

THE EXPERIMENTAL USE OF THE DIAPHRAGM AS AN AUXILIARY MYOCARDIUM *

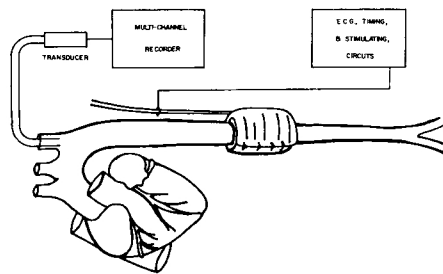
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In recent years, numerous efforts have been made to improve the blood supply to the myocardium. We know of no work, however, directed toward increasing the mass of functioning, self supporting myocardium. The purpose of these experiments was to explore the possibility of using the motor power of the diaphragm to share the workload of the myocardium.

METHOD

In these acute experiments, 29 adult mongrel dogs were anesthetized with nembutal. A long midline incision exposed the diaphragm. The diaphragm was divided in the midline and the left hemidiaphragm dissected from its peripheral attachments. Its major blood supply and the left phrenic nerve were not disturbed.

Two groups of experiments were performed. In the first group of 8 dogs, the fan-shaped leaf of diaphragm was sutured around the heart so that its muscle fibers ran transversely to the long axis of the ventricles. In the second group of 21 dogs, the hemidiaphragm was folded into a rectangular shape with the muscle fibers running in the long axis. This muscle mass was wrapped around the mobilized distal thoracic aorta so that its fibers ran perpendicular to the aorta. In both series, two silver wire electrodes were attached to the left phrenic nerve and brought out to a terminal strip. Output leads of an electronic stimulator were attached to the left phrenic nerve



terminal strip. With this instrument a stimulus could be varied in relation to time, intensity and wave form. In both groups of experiments the stimulator was triggered by the QRS complex of the electrocardiogram. In the cardiac group of 8 experiments, the stimulus was supplied throughout systole and in the aortic group of 21 experiments during diastole. Carotid artery pressures were recorded by means of a strain gauge in all experiments. Electrocardiograms were simultaneously recorded on the multi-channel oscillograph. Carotid artery blood flows were recorded with a rotameter in some experiments.

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RESULTS

In the initial experiments, the mobilized left leaf of the diaphragm was sutured around the heart and the left phrenic nerve stimulated synchronously with each systole. However, the arterial pressures were not significantly higher than those of the control studies where the diaphragm was not stimulated.

The mobilized left hemidiaphragm was wrapped around the distal portion of the thoracic aorta in the second group of experiments. Here the electronic circuit was arranged to pick up the ECG and deliver a stimulus to the left phrenic nerve during diastole. There was a significant rise in diastolic pressure as compared to the control studies in all of these experiments. Figure 2 gives the results of a typical experiment. Mean pressures were recorded for 32 seconds. The mean pressure averaged 98 mm. Hg during the initial 8 second control period. After that interval, the diaphragm wrapped around the aorta was stimulated during each diastole. The mean pressure gradually rose and reached a peak of 124 mm. Hg within 10 seconds, an increase of 26.5%. By 15 seconds of stimulation the mean pressure had returned to its control level. Thereafter there was some overshooting but it soon returned and stayed at control levels until the stimulus was removed. The electrocardiogram recorded on the lower portion of the figure is not easily read because of the slow paper speed. Figure 3 is a tracing from the same experiment recorded at higher paper speed. In section A, the carotid artery pressures of two cardiac cycles are shown with a simultaneously recorded ECG. The systolic peak was 108 mm. Hg and the end diastolic level 78 mm. Hg. The highest diastolic pressure attained during this control period was 92 mm. Hg. Section B, taken 4 seconds later, records similar pressures when a stimulus was given to the left phrenic nerve during each diastole. The stimulus appears on the electrocardiogram and its amplitude is 500 millivolts. It was delayed 0.2 seconds and lasted 0.16 seconds. The systolic peak was 107 mm. Hg. However, the diastolic pressure was significantly increased. The end diastolic pressure was 90 mm. Hg and the highest diastolic pressure was 105 mm. which was almost as much as the systolic pressure.

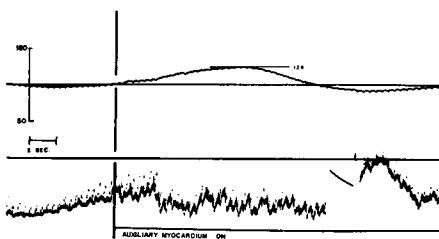


Fig. 2. Effect of auxiliary heart on mean arterial pressure.

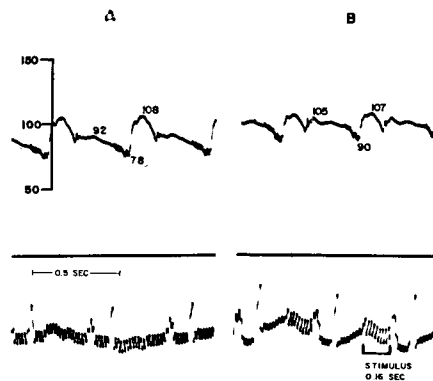


Fig. 3. Effect of auxiliary heart on pressure pulses.

DISCUSSION

Our interpretation of the dynamics of these events is essentially as follows: the left ventricle contracts and delivers its stroke volume of blood into the aorta. This raises aortic pressure to its systolic peak. The auxiliary heart contracts at the beginning of diastole and delivers its stroke volume both cephalad and caudad into the aorta. This effectively raises the diastolic pressure in the aorta in two ways: 1) It pushes a quantity of blood into the thoracic and abdominal aortas. 2) It decreases the volume of the aorta available to contain this blood by the amount of collapsed aorta beneath the wrapped diaphragm. The result of these two effects is to markedly increase diastolic pressure and therefore mean pressure in the aorta. The increased aortic mean pressure results in an increased blood flow to all of the arterial branches including the coronary arteries. The coronary artery flow is particularly sensitive to increases in diastolic pressure as we have shown in previous work.¹ The homeostatic mechanisms of the animal respond to either this increased mean pressure or increased flow (either or both, this is not yet clear) by controlling the only part of the pumping mechanism under its control, i.e. the left ventricle. The mean pressure returns to normal. Normal mean pressures are now maintained by the left ventricle which works less vigorously with lower systolic pressures and by the auxiliary ventricle which maintains higher diastolic pressures. Because the left ventricular work is performed only during systole and is quantitatively related to the systolic pressures, decreased systolic pressures mean decreased ventricular work.

The left hemidiaphragm is adaptable for this purpose. It is a powerful muscle which can be mobilized at its periphery without disturbing the phrenic nerve or the base which contains its blood supply. Because its reaction time is short, it can function at the frequencies necessary. It is flat, can be folded easily and wrapped around the adjacent aorta. It is expendable as we and others have shown.² We have a series of dogs, to be reported at a later date, where the defect has been repaired with dacron cloth and the animals have survived with apparently no ill effects. In order to avoid the necessity of passing electrodes through the skin to the phrenic nerve, the feasibility of constructing an electronic device which would transmit a properly timed impulse through the intact skin is now being explored.

SUMMARY

1. This report describes exploration of methods to develop an auxiliary myocardium which would act in effect as a "booster heart."

2. The method consists of mobilizing the left leaf of the diaphragm peripherally in such a manner as to preserve its blood and nerve supplies. The muscle is wrapped around the distal portion of the thoracic aorta and stimulated during each diastole.

3. Measurements of arterial pressures and flows reveals that the "auxiliary heart" adds energy toward propelling the arterial blood peripherally.

REFERENCES

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