STUDIES OF THE EFFECT OF EXPERIMENTAL MYOCARDIAL REVASCULARISATION ON VENTRICULAR FUNCTION

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SUMMARY

To evaluate the aorto-coronary bypass graft procedure two animal preparations were used. In the first the effect of myocardial revascularisation on the function of the chronically ischaemic ventricle was studied. The second preparation was used to study the effect on the ventricle of restoring blood flow after one hour of coronary occlusion.

Chronic myocardial ischaemia in dogs was produced surgically by implanted coronary constrictors. Revascularisation was performed using a cannula with a built-in flowmeter. Ventricular function curves were generated using the right heart bypass technique. The thirteen animals studied could be divided into two groups on the basis of the change in ventricular function produced by revascularisation. Group A comprised six dogs in which opening the graft produced an immediate improvement in ventricular function. Group B comprised seven dogs showing no immediate alteration in ventricular function on opening the graft. In Group A the mean graft flow: 48 ± 7.3 ml/min, was greater than Group B, 26 ± 4.0 ml/min (P:0.05). In Group A mean collateral flow: 16±0.3 ml/min was lower than Group 8: 43 ± 4.1 ml/min (P:0.01). With the passage of time after the graft had been opened collateral flow decreased, graft flow increased and the ventricle exhibited the "graft dependence effect", i.e. it became more dependent on the blood supply from the graft. It is concluded that the functional improvement brought about by revascularising

the chronically ischaemic ventricle is not large but is seen most clearly when graft flow is high and collateral flow low.

In the second series of experiments acute myocardial infarction was produced in awake dogs using an occluder implanted on the circumflex coronary artery. Release of the occlusion after one hour allowed reperfusion of the acutely ischaemic area. An acute increase in aortic pressure produced by phenylephrine administration was used to evaluate ventricular function.

In 10 normal dogs a sudden rise in aortic pressure to 190 mm Hg produced a rise in left ventricular end-diastolic pressure from 8 mm Hg to 20 mm Hg, a fall in stroke volume from 24 mls to 19 mls but no significant change in stroke work. The ventricular function curve produced by plotting stroke work against left ventricular end-diastolic pressure was thus approximately horizontal. Coronary occlusion increased left ventricular end-diastolic pressure from 7 mm Hg to 11 mm Hg, reduced stroke volume by 28%, systolic left ventricular pressure by 10% and stroke work by 37%. These changes were not accentuated by aortic pressure loading. The fall in stroke work was found to be the best measure of ventricular depression produced by coronary occlusion.

To assess the effect of revascularisation of an acute myocardial infarction, the coronary occlusion was released after one hour in seven dogs. This group was compared with a control group of nine dogs in which the occlusion was not released. The revascularised

dogs had a smaller mean infarct size: 9.0±2.3% of the left ventricle than the controls: 29.3±3.1% (P<0.005). Of the six revascularised dogs followed for one week, five were alive and four of these showed good recovery of ventricular function, whereas only three of the nine controls survived for one week with poor recovery of function. It is concluded that restoration of flow following one hour of acute coronary occlusion can reduce the size of the infarct and improve the function of the ventricle.