

Multiple prosthetic vascular implants into the acutely ischaemic myocardium

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In nine dogs submitted to a severe myocardial ischaemic challenge, multiple prosthetic implants running from the descending aorta to myocardial tunnels were successful in sustaining effective ventricular contraction for a mean period of 27 minutes. A control series of five dogs and five animals, in which the prostheses clotted, developed ventricular fibrillation within six minutes of the ligation of either the left or both coronary arteries. It is suggested that these findings, although not applicable to clinical practice, should stimulate further investigation into the surgical support of the acutely ischaemic myocardium.

The place of myocardial revascularization operations in the management of the chronically ischaemic heart seems to have been well established by Vineberg (1946), Favaloro, Effler, Groves, Sones, and Fergusson (1967), and Sewell, Sones, Fish, Joyner, and Effler (1965). It is agreed that arterial implants have no place in supporting the acutely ischaemic myocardium. Initially the flows recorded from such implants are very small, of the order of 5 ml./min. (Provan, Hammond, and Austen, 1966). This is insufficient to prevent infarction. Three to six months must elapse before effective collateral communications develop. The present study was designed to determine whether the sum of the flows from multiple vascular implants into acutely ischaemic areas of the myocardium would prevent or reduce the area of subsequent infarction.

METHODS AND MATERIALS

There are not enough suitable arteries available for multiple implantations in the thorax of the dog, accordingly a prosthesis was constructed.

Figure 1 shows the design of the prosthesis which was used in the series of experiments now presented. Three tubes, 12 cm. in length with an internal diameter of 3 mm., were mounted on an elliptical base and two side holes were cut near their free ends. The prosthesis was made from woven silicone rubber as described by Ashton, Lightwood, and Hardman (1967). Because of the small calibre of these vessels and the low flow rates obtained, heparin (1 mg./kg.) was given. The prosthesis was preclotted.

A series of 19 experiments was performed. The mediastinum was exposed through a standard left 5th

or 6th interspace thoracotomy. The elliptical basal plate of the prosthesis was sewn as a gusset into the side of the descending thoracic aorta. Three myocardial tunnels, each 3 to 4 cm. in length, were made in the wall of the left ventricle. One tunnel ran parallel to the anterior descending branch of the left

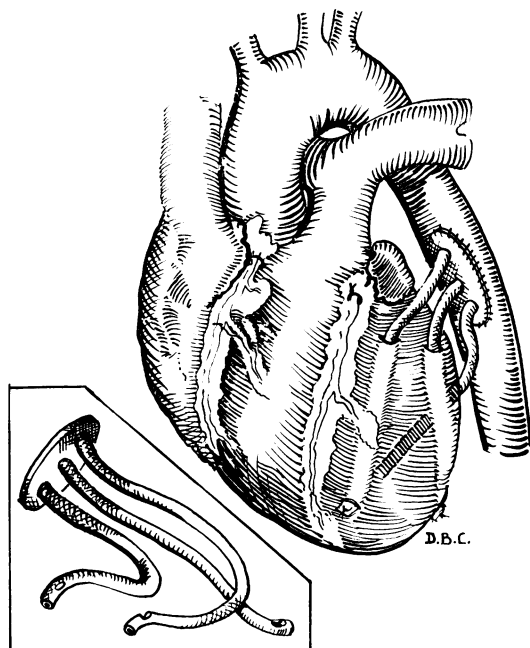


FIG. 1. Multiple prosthetic vascular implants running from the descending aorta to tunnels in the left ventricular myocardium. Inset: the silicone rubber prosthesis.

coronary artery, one along the lateral border of the left ventricle and a third on its postero-inferior aspect, parallel to the circumflex coronary artery. The tunnels were situated so as to pass under major branches of these arteries in the manner described by Sewell *et al.* (1965). The clamps were then removed from each prosthetic vessel in turn, and the freely bleeding ends were introduced into the tunnels by traction sutures mounted on a large blunt needle. These sutures were then brought out through the myocardium and tied over anchoring pledgets of gauze.

Both right and left coronary arteries were ligated close to their origins or alternatively both major branches of the left coronary artery were occluded.

Such a massive ischaemic challenge was felt to be necessary as the assessment of the efficiency of any procedure designed to support the acutely ischaemic myocardium is bedevilled by the variations in distribution and collateral anastomoses between the branches of the coronary arteries. It has been shown that 83% of dogs can survive ligation of the anterior descending branch of the left coronary artery and 25% can survive ligation of the circumflex branch (Pifarré, Yokoyama, Ilano, and Hufnagel, 1967). Dissection at the conclusion of each experiment confirmed that complete occlusion had been obtained.

In a control series of five dogs, both of these methods of producing acute myocardial ischaemia led to ventricular fibrillation within 10 minutes (mean 6 minutes), results which are similar to those described by other workers. This ventricular fibrillation was irreversible.

RESULTS

The 14 animals in the experimental series may be considered as two groups.

Group A consisted of nine dogs in which a satisfactory technical procedure was achieved; operative blood loss was kept to acceptable levels and no thrombosis was found in the prosthetic vessels at the conclusion of the experiment.

Group B comprised five animals which were regarded as technical failures because of either excessive haemorrhage or thrombosis of the prosthesis. Figure 2 shows the time taken for ventricular fibrillation to develop in the control group and in groups A and B after coronary artery ligation.

In the animals in group A an initial fall in blood pressure was noted after coronary ligation, which gradually rose to a level of 90–100 mm. Hg systolic. This pressure was maintained until just before the onset of ventricular fibrillation, when a sharp fall was invariably recorded. In two of the animals in this group ventricular fibrillation occurred within five minutes of coronary ligation, but a short period of cardiac massage followed

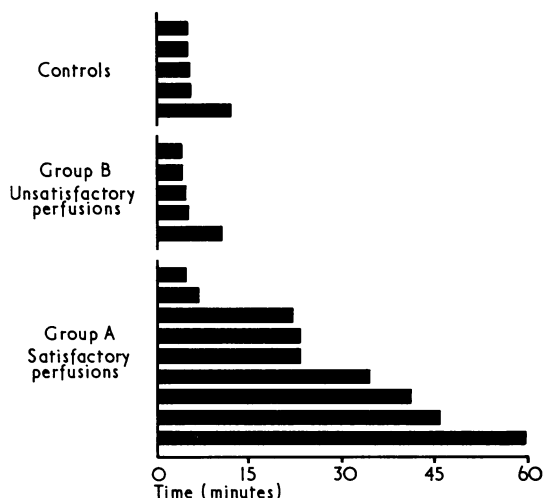


FIG. 2. Time to onset of ventricular fibrillation after coronary artery ligation.

by a DC defibrillating shock succeeded in restoring effective ventricular contraction on each occasion (Fig. 3).

Some difficulty was encountered in obtaining a quantitative assessment of the effectiveness of the procedure. As much of the coronary venous return in the dog passes directly to the ventricular cavities, the measurement of coronary sinus blood flow was of limited value. Blood flow in the prostheses was not measured, as the flow occurs in a to and fro fashion with a small forward progression. Conventional recording would have given an artificially high reading in these circumstances. A technique developed by Subramanian and Wellings (1964) was adopted in which a 5% solu-

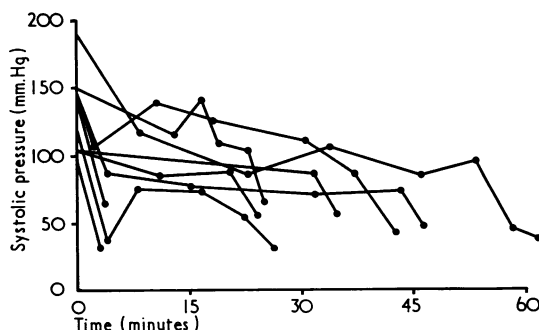


FIG. 3. Systolic pressures of group A dogs after ligation of coronary arteries.

FIG. 4. Slices of heart in control animal in which fluorescein was injected after ligation of both main branches of the coronary artery. The specimen has been photographed under ultra-violet light. There is no fluorescence of the left ventricular mass.

FIG. 5. Slice of heart in group B animal photographed under ultra-violet light. Two of the prosthetic vessels had thrombosed and there is fluorescence only in the region of the patent silicone rubber tube.

FIG. 6. Animal in group A, in which the prosthesis remained patent. There is diffuse fluorescence of the whole of the left ventricular muscle mass. Ventricular fibrillation developed 41 minutes after coronary artery ligation.

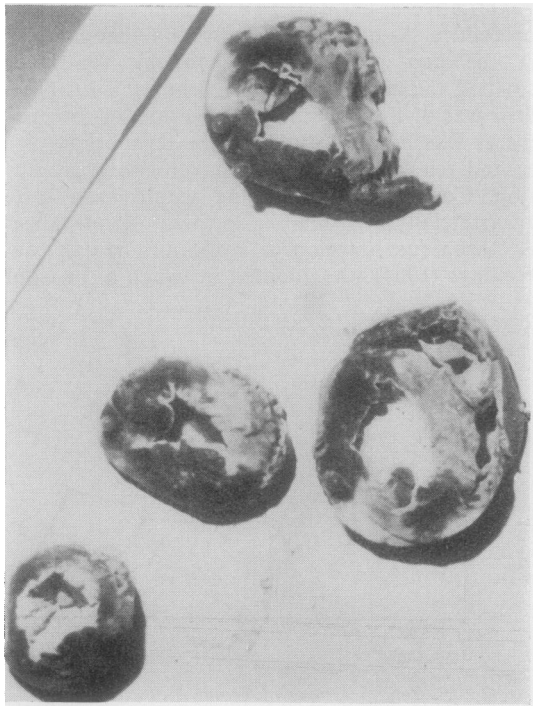


FIG. 4



FIG. 5

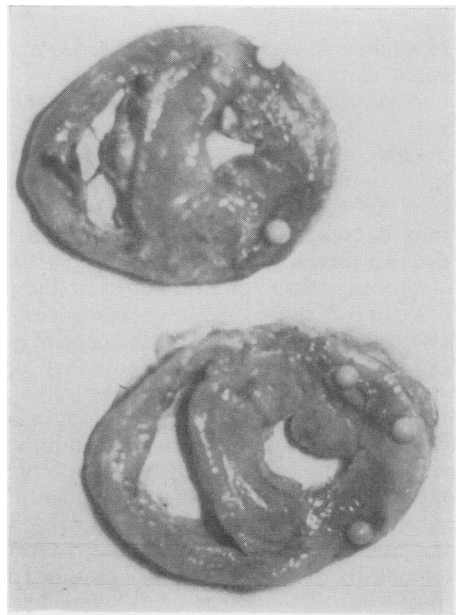


FIG. 6

tion of sodium fluorescein in a dosage of 5.4 mg./kg. was injected intravenously immediately following coronary artery ligation. At the conclusion of the experiment the heart was removed, washed and cut into slices. These were photographed in ultraviolet light with a green filter.

Figure 4 shows slices from the heart of a control animal in which ventricular fibrillation was induced by ligating both main branches of the left coronary artery. The right ventricle and the septum fluoresced, but as no dye reached the bulk of the left ventricular muscle mass it remained dark.

The heart in Fig. 5 is that of a group B animal in which two of the prosthetic vessels clotted and ventricular fibrillation occurred at five minutes. Some fluorescence occurred around the only patent prosthetic vessel. Figure 6 shows the heart of an animal from group A in which ventricular fibrillation developed 41 minutes after coronary artery ligation. Diffuse fluorescence was seen throughout the wall of the left ventricle.

DISCUSSION

Multiple prosthetic vascular implants to the acutely ischaemic left ventricle appeared to prolong the period of effective myocardial contraction before the onset of fibrillation in these experiments.

Figure 2 shows that the mean time from coronary artery ligation to irreversible ventricular

fibrillation in the animals in group A was 27 minutes, compared with 6 minutes in the control group and in group B. The fluorescein studies demonstrated that blood was conducted to the myocardium along the prosthetic vessels. No quantitative assessment of blood flow could be made.

It is not claimed that the technique described is suitable for clinical application. However, these results are of interest as they suggest that acutely ischaemic heart muscle can be temporarily supported by surgical means.

The problems encountered in these experiments were those of clotting in the small diameter tubes and the relatively low blood flows obtained.

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