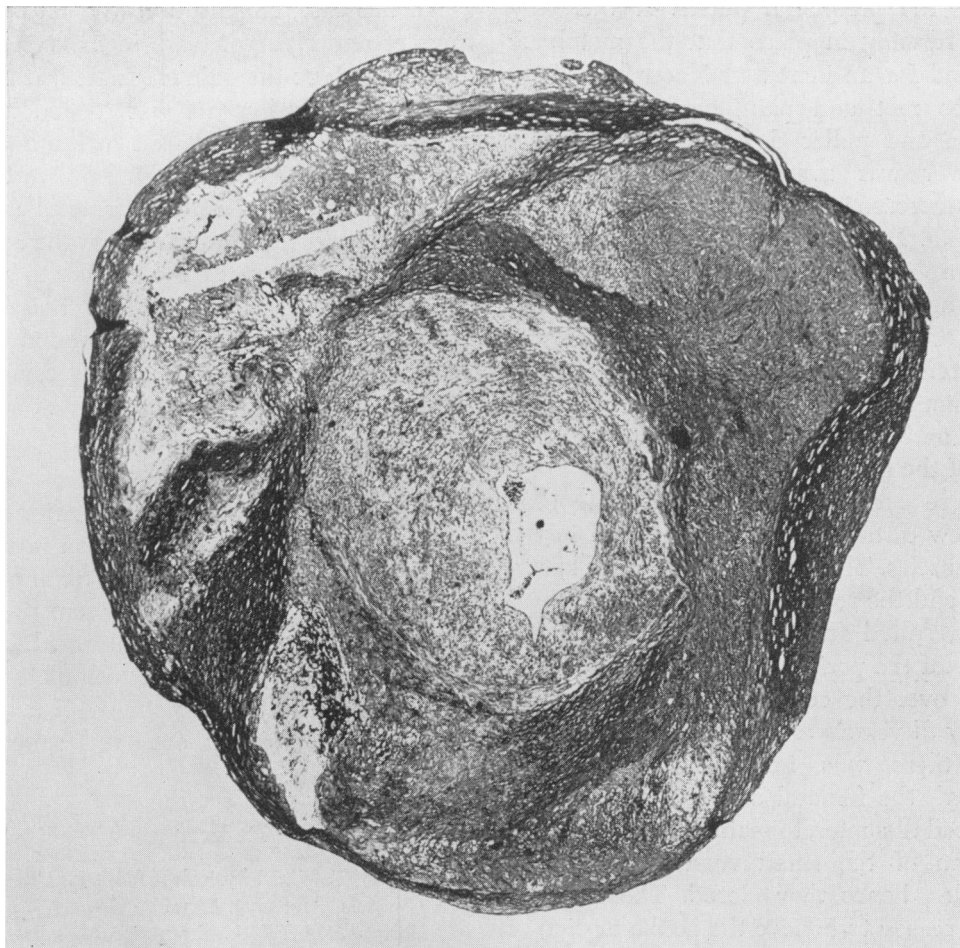


FIG. 2. Photomicrograph of the thrombosed atheroma that was removed by endarterectomy. The lumen is incompletely occluded. Elastic tissue in black.

thrombin time. Six hours after endarterectomy the oscillometric readings were improved.

Postop.	Pressure 120			Pulses	
	R	L		R	L
Calf	3	2	Femoral	3+	3+
Ankle	2.5	1.5	Popliteal	5+	3+
			Dors. ped.	3+	3+
			Post. tib.	3+	3+

He was discharged eight days after operation and returned to clinic one week later. At that time the oscillometric readings were equal in both ankles with two units of oscillation. Postoperative arteriograms revealed a patent arterial system without femoral artery narrowing and a good three vessel distal runoff (Fig. 1). On his four week clinic visit he was able to run in place under the same conditions as preoperatively for five min-

utes without developing calf claudication or muscle tightening. The test was stopped because of claudication in the opposite leg.

Discussion

The arterial spasm which developed after muscular activity associated with claudication was of a marked degree. It obliterated both the dorsalis pedis and the posterior tibial pulses and was not reduced by paravertebral block that produced a sustained skin temperature rise in the toes. The importance of typical claudication in spite of the pre-exercise presence of good peripheral pulse should be emphasized. To palpation there was little difference be-

tween the peripheral pulses. It was only after running in place that the metabolic demand for an increased blood flow could not be met and pain, occlusive arterial spasm, and pallor developed. The spasm however was overcome by a relatively small increase in the arterial pressure (from 124 mm. Hg to about 146 mm. Hg). This pressure increase was accomplished by increasing the inside diameter of a short segment of incompletely occluded distal superficial femoral artery (Fig. 2). The operation was limited to the femoral artery in the thigh and did not directly involve the vessels of the calf muscles.

There certainly was not adequate time for new distribution vessels to form in the leg muscles. This immediate relief of symptoms and the restoration of adequate blood flow resulted from elevation and maintenance of the peripheral blood pressure to a point over the critical closing pressure of the small vessels of the leg. Prior to operation there was insufficient blood flow through the femoral artery, because of its reduced diameter, to maintain a stable equilibrium of the small vessels of the calf muscles, hence they closed. There is also good reason to believe the active tension of this patient's blood vessels (the vasoconstrictor activity) is high. His critical closing pressure is therefore likely to be high. This has been observed by Burton in his exposition of the importance of LaPlace's law as related to peripheral blood flow.¹

In this case, claudication was not indicative of underlying disease in the vessels of the calf where the pain originated. It seems likely that the hypoxia was related to the closure of the small vessels in the muscles secondary to a reduced blood pressure distal to a partially occluded femoral artery. There is evidence to suggest that the critical closing pressure was elevated due to an increased active arterial tension. This vasoconstriction was overcome with an increase in the distal arterial pressure by removing a partial segmental constriction.

Summary

A case is presented with anterior tibial compartment muscle claudication in which both dorsal pedis and posterior tibial pulses, while on initial examination were present, after exercise were both obliterated. These signs and symptoms were completely relieved by an endarterectomy of a short segment of partially occluded superficial femoral artery.

References

1. Burton, A. C.: Physical Equilibrium of Blood Vessels. *Am. of J. Physio.*, **164**:319, 1951.
2. Horton, B. F.: Intermittent Claudication in the Extremities with Pulsating Vessels. *Medical Clinics of N. Am.*, **14**:783, 1930.
3. Leary, W. V.: Intermittent Claudication as a Result of Arterial Spasm Induced by Walking. *Am. Heart J.* **22**:719, 1941.
4. Veal, J. R.: Vascular Changes in Intermittent Claudication. *Am. J. Med. Sc.*, **192**:113, 1936.