evidence of clearance of gross obstruction to blood flow either proximal or distal to the site of reconstruction.

In recent years we have increasingly operated on the deep femoral (profunda femoris) artery for atherosclerotic disease mainly sited below the level of the inguinal ligament by the operation we have named extended deep femoral angioplasty (Berguer *et al.* 1973). In addition we have used a variety of methods to improve flow in the deep femoral artery: aorta to deep femoral endarterectomy; aorta to deep femoral bypass; femoro-deep femoral cross-over bypass; and short vein bypass from the common femoral to the distal deep femoral arteries (Berguer & Cotton 1973, Berguer *et al.* 1973).

Measurement of arterial blood flow showed that extended deep femoral angioplasty increased mean flow from 106 to 163 ml/min, and papaverine injection raised the flow to 332 ml/min before reconstruction and 501 ml/min following reconstruction (Cotton *et al.* 1972).

We had hoped that flow metering might be of help in deciding what operation should be done for arterial disease below the level of the inguinal ligament where the choice is often between femoropopliteal bypass, extended deep femoral angioplasty, lumbar sympathectomy or even amputation. This hope has not been justified. No evidence has been obtained from examination of the flow records by which the success or failure of the operation might have been forecast.

The greatest value of flow recording has been in detecting potential technical failure during arterial reconstruction due to thrombus formation or intimal flap dissection. Either may be minute in amount yet detectable by flow metering and correctable before the patient leaves the operating table. Not only is it important to increase arterial blood flow after operation but also to 'normalize' the flow wave form. A good wave form has a rapidly rising and equally rapidly falling peak with a wide amplitude indicating that flow is free of obstruction certainly near the site of reconstruction. We leave the probe on the reconstructed artery for 20 minutes and watch for signs of deterioration such as flattening of the wave form. We find faults requiring reconstruction in about 11% of operations. It is very rare that a patient needs to be brought back to the operating theatre in the postoperative period.

In conclusion, flow metering during arterial reconstruction tells the surgeon how much his reconstruction has improved blood flow and also almost infallibly picks up minor defects, that could later cause disaster, at a time when they can easily be corrected.

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## Importance of Blood Viscosity. Rheological Claudication

There is a group of patients with the symptoms of intermittent claudication in whom the principal cause of circulatory insufficiency is an abnormally high blood viscosity rather than narrowing of the arteries. We suggest the term 'rheological claudication' to describe this group of cases.

We measured the whole blood viscosity of an unselected group of intermittent claudicants at four shear rates (from 230 to 0.77 $s^{-1}$). At all these shear rates the viscosity was significantly higher ($P<0.001$) than that of a normal age-matched control group. The difference was greatest

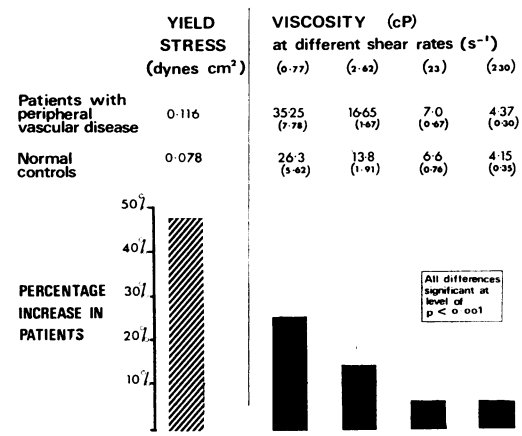


Fig 1 *Whole blood viscosity at 4 shear rates and yieldstress in 120 patients with intermittent claudication compared to a matched normal control group*

(+22%) at the lowest shear rate. The difference in yield stress, which is the minimum force necessary to initiate flow, was even greater, almost 50% (Fig 1). Although these results were obtained using all intermittent claudicants almost two-thirds of them had a viscosity within the normal range and the very high blood viscosities were only found in a third of the cases.

There were a number of differences between patients with intermittent claudication who had a high blood viscosity and those who did not. Many of the patients with abnormally high blood viscosity had a surprisingly normal arteriogram despite severe symptoms of claudication. They also tended to have more severe symptoms; for instance patients with a blood viscosity above 4.5 cP (at a shear rate of 230 $s^{-1}$) had an average claudication distance of 138 yards (126 m) compared to 316 yards (289 m) for the patients with a blood viscosity below that level. Their prognosis was also different. Those with an initially high blood viscosity had a significantly worse prognosis than claudicants with a normal viscosity.

These results were found in a group of intermittent claudicants who had not received any form of therapy. It is impossible to draw statistically significant conclusions in patients who had undergone reconstructive arterial surgery because of the difficulty of comparing operations. However, patients with a normal blood viscosity and a purely arterial lesion were much better a year after reconstructive surgery than patients with a mixture of arterial narrowing and high blood viscosity.

The basic cause of the high blood viscosity in these patients is now known. It is not due to a difference in hæmatocrit, which is the most critical determinant of viscosity we know. There was, however, a significant correlation between high blood viscosity and plasma fibrinogen (and we believe this to be a far more important biochemical abnormality in patients with circulatory disease than any change in plasma lipid composition).

In summary, we suggest the term 'rheological claudication' to describe those patients with intermittent claudication who have an abnormally high blood viscosity. This is present in approximately 25–30% of all moderate to severe claudicants. In some cases an abnormal blood viscosity may represent the principal etiological factor, while in others it may coexist with some degree of arterial narrowing. We believe it is important to recognize this group of rheological claudicants because their prognosis is significantly worse' and reconstructive surgery is contraindicated. Finally, treatment in these cases should be primarily directed towards correcting their rheological rather than their arterial defect.

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**Arterial Assessment by Doppler-shift Ultrasound**

It has been shown that Doppler-shifted ultrasound signals can be obtained easily and transcutaneously from many blood vessels in both normal and atherosclerotic subjects (Rushmer *et al.* 1966, Strandness 1969, Kalmanson *et al.* 1968, Gosling *et al.* 1969). Using spectral analysis these Doppler signals present a distribution of frequencies (sonagrams) the envelope of which has a waveform (cardiac period) characteristic of the vessel site in normal subjects (Gosling & King 1974). Disease in the arterial system changes the shape of these 'normal' waveforms. A simple system of quantifying such changes is offered here.

When a specific vessel pathway is defined by using two Doppler probes simultaneously, proximal and distal to the pathway, the sonagrams of both signals can be displayed and read out simultaneously or analysed from tape afterwards (Coghlan *et al.* 1974). Three parameters may be measured from the two simultaneously displayed sonagram waveforms: pulsatility index (PI), damping factor ($\Delta$) and transit time (T). Normal values have been established and departure from these indicates the presence of lesions in the defined pathway (Gosling *et al.* 1971, Fitzgerald *et al.* 1971, Woodcock *et al.* 1972).

*Calculation of Key Parameters*
*Pulsatility index* (PI)=(Peak to peak height of sonagram waveform)/(mean height over one cardiac cycle). PI value is therefore independent of probe angle to vessel, carrier frequency and velocity of sound in tissues traversed.
*Damping factor* ($\Delta$)=(PI proximal)/(PI distal). Like PI, this is independent of probe/vessel factors.
*Transit time* (T)=(Foot to foot distance between displayed waveforms)/(time base calibration of display). Usually expressed in milliseconds.