Effect of Bilateral Internal Mammary Artery Ligation on Coronary Circulation in Dogs

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The internal mammary arteries of dogs were ligated bilaterally subsequent to surgical narrowing of the circumflex arteries. Six weeks later measurements of retrograde circumflex flows were made to assess the extent of collateral development. The results when compared with control dogs indicated that internal mammary ligation did not significantly improve the blood supply to the myocardium.

Bilateral ligation of the internal mammary arteries was proposed by Fieschi as a simple surgical means of treating patients with coronary artery disease.1 The procedure aims to increase the blood pressure in the pericardiophrenic branches of the internal mammary arteries and, thereby enhancing coronary anastomoses to the myocardium. Observations made on patients have been conflicting,1,2 as have the data based on animal experiments.1,3,4 These experiments were therefore designed to test whether bilateral internal mammary ligation actually improves the collateral arterial blood supply to the myocardium of the dog subsequent to surgical narrowing of a coronary artery and to assess the effect of internal mammary artery occlusion on central internal mammary arterial pressure.

Methods

Each of 7 healthy mongrel dogs (14.5 to 16.9 Kg.) was anesthetized with 2 mg. of morphine and 25 to 30 mg. of pentobarbital/Kg. Intermittent positive pressure respiration was instituted through intratracheal tubes. The left chests were opened, and the circumflex arteries were narrowed by tying over probes as previously described.5 Bilateral internal mammary artery ligation in the second intercostal space was done immediately in 4 of the dogs. In the remaining 3 the internal mammary arteries were ligated through skin incisions 7 days later.

Six weeks later the dogs were anesthetized and retrograde flows from the circumflex arteries and through the constricting ligatures were measured5 to assess the extent of collateral function. The internal mammary arteries were then clamped at their origins and circumflex retrograde flow measurements were repeated. The flow rates were calculated per 100 Gm. of myocardium/min,5 and compared with values found in a large group of control dogs having similar degrees of circumflex narrowing but with intact internal mammary arteries.

Electrocardiograms were recorded during complete occlusion of the circumflex arteries and during retrograde circumflex bleeding, both with the origins of the internal mammary arteries intact and clamped.

Finally, needles connected to optical pressure manometers were inserted into the internal mammary arteries of 3 anesthetized normal dogs with open chests. Pressures were recorded before and after sudden distal internal mammary artery occlusion.

Results

Three of the 4 dogs which had bilateral internal mammary ligation and circumflex artery narrowing at the same operation died within 24 hours. No gross pathologic findings were present to account for these deaths.

Figure 1 shows that the retrograde flows in the remaining 4 dogs do not differ from the
retrograde flows in the control animals with similar degrees of circumflex narrowing but without internal mammary artery ligation. Repeated flow measurements made before and after clamping the central internal mammary artery were essentially the same.

Electrocardiograms remained normal in all animals during prolonged periods of complete circumflex artery occlusion even after the origins of the internal mammary arteries were occluded. In 3 animals ST-T wave changes occurred after the peripheral circumflex arteries were permitted to bleed to atmospheric pressure, and these changes were not modified either by opening or clamping the origins of the internal mammary arteries.

The pressure changes induced by clamping the internal mammary artery were as follows: in 1 dog the central internal mammary artery pressure increased from 178/132 to 181/133 mm. Hg immediately following the occlusion of the artery at the level of the second intercostal space; in a second animal the pressure increased from 111/93 to 115/96 mm. Hg while in a third animal there was no measurable effect induced by clamping the artery distally.

DISCUSSION

It has been previously shown that the extent of collateral development in the dog heart is proportional to the degree of coronary artery narrowing. This suggests that the stimulus to collateral growth depends on a pressure differential between arteries or a region of myocardial hypoxia or both. Although the surgically induced circumflex narrowing in these experiments was sufficient to create a stimulus for collateral growth, the direct retrograde flow measurements fail to show functional anastomoses between the pericardiophrenic and coronary arteries even under the optimal conditions created during the measurements. These conclusions are supported by the lack of electrocardiographic changes following the removal of the potential source of collateral flow due to clamping the central internal mammary arteries during both circumflex occlusion and during retrograde circumflex bleeding.

The pressure elevations in the central internal mammary artery following its distal occlusion are similar to those reported by Glover. Even though elevations in pressure follow internal mammary ligation it is hazardous to predict that elevations in pressure of this magnitude can stimulate collateral growth. Measurements in this laboratory suggest that a pressure gradient of 12 to 15 mm. Hg may be necessary to promote collateral development. If it were possible to create pressure elevations in the internal mammary artery it is likely that considerable loss might be sustained due to frictional resistance in the long, narrow, pericardiophrenic artery.

It is difficult to square the present findings with the demonstration by Hudson, Moritz, and Wearn of extracoronary anastomoses in human hearts. Two possibilities suggest themselves. Either these human extracoronary anastomoses are not functionally significant or it is more likely that in certain human hearts widespread coronary disease limits effective intercoronary artery pressure gad-
BILATERAL INTERNAL MAMMARY LIGATION

ients with the result that pressure gradients exist over the extracoronary communications to promote their enlargement.

SUMMARY
The evidence derived from anesthetized dogs fails to support the hypothesis that bilateral ligation of the internal mammary arteries raises pressure in the pericardio-phrenic arteries to a degree sufficient to improve the blood supply to the myocardium.

ACKNOWLEDGMENT
The authors appreciate the technical assistance given by Mrs. Jacqueline Chadwick and Mr. John Dattilo.

SUMMARIO IN INTERLINGUA
Observationes in anesthesiate canes non supporta le hypothese que le ligation bilateral del arterias mammari interne augmenta le tension in le arterias pericardio-phrenic usque a un grado sufficiente pro meliorar le provision de sanguine al myocardio.

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Circ Res. 1959;7:571-573
doi: 10.1161/01.RES.7.4.571

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