BRITISH MEDICAL JOURNAL

LONDON SATURDAY 16 APRIL 1966

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Treatment of Claudication

Intermittent claudication, or limping brought on by ischaemic pain, is almost invariably due to a severe reduction of blood flow along the main arteries to the legs, the result of complete occlusion or gross narrowing of the aorta or the iliac, femoral, popliteal, or tibial arteries. These are often affected in several places. When the narrowing is slowly progressive, a collateral circulation develops synchronously and the symptoms of claudication creep on the patient. But a thrombosis may occur suddenly in a relatively unobstructed vessel, and the moment may be fixed in the patient's mind as his leg becomes cold and his walking slows to a hobble or a halt.

If the condition has come on slowly and a collateral circulation is already well developed when the patient first presents himself for treatment, little spontaneous improvement is likely. But if occlusion is sudden in a vessel which has been previously transmitting a large flow of blood, provided the onset of ischaemia has not been so severe as to lead to gangrene, compensation can be expected by the development of a collateral circulation over the next few months. If gangrene has not occurred, improvement is spontaneous whether the patient takes a vasodilator drug or not. Indeed this spontaneous improvement must have made the reputation of many such drugs. When intermittent claudication is the presenting symptom and the foot is not severely ischaemic, long follow-up of patients has shown that only 10% come to amputation, and associated hypertension seems to halve this figure. If pain at rest is the first complaint the limb stands a chance of over 50% of being amputated.[1]

The patient's history then is an important guide to prognosis. Clinical examination will usually locate the site of the occlusion. The presence of an abdominal bruit together with a bruit and a weak or even absent pulse over the common femoral artery on one or both sides indicates disease of the aorto-iliac system. Weak or absent popliteal pulses in the upper or lower parts of the popliteal fossa, with normal pulses proximally, show that the occlusion is in the superficial femoral or popliteal arteries. Further information can be gained from palpation of the ankle pulses. The effect on the limb of reduced flow of blood will be reflected in its general condition, and the finding of evidence of vascular or other disease elsewhere in the patient is of great importance.

What is to be done, if anything ? Obviously if the leg is severely ischaemic and the patient suffers pain at rest, or if pregangrenous changes have developed, immediate investigation and treatment are indicated. But the claudicant patient whose exercise tolerance has stabilized and whose limb is not grossly ischaemic presents a more difficult problem. Although in theory there seems to be no reason why all those with claudication should not be investigated with a view to surgical treatment, the large number of patients presenting makes some form of selection necessary. For the older patient whose handicap is not great and the occlusive disease apparently static, surgery is not justified. But many vascular surgeons would consider suitable for treatment those patients who are

economically handicapped by their low exercise tolerance and those, still relatively young, whose activities have been cut off in full bloom. Even so, it is still not possible to judge the rate of progress of arteriosclerotic disease, and sometimes after extensive reconstructive arterial surgery the patient returns home to die all too soon from a coronary thrombosis or a cerebrovascular accident. Better methods of selection are clearly required.

The conservative treatment of claudication by means of vasodilator drugs is of little value,[2] and even the theoretically attractive drug isoxsuprine[3] has not yet justified itself. On the other hand, arterial surgery, whose successful outcome is to increase the flow of blood through the limb to nearly normal, may have much to offer.

When the patient is admitted to hospital adequate arteriography is needed in addition to a general assessment to show where the arterial disease is located and how extensive it is. Disease may be so widespread and severe as to preclude the possibility of a direct approach to the arterial tree, and the only possible surgical alternative so far has been lumbar sympathectomy. In the *B.M.J.* last week (page 879) and this week (page 943) Mr. K. A. Myers and Professor W. T. Irvine report an objective reassessment of what this operation has to offer the patient with an ischaemic leg. Their findings lend weight to the conclusion that sympathectomy gives virtually no benefit to the patient complaining only of intermittent claudication. But it often helps by increasing the flow of blood to the foot, provided this is more than minimal before operation.

Often the disease is not too extensive to preclude a direct attempt to open up the main artery by endarterectomy (disobliteration) or to insert a by-pass graft, which amounts to the insertion of a wide-bore collateral artery. Both endarterectomy and by-pass grafting can be carried out from the aorta to the popliteal artery or, less usefully for the claudicant patient, to the profunda femoris artery.[4] For success both techniques require an adequate outflow of blood peripherally so that there is a brisk flow along the graft or cleared artery. Grafts used below the inguinal ligament are now almost always taken from the patient's own veins, since arteries from another patient become aneurysmal and cloth prostheses soon undergo thrombosis. For the claudicant patient the blood is best led into the lower popliteal artery, which for some reason is frequently less affected by arteriosclerosis.[5] Whether grafting or disobliteration is employed below the inguinal ligament, it is essential to ensure that the artery is not occluded higher up. If it is, it must be treated before or at the same time as the more peripheral block.

The results are good and still improving with more advanced techniques and better selection. Patients usually lose their claudication and any ischaemic skin lesions heal. The new channel may remain patent for many years. The operation may fail for technical reasons, or because the arteriosclerosis progresses, or for other reasons which are not always clear. Diabetes worsens the prognosis. Long follow-up shows that vein by-pass grafts give better results than endarterectomy.[6] Recently successful attempts have been made to reopen occluded lengths of artery by percutaneous intra-arterial bougienage under local anaesthesia.[7] This method of treatment certainly requires assessment, particularly for the poor-risk patient. The passing of sympathectomy as a primary operation for claudication will not be regretted by the vascular surgeon, who has now several highly effective alternative techniques. Nevertheless there is still much room for improvement.

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3 Hyman, C., and Winsor, T., *Acta Pharmacol. (Kbh.)*, 1960, **17**, 59.
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5 Singer, A., *ibid.*, 1963, **87**, 384.
6 De Weese, J. A., Barner, H. B., Mahoney, E. B., and Rob, C. G., *Ann. Surg.*, 1966, **163**, 205.
7 Dow, J., and Hardwick, C., *Lancet*, 1966, **1**, 73.

Detecting Foetal Abnormalities

If the law of abortion is to be extended and not merely clarified, one proposal that has found more favour than most is to allow termination of pregnancy when the foetus runs a high probability of being defective. Three problems immediately arise: How severe a defect? What degree of probability? How is this to be ascertained in the individual case? Lord Brain[1] recently indicated the complexities of these questions. The report of the Council of the Royal College of Obstetricians and Gynaecologists[2] stated that the great majority of gynaecologists would find it possible to work to an Act which permitted abortion if the continuance of pregnancy would "involve substantial risk that the child if born would suffer from such physical or mental abnormalities as to deprive it of any prospect of reasonable enjoyment of life." Some recently developed techniques may be of help in certain circumstances, enabling the woman's medical attendant to say that the foetus is virtually certain to be defective.

Ten years ago workers[3-5] in Britain and Denmark showed that the sex of a foetus could sometimes be determined before birth from examination of the sex chromatin in the nuclei of cells in the liquor amnii. Recently M. W. Steele and W. R. Breg, jun.,[6] in the U.S.A. have extended the technique and found that it is possible to analyse the chromosomes in some cells from amniotic fluid after growing them in culture media. Their specimens of amniotic fluid were obtained by abdominal amniocentesis at 20–37 weeks' gestation. Prediction of foetal sex was accurate in all 21 cases they had examined, apart from a further one in which confirmation was impossible. Thus, in the case of the child of a male haemophiliac determination of the foetal sex could be used to establish whether the foetus was a carrier of the disease. The ability to study the chromosomes by this technique suggests that it should also be possible to forecast whether a baby will be affected by one of the syndromes, such as mongolism, known to be associated with abnormalities in structure or number of the chromosomes. One immediate problem to be solved would seem to be how to arrive at a cytological diagnosis much earlier in pregnancy than has yet been achieved.

Certainly the indications for abortion based on such relatively exact data are stronger than those sometimes advocated at present, such as rubella in the first three months of pregnancy or mental defect in the parents. Though the proportion of cases of congenital defect in which a cytological diagnosis could be obtained from foetal cells is small, the diagnosis, when made, would be reliable. However, the technique for culturing such cells must be regarded as still in an early exploratory stage.

1 Brain, Lord, *Brit. med. J.*, 1966, **1**, 727.
2 Council of the Royal College of Obstetricians and Gynaecologists, ibid., 1966, **1**, 850.
3 James, F., *Lancet*, 1956, **1**, 202.
4 Fuchs, F., and Riis, P., *Nature (Lond.)*, 1956, **177**, 330.
5 Dewhurst, C. J., *Lancet*, 1956, **1**, 471.
6 Steele, M. W., and Breg, W. R., jun., ibid., 1966, **1**, 383.