More recently Weber[2] has published the results of a trial of traction in 72 patients, which showed no advantage for this form of treatment in patients with lumbago and sciatica. The control procedure used was homoeopathic traction; the traction used for treatment was given on a split couch with a force of one-third of the body weight for 20 minutes at a time, but in a "pulsed" manner (with pauses) each day for five to seven days. Double-blind conditions were achieved, but it should be pointed out that there is no evidence of the actual effect on the lumbar spine of traction given in this manner.

Mathews[3] showed by a contrast radiographic technique that traction of about 100 lb (45 kg) on conventional couches can reduce the height of disc bulges, apparently by distracting vertebrae. This traction technique has now been used in a small but carefully controlled double-blind study[4] of the treatment of 27 patients with lumbago with sciatica but no neurological deficit. Again, no advantage was shown from traction. What may be important is that a few patients, some of whom had previously been deteriorating, improved with impressive speed when crossed over from placebo to effective traction. This phenomenon is possibly comparable with that observed in the multicentre study of manipulation in the treatment of low back pain organised by the British Association for Rheumatology and Rehabilitation,[5] in which a small subgroup of patients with low back pain who were subjected to manipulation also improved rapidly. In neither instance were the results in these small subgroups statistically significant, nor was any method of identifying these patients in advance apparent. Sceptics hold the view that these and other unproved forms of physiotherapy should be discontinued forthwith. An alternative and more positive approach is to build on current experience to mount bigger and better trials.

[1] Christie, B G B, *Proceedings of the Royal Society of Medicine*, 1955, **48**, 811.
[2] Weber, H, *Journal of the Oslo City Hospital*, 1973, **23**, 167.
[3] Mathews, J A, *Annals of Physical Medicine*, 1968, **9**, 275.
[4] Mathews, J A, and Hickling, J, *Rheumatology and Rehabilitation*, 1975, **14**, 222.
[5] Doran, D M L, and Newell, D J, *British Medical Journal*, 1975, **2**, 161.

Help for the left ventricle

In some circumstances a left ventricle whose muscle has become irretrievably and permanently damaged may be replaced by a whole heart transplant[1] or reinforced by joining to a whole heart implant.[2] In the future it may be possible even to exchange it for a mechanical heart substitute.[3] For the left ventricle which becomes temporarily inadequate there are now short-term means to support life until the muscle recovers, and these techniques need to be considered when the cardiac output is low after open heart surgery or myocardial infarction but before the stage of irreversible cardiogenic shock occurs. Several ingenious mechanical devices for whole or partial left ventricular bypass have been described, designed to promote a more normal cardiac output; to lower pressure in the left atrium, thereby relieving pulmonary congestion; to aid left ventricular recovery by decompressing it; and to ensure an optimal cardiac rate, so making best use of the available stroke volume.

Already in fairly widespread use, but able to offer more limited help to the failing left ventricle, is the intra-aortic balloon pump (IABP), which uses the concept of counterpulsation.[4] In the early systems an external mechanically operated syringe removed blood rapidly during left ventricular systole and reinfused the same blood during diastole. The idea was to reduce left ventricular pressure work in systole and to improve coronary blood flow in diastole. Unfortunately, the suction and turbulence caused considerable haemolysis, and sufficient blood could be moved only when bilateral femoral artery cut-downs were used. Furthermore, when counterpulsation was operated as far away from the heart as the femoral artery the diastolic input failed to get back to the coronary arteries. Three years later Moulopoulos suggested placing the device in the descending thoracic aorta; he used a balloon which was inflated in diastole as the aortic valve closed and deflated rapidly at the onset of ventricular systole.[5] Balloon inflation displaced blood towards the heart and coronary arteries and collapse of the balloon created a "sink" which reduced left ventricular work. In 1968 Kantrowitz reported the first successful clinical application of such a system.[6] Since then further technological advances have been made and the complications have diminished. A single femoral arteriotomy is needed; there is only minimal haemolysis; and left ventricular help may be continued for longer.

IABP augments coronary diastolic blood flow,[7] and in 1971 the original Kantrowitz design was modified by introducing a dual chamber intra-aortic balloon,[8] the distal of the two balloons being used to occlude the aorta, thus ensuring that diastolic blood displacement is wholly centripetal. This is more effective in increasing coronary blood flow than the single chamber balloon, which displaces blood in all directions. Deflation is triggered by the R wave of the electrocardiogram, so that the balloon is never inflated during left ventricular contraction. If the patient's heart rate is suboptimal or there are dysrhythmias a pacemaker may be used to trigger the machine. The balloon is inflated with carbon dioxide so that if it ruptures it would not endanger life; deflation is achieved by applying a vacuum. These machines may be made operated by batteries and hence are portable. Though its theoretical rationale is impeccable, IABP has been slow in development and adoption, probably because of the difficulty or impossibility of designing prospective controlled trials to show whether or not it improves survival.[9] Perhaps, too, the innate conservatism of the cardiologists has made them lag behind the surgeons, for whom the concept of IABP holds fewer terrors.

Limitation of the size of an infarct is a main objective of research in coronary care and the aim of every physician concerned in it. Infarcts do not happen all at once but may advance over many hours, during which a variable amount of myocardial muscle may be perilously ischaemic with its fate hanging in the balance. Prolonged pain after infarction, extensive ST segment elevation, and continuing high plasma creatine kinase levels all indicate massive enzyme leakage from dying myocardial cells and provide grim warning signals. Vasodilator treatment may do good at this stage because a reduction in left ventricular pressure-work may decompress the left ventricle and permit more effective contraction, with a rise in stroke output but a fall in metabolic demand. When the vasodilator treatment fails, however, the blood pressure and life-sustaining coronary blood flow both fall, as do the cerebral and renal flows. Seemingly paradoxically, though peripheral vasoconstrictors (long out of vogue) increase blood pressure and metabolic demand, their use may yet improve left ventricular performance and reduce infarct size through an increase in coronary blood flow—and especially the flow through partially obstructed coronary arteries.

Beta-adrenergic receptor blockade provides a third way of possibly limiting the size of the infarct size, which also works well in animals. These drugs achieve their beneficial effect by counteracting the oxygen-wasting effects of excessive medullary

and cardiac catecholamine release, so lessening myocardial oxygen demand, reducing the overdraft, and improving true efficiency. After treatment with a beta-blocker the left ventricle beats more slowly; diastolic coronary perfusion time is increased; and the velocity of contraction is reduced. Unfortunately, slowing the heart rate will reduce minute output if the stroke volume fails to increase. In the patient who has had an infarct and is sick or deteriorating IABP combines the virtues of the three different classes of drug by aiding delivery of blood to the tissues, improving coronary perfusion, and reducing myocardial oxygen demand.[10-14] No single drug or combination of drugs fulfils all three objectives.

IABP should, then, reduce final infarct size provided pumping is started as soon as possible after the onset of symptoms of infarction. It is no good starting IABP after cardiogenic shock has developed, for by this time usually too many myocardial cells have died to permit survival.[9] Furthermore, ischaemic but still viable myocardial cells will not contract, even though they can still recover if their blood supply is enhanced. If after 48 hours of IABP the patient with an infarct is still dependent on it, then the prognosis becomes extremely grave. One possibility under discussion at present is the use of IABP to help the patient through surgery for emergency coronary artery bypass grafting, so revascularising the ischaemic halo surrounding an infarct, but this procedure carries the hazard of possible haemorrhagic infarction of the already necrotic territory.

The value of IABP in helping a high-risk patient through cardiac surgery or in tiding him through a period of unsatisfactory low output postoperatively is much more generally agreed. Reversible myocardial cell injury is an unwelcome but still partially unavoidable concomitant of open heart surgery, and while this left ventricular disability is temporary it may tip the balance against survival in a minority of patients.

In common with the totally artificial heart, left ventricular bypass pumps have failed to graduate into clinical use because their development has been hampered by problems of thromboembolism or bleeding associated with the necessary anticoagulant regimen as well as with destruction of blood cells by the pump. In a recent article Bernstein *et al* have described a new compact centrifugal blood pump system for temporary left ventricular bypass.[15] No thoracotomy is required, access to the left ventricle being obtained through a thin-walled flexible non-kinking cannula, whose tip is introduced into an extrathoracic artery and then advanced retrogradely into the left ventricle. The outflow cannula of the pump is inserted into another artery, and the pump is then allowed to propel blood from the cavity of the left ventricle to the external artery. Though still in the research stage, this mechanical means of temporary whole or partial left ventricular bypass represents a further advance towards a system which can be applied with relative ease and safety.

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[13] Dilley, R B, Ross, J jun, and Bernstein, E F, *Circulation*, 1973, **47**, Suppl III, 99.
[14] Leinbach, R C, *et al*, *Circulation*, 1973, **48**, Suppl IV, 100.
[15] Bernstein, E F, *et al*, *Annals of Surgery*, 1975, **181**, 412.

Antibiotic treatment in kidneys of unequal function

The bacteria most often found in urinary tract infections originate in the rectum or on the surface of the perineum and enter the urinary tract through the urethra. Once in the bladder, they multiply in the urine (which is a good culture medium) and may then ascend the urinary tract to infect the kidneys. In some cases this ascending infection may be helped by the presence of vesicoureteric reflux. Haematogenous infections of the kidney are rare. This concept of how most urinary tract infections arise is now generally agreed, and it has resulted in a consensus on the principles of management: elimination of bacteria from the urine in the bladder by the use of antibiotics achieving high concentrations in the urine; the maintenance of a high urine flow; and frequent emptying of the bladder. Few would argue with this approach when lower urinary tract infections are being treated, and then tissue levels of antibiotics are probably unimportant, so that use may be made of urinary antiseptics such as nitrofurantoin and nalidixic acid, which achieve high urine concentrations but low tissue levels.

When, on the other hand, there is evidence of infection of the kidney itself with systemic signs of infection and kidney pain or tenderness, there are attendant risks of Gram-negative bacteraemia or septicaemia. Antibiotic treatment should then be directed to the eradication of bacteria from the kidney by using antibiotics which achieve effective levels in the tissues as well as in the urine.[1] Nitrofurantoin and nalidixic acid are inappropriate in these cases, and antibiotics such as the penicillins, the cephalosporins, and the aminoglycosides should be used. Nevertheless, in treating relapsing infections (in which it is implied that organisms persist in the kidney tissue) Williams *et al*[2] found no evidence that ampicillin was any better than nitrofurantoin.

The dosage of antibiotics may have to be adjusted in patients with renal failure so that effective serum levels are achieved without causing accumulation of the drug, which could lead to toxic effects. In addition, adequate concentrations must be achieved in the urine. Nitrofurantoin is contraindicated in renal failure because inadequate urine concentrations are achieved[3] and more importantly because of the dangers of toxic effects (particularly polyneuropathy) which occur in patients with renal failure.[4 5]

Sullivan *et al*[6] have recently studied the urinary concentrations of nitrofurantoin, sulfamethizole, and cephalexin in patients with unequally functioning pyelonephritic kidneys and in monkeys with experimentally induced unilateral pyelonephritis. In all patients the blood urea nitrogen and serum creatinine concentrations were normal. They found that nitrofurantoin in the usual recommended dosage did not reach minimum inhibitory concentrations in the urine of those kidneys with a unilateral creatinine clearance of less than 20 ml per minute. Sulfamethizole and cephalexin, however, both achieved peak urinary concentrations greater than the minimum inhibitory concentration at the lowest studied