THE MECHANICAL INFLUENCE OF THE PERICARDIUM UPON CARDIAC FUNCTION

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The action of the heart may be profoundly disturbed by alterations in the pressure maintained over its outer surface. Tamponade of the heart by an intrapericardial hemorrhage or effusion is the most familiar example and is such a classical phenomenon, both clinically and experimentally, that description would be superfluous. Adhesive pericarditis of the type called *concretio pericardii*, commonly termed Pick's disease, may produce many of the signs and symptoms of cardiac tamponade over a period of years. The essential lesion in this condition is the contracted and scarred pericardium that tightly grasps the chambers of the heart as if in a vise. The impeded diastolic filling of the heart causes an increased venous pressure with chronic passive congestion, ascites, and edema, even in the presence of a normal myocardium and competent valves. The operative relief of this condition constitutes one of the most dramatic chapters of modern surgery (1) (2).

Since the proper action of a heart of normal size can be restored by freeing it from a constricting pericardium, the suggestion has been made that the function of an hypertrophied and dilated heart might also be improved by removal of the structures that limit diastolic filling. As the normal pericardium stretches almost without limit in the face of a slowly developing cardiac enlargement, it is the unyielding bony framework of the chest that serves ultimately to restrict the greatly dilated or hypertrophied heart. Assuming at the outset that further dilatation of the heart will increase its functional capacity an effort may be made to give room for further dilatation by surgical removal of the overlying ribs and costal cartilages—so-called "decompression."

With these clinical problems in mind the action of the cat's heart has been embarrassed by imposing a partial obstruction to the outflow of blood. The function of the pericardium under such circumstances has been considered, with particular reference to the deleterious effects on the circulation that may result from the limitation of cardiac dilatation.

The experimental literature on the function of the pericardium is not extensive. Barnard (3) in 1898 reported to the Physiological Society certain studies on the pericardium and concluded that it was an inelastic structure preventing excessive dilatation of the heart.

Kuno (4) in 1915, using a Starling heart-lung preparation and an experimental method enabling him to open and close the pericardium at will, found that opening the pericardium caused a fall in venous pressure, a rise in arterial pressure and an increase in cardiac output. This effect was more marked at low venous pressures and with slow pulse rates. He also noted that hearts unsupported by the pericardium were apt to show hemorrhages beneath the epicardium and valvular insufficiency at high venous pressures. These effects were not observed with high venous pressures when the pericardium was intact. He concluded that "the existence of the pericardium is thus necessary for the unimpaired working of the heart in normal life."

Yamada (5) in 1917 confirmed these observations of Kuno on the heart-lung preparation. He also widely opened the pericardium in dogs, exercised them by running and examined the heart two to eight days after operation. He failed to find any evidence of dilatation or intramural hemorrhage and thought the absence of these conditions due to the rapid pulse rate occurring with exercise. The experiments in which he attempted to demonstrate the effects of rapid heart rates are not convincing because adrenalin was employed to increase the heart rate while its other physiologic effects were apparently not considered.

Wilson and Meek (6) in 1927 studied in roentgenograms the restraining influence of the pericardium on the size of the heart. After severing the diaphragmatic attachment of the pericardium in the dog they found that the pericardium began to exert a restraining influence on the diastolic size of the heart at a venous pressure of 0 cm. of water and that at a venous pressure of 8 cm. the heart completely filled the pericardium.

Van Liere and his collaborators (7) (8) in 1927 and 1930 produced acute cardiac dilatation by anoxemia and stimulation of the vagus nerve simultaneously recording the size of the heart shadow by roentgenograms. They found in the majority of their experiments a greater dilatation of the heart, after removal of the pericardium.

On the other hand, Felix (9) studying the influence of the pericardium upon hearts with artificially produced valvular defects, concluded that opening the pericardium had a favorable influence upon hearts with mitral or tricuspid lesions and suggested adopting this procedure in patients. In a recent article (10) he has published some further investigations on this question in both acute and survival experiments. In experimentally produced mitral insufficiency of several weeks duration he fails to note any circulatory improvement on opening the pericardium. It seems not unlikely that the findings in his acute experiments are due to the production of a certain degree of tamponade by the closure of the pericardium after creating the valvular defect. The reopening of the pericardium then improved cardiac action as a matter of course.

Moore (11) and Grant (12) reviewing the cases of congenital pericardial deficiency reported in the literature, conclude that the pericardium does not play an important part in supporting the wall of the heart. Experimental removal of the pericardium in animals is apparently compatible with active life (13).

In the following experiments we have studied: (A), the effect of opening the pericardium upon the function of the normal heart as indicated by systemic and venous blood pressures; (B), the effect of opening the pericardium when the work of the heart has been greatly increased by a partial obstruction to the outflow of blood from one ventricle; (C), the effect of removing a constricting pericardium in the presence of a partial occlusion of the pulmonary artery or aorta; and (D), the tamponade effects produced by increasing the work of the heart within a snugly adjusted pericardium.

TECHNIC

The experiments were performed on cats anesthetized by the intraperitoneal injection of a 10 per cent solution of sodium barbital, 4.5 cc. per kilogram of body weight. The injection was made one half to

three quarters of an hour before the experiment was started. Access to the pericardium was obtained by removal of the lower two thirds of the sternum. Both pleural cavities were widely opened and artificial respiration was maintained throughout the experiment by intermittent intratracheal insufflation. The mean carotid pressure was recorded in the usual manner with a mercury manometer. The venous tracing was obtained by the method described by Lewis (14). A long slender glass cannula was passed through the external jugular vein into the superior vena cava and connected with a saline manometer at the top of which was a small Brodie bellows recorder. A solution of heparin was given intravenously to prevent clotting.

These preliminary procedures were carried out in all the experiments described below. It should be noted that this preparation entails the exposure of all the intrathoracic structures to atmospheric pressure with a consequent diminution in cardiac output (15) and presumably a slight decrease in diastolic heart size.

RESULTS

A. Effect on the normal heart of opening the pericardium. The effect of opening the pericardium upon the venous and arterial pressures was noted in six experiments. The pericardium was slit wide open from base to apex with a pair of fine scissors. No change in venous or arterial pressure was observed in any of the experiments (fig.1). Occasionally there was a slight initial disturbance with a quick return to previous levels. This disturbance could be reproduced by pressing the scissors upon the right ventricle or by drawing up on the pericardium with a pair of forceps, and was probably caused by such manipulation rather than by the actual opening of the pericardium.

These observations fail to corroborate those made in heart-lung preparations by Kuno (4) and Yamada (5) who found a fall in venous pressure, a rise in arterial pressure and an increased cardiac output on opening the pericardium. It should be recalled, however, that their observations were not made on the intact pericardium, but on one which had been previously opened and then drawn together again with sutures. That such a method can very easily cause cardiac tamponade is shown below (C). Neither author records the effect upon the venous and arterial pressures of the initial opening of the intact

pericardium. The pulse rate in these six experiments varied from 136 to 224 per minute. There was no change in rate on opening the pericardium. The cardiac rate of the dog's heart used in heart-lung preparations is often only one half as rapid. According to the views of Yamada this may possibly account for some of the discrepancies between the results recorded here and those obtained by Kuno.

B. Effect of opening the pericardium upon a heart with a partial obstruction to the outflow of blood from one ventricle. The work performed by the heart is roughly the product of the cardiac output multiplied by the arterial resistance (16) (17). In these experiments the

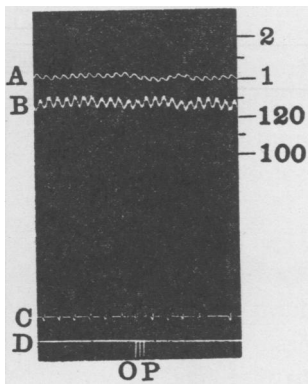

FIG. 1. KYMOGRAPHIC TRACING AT MOMENT OF OPENING PERICARDIAL CAVITY

A, venous pressure in centimeters of water recorded from the superior vena cava. *B*, arterial pressure in millimeters of mercury recorded from left carotid artery. *C*, time in 5 second intervals. *D*, signal marker. At *OP* the anterior surface of the pericardium was slit from base to apex.

work of the heart was increased by augmenting the arterial resistance. For this purpose a special clamp was devised for graduated compression of the pulmonary artery or aorta. The clamp is very similar to the one used by Haggart and Walker (18). However, it is capable of finer adjustments and can be employed in the "closed" chest. It consists of a tube *A* (fig. 2) to which is fixed the upper jaw of the clamp. The rod *BC* passes through the tube *A* and is bent to form the lower jaw of the clamp. The upper half of *BC* is wormed and articulates with the knurled nut *D*. An eighth of a turn of the nut moves the lower jaw of the clamp 0.079 mm.

Graduated compression of the pulmonary artery or aorta by this clamp was employed in these experiments only as a means of increasing the work of the heart by increasing the peripheral resistance. The circulatory disturbances resulting from such compression will be more fully described in another communication. It is interesting to note, however, that the sudden fall in arterial and rise in venous pressures

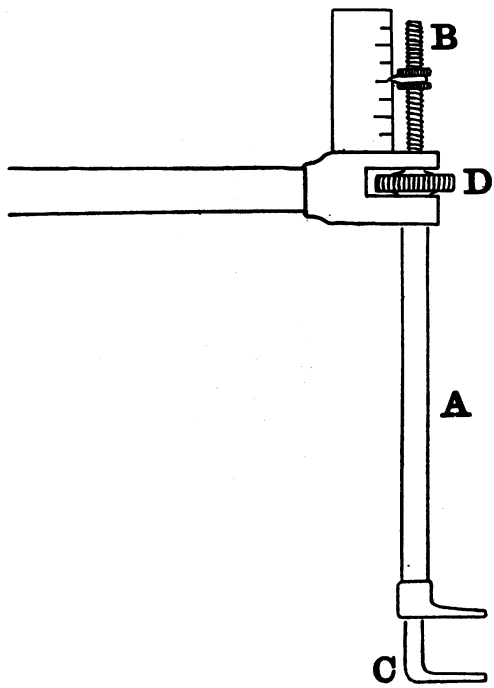

FIG. 2. CLAMP DESIGNED FOR GRADUATED COMPRESSION OF THE PULMONARY ARTERY OR AORTA
(See text)

observed by other investigators, Cohnheim (19), Haggart and Walker (18) and Moore and Binger (20), during compression of the pulmonary artery or aorta have not been observed in our experiments when the compression was very gradually induced. There is on the contrary, a slow fall in arterial pressure and a corresponding rise in venous pressure. The kymographic record from Experiment 21 showing this

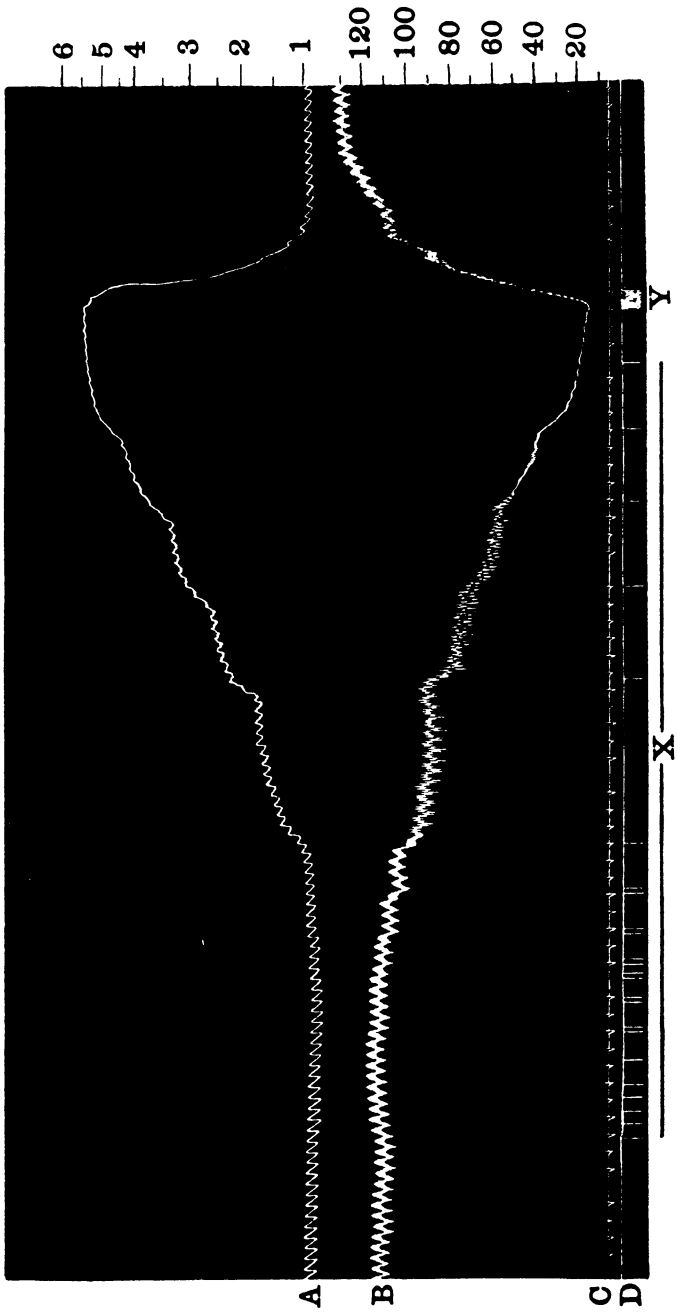

FIG. 3. KYMOGRAPHIC TRACING, EXPERIMENT 21

Venous and arterial pressures as in figure 1. During the time interval *X*, the clamp on the pulmonary artery was tightened by successive increments recorded by the signal marker. A slow fall in arterial and a rise in venous pressures resulted. At *Y* the clamp was completely released.

effect is reproduced in figure 3. In this experiment the anterior surface of the pericardium was incised from base to apex, completely freeing the heart. The clamp was then adjusted about the pulmonary artery just above the pulmonary valves. Very gradual compression of the artery with the clamp (0.079 mm. at a time) produced a slow decline of arterial pressure and a slow rise in venous pressure. Figure 4 is a tracing from the same experiment a few minutes later, illustrating the effect of more abrupt compression. Here the successive incre-

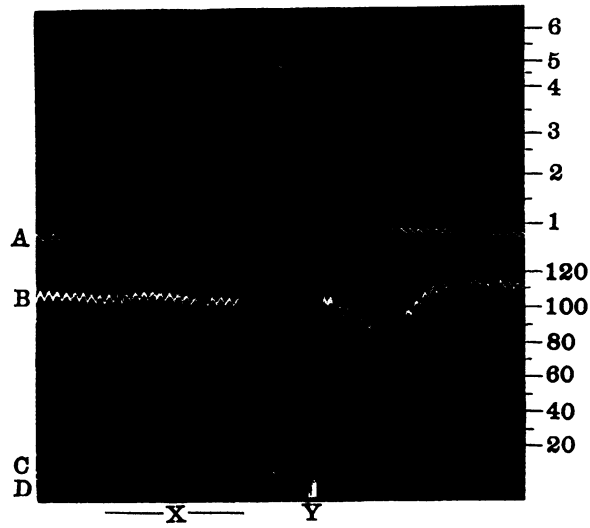

FIG. 4. KYMOGRAPHIC TRACING, EXPERIMENT 21

Venous and arterial pressures as in figure 1. Clamp tightened during time interval *X* and released at *Y*. Compression of the pulmonary artery in a more abrupt manner than shown in figure 3 produced a sudden failure of the heart.

ments of compression were larger (0.635 mm.) and heart failure occurred abruptly as has been described by the above mentioned observers. The same results have also been observed during slow and rapid compression of the aorta.

Opening the pericardium after graduated compression of the pulmonary artery or aorta was carried out in ten experiments. In three of these the aorta was compressed and in seven the pulmonary artery. A small transverse incision, 1.5 cm. long, was made in the base of the

pericardium directly overlying the pulmonary artery or aorta. The incision exposed only that portion of the vessel about which the clamp was to be placed and did not overlie the heart itself or presumably affect the normal relationship between the pericardium and the heart. The clamp was then placed in position and the artery compressed until there was a definite fall in arterial pressure and a rise in venous pressure. Opening the pericardium at this point by incising its anterior surface from base to apex was without effect on either venous or arterial pressures in five experiments. In one experiment the arterial pressure fell slightly and in another rose slightly. In two experiments the arterial pressure was progressively falling and the venous pressure rising when the pericardium was opened; the heart continued to fail following the procedure. Finally, in one experiment there was some improvement in the circulation on opening the pericardium (*OP*, fig. 5), evidenced by a slight rise in the carotid pressure and a slight fall in superior caval pressure.

Under the conditions of these experiments the pericardium seems to offer no material support to a heart seriously decompensated by an obstruction to the outflow of blood. In no instance did removal of the pericardium in itself precipitate heart failure. In one instance (fig. 5) the removal of the pericardium actually enabled the heart to overcome its handicap more effectively.

C. Effect of removing a constricting pericardium in the presence of an obstruction to the outflow of blood from one ventricle. In order to study the functional relations of the pericardium to the heart under different experimental conditions, a method for opening and closing the pericardium was employed which is very similar to the method devised by Kuno. The anterior surface of the pericardium was incised longitudinally from base to apex. Three or four interrupted silk sutures were placed in the cut edges and drawn through holes in a small strip of celluloid (fig. 6). By drawing these sutures up through the strip of celluloid and clamping them with a small artery clip the pericardium could be closed, and by releasing the threads, opened. If the threads were drawn up tightly and clamped, tamponade effects resulted (*CP*, fig. 7). On releasing the threads, and thus opening the pericardium, the arterial and venous pressures returned to their for-

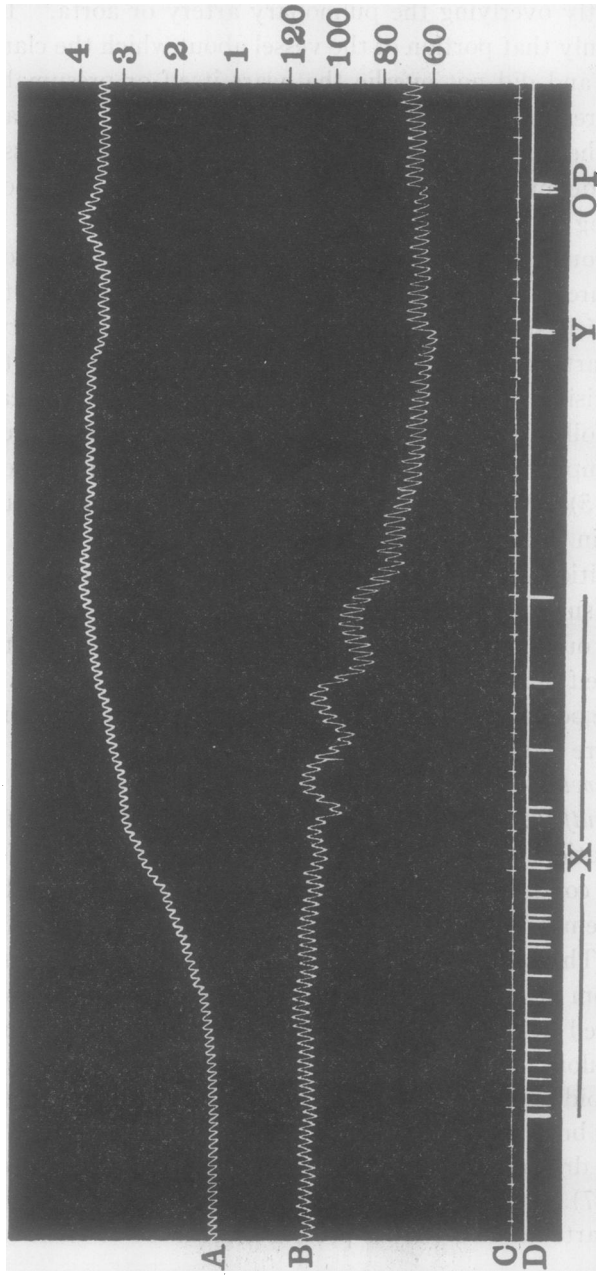

FIG. 5. KYMOGRAPHIC TRACING ILLUSTRATING THE OPENING OF THE PERICARDIUM IN THE PRESENCE OF PARTIAL OCCLUSION OF THE PULMONARY ARTERY

Venous and arterial pressures as in figure 1. Pulmonary artery gradually compressed during time interval *X*. Slight release of clamp at *Y* to prevent further cardiac failure. Opening of the pericardium (*OP*) gave but minimal circulatory improvement.

mer levels (*OP*, fig. 7). This effect was recorded thirty-one times in thirteen experiments.

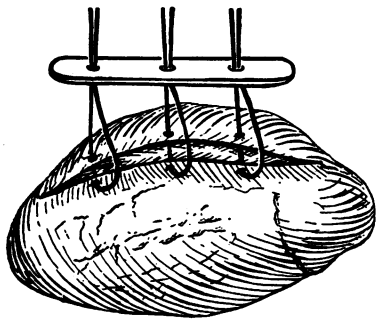

FIG. 6. DIAGRAM ILLUSTRATING METHOD OF OPENING AND CLOSING PERICARDIUM

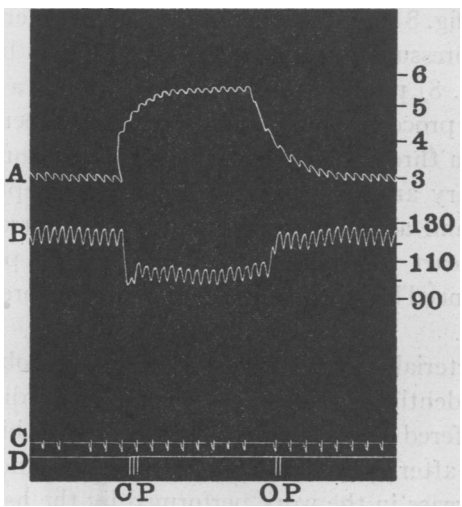

FIG. 7. KYMOGRAPHIC TRACING SHOWING TAMPONADE EFFECTS OBTAINED BY CLOSING PERICARDIUM AFTER METHOD OF KUNO

Arterial and venous pressures as in figure 1. At point *CP* the pericardium was closed and at *OP* opened.

The cardiac tamponade so produced is similar to that occasioned by an experimentally produced pericardial effusion (19) (21). The extent of the deviations in arterial and venous pressure levels was

found to be dependent upon the degree of diminution in the original volume of the pericardium. The tighter the threads were drawn together, the more marked was the increase in venous pressure and the decline in arterial pressure. Releasing the threads and thus opening the pericardium, always resulted in a rise in arterial and a fall in venous pressures. It was then determined whether this effect is to be observed after greatly increasing the work of the heart by partial obstruction of the pulmonary artery or aorta and thus still further lowering the arterial and raising the venous pressures.

In Experiment 20 the pericardium was incised from base to apex. The clamp was placed about the pulmonary artery, and sutures for artificially opening and closing the pericardium were placed as described above. The threads were drawn up tightly, producing a slight cardiac tamponade (*CP*, fig. 8). The pulmonary artery was compressed (*X*, fig. 8), producing a further fall in arterial pressure and rise in venous pressure. Opening the pericardium by releasing the threads (*OP*, fig. 8) produced a fall in venous, and a rise in arterial, pressure. This procedure was carried out seventeen times in nine experiments. In three experiments the aorta was compressed and in six the pulmonary artery. In every instance, except one, a similar rise in arterial and a fall in venous pressures occurred. In the one exception the heart was rapidly failing when the pericardium was opened, as evidenced by a constantly falling carotid pressure and rising venous pressure.

The rise in arterial and fall in venous pressures observed in these experiments evidently indicates an increase in cardiac output. As the resistance offered by the clamp to the outflow of blood is the same both before and after opening the pericardium, the increase in output indicates an increase in the work performed by the heart. Therefore these results apparently indicate that the heart can accomplish more work if the restraining influence of a constricting pericardium is removed.

The cardiac tamponade resulting from tight closure of the pericardium by sutures is obviously very similar to that existing in cases of *concretio pericardii*, where the normal return of venous blood to the heart is impeded by a rigid, thickened pericardium. Removal of such a pericardium results in circulatory improvement, as does opening of

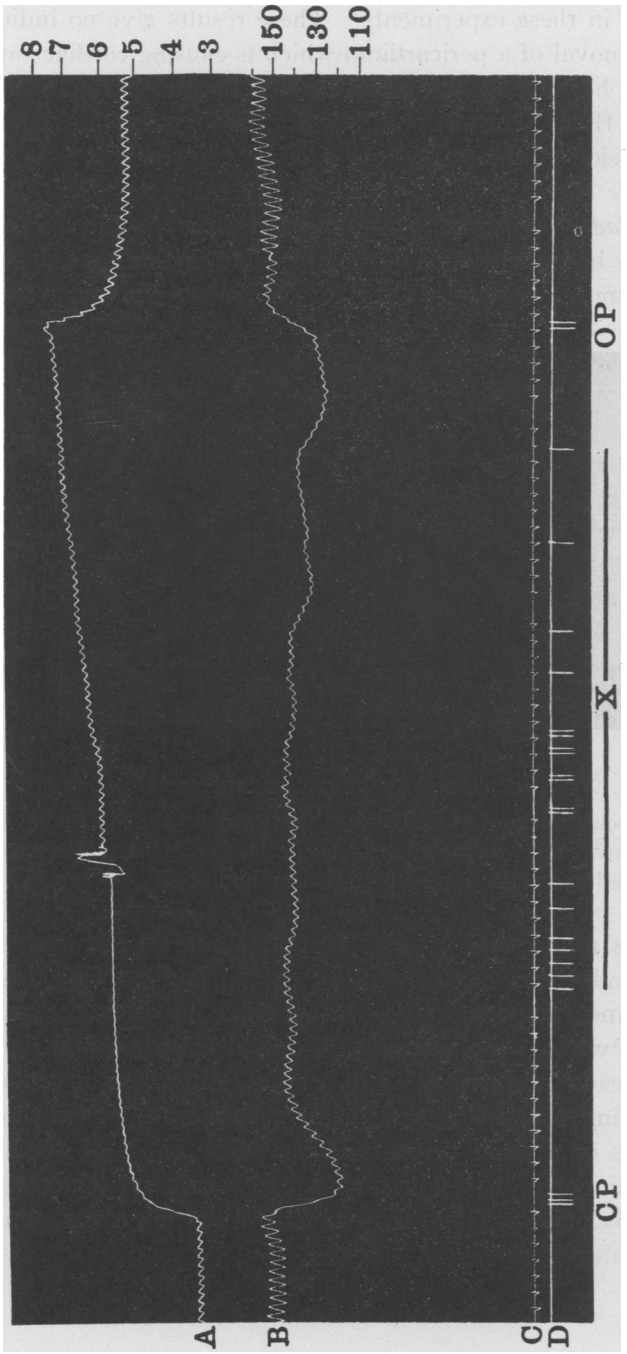

FIG. 8. KYMOGRAPHIC TRACING, EXPERIMENT 20

Arterial and venous pressures as in figure 1. Pericardium was closed at point *CP* producing moderate tamponade. Clamp about pulmonary artery tightened during time interval *X*. Opening of pericardium at *OP*.

the pericardium in these experiments. These results give no indication that the removal of a pericardium which is causing cardiac tamponade may be harmful in the presence of a normal heart muscle. This is true even though the work of the heart may be greatly increased by partial occlusion of the pulmonary artery or aorta.

D. Tamponade effects produced by increasing the work of the heart within a snugly adjusted pericardium and relieved by opening the pericardium. It has been mentioned in the preceding section that closure of the pericardium by means of sutures resulted in cardiac tamponade.

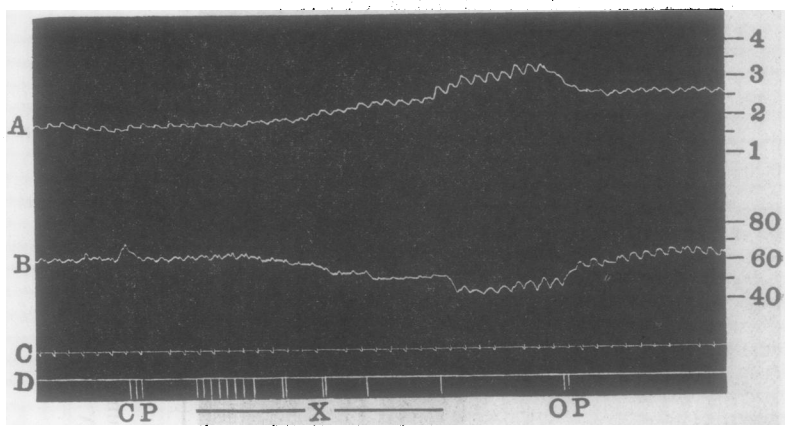

FIG. 9. KYMOGRAPHIC TRACING, EXPERIMENT 17

Arterial and venous pressures as in figure 1. Pericardium closed at point *CP* to snugly envelop heart but without tamponade effects. Pulmonary artery compressed during time interval *X*. Opening of pericardium at *OP*.

However, it was found that by exerting great care in drawing the sutures up through the celluloid strip, the pericardium may be closed without producing tamponade. It was possible to draw the pericardium so snugly about the heart that the slightest additional tightening of the threads produced a fall in arterial, and a rise in venous, pressure. Opening the pericardium after such closure was without effect on venous or arterial pressure levels. The effect of opening such a pericardium on a heart whose work had been greatly increased by means of the clamp was determined in the following experiments.

In Experiment 17 the pericardium was incised longitudinally from base to apex. The clamp was placed about the pulmonary artery and sutures for artificially opening and closing the pericardium were placed as described above. The threads were drawn as tightly as possible without affecting either arterial or venous pressures (*CP*, fig. 9) and clamped. The pericardium was thus drawn up snugly about the heart without producing the slightest degree of cardiac tamponade. The pulmonary artery was compressed until a well marked rise in venous pressure and fall in arterial pressure occurred (*X*, fig. 9). When the pericardium was then opened by releasing the threads, a fall in venous, and a rise in arterial, pressure occurred (*OP*, fig. 9).

This procedure was carried out eighteen times in seven experiments. In ten instances opening the pericardium caused a rise and fall in arterial and venous pressures respectively, similar to that shown in figure 9. In five instances the arterial pressure was unchanged but the venous pressure fell. In two instances no effect was observed in either venous or arterial pressures, and in one instance the heart was failing before the pericardium was opened and continued to fail thereafter.

The method of closing the pericardium employed here, namely drawing up on the sutures until any further tightening caused a rise in venous and a fall in arterial pressures, may be said to cause a diminution in the original volume of the pericardial cavity. Such a conclusion appears justified by a comparison of the results obtained on opening the pericardium in these experiments and in those described under section B. The relationship between the heart and pericardium in the experiments just described bears some similarity to that which exists between an enlarged heart and the structures that limit its diastolic expansion. In one case the pericardium has been made smaller, and in the other the heart has become larger. In both instances the difference between the diastolic heart size and the room available for expansion is diminished. These experiments then lend support to the observations already on record (9), that decompression of the heart may in favorable instances enable it to compensate more adequately for valvular defects.

SUMMARY AND CONCLUSIONS

The following observations have been made on cats with open chests under artificial respiration:

1. Opening the pericardium is without effect upon either arterial or venous pressures.

2. Gradual compression of the pulmonary artery results in a slow fall in arterial pressure and a corresponding rise in venous pressure.

3. Opening the pericardium when cardiac decompensation has been produced by compression of the pulmonary artery or aorta is without significant effect on the elevated venous pressure or the low arterial pressure.

4. Tight closure of the pericardium with sutures produces cardiac tamponade with a fall in arterial and a rise in venous pressures. On opening the pericardium again, the pressures return to their former level.

5. When the pericardium has been closed with the production of cardiac tamponade and partial obstruction of the pulmonary artery superimposed, opening the pericardium causes a rise in the carotid pressure and a fall in the superior caval pressure.

6. By exercising considerable care it is possible to close the pericardium with sutures without affecting either venous or arterial pressures. Subsequent opening of the pericardium is similarly without effect on pressure levels.

7. When the pericardium has been snugly closed by sutures without affecting either arterial or venous pressures and cardiac decompensation produced by compression of the pulmonary artery, opening the pericardium causes a rise in arterial pressure and a fall in venous pressure.

8. In none of the twenty-eight experiments performed was the removal of the pericardium detrimental to the circulation, even when signs of cardiac decompensation had been experimentally produced.

9. Increased work is accomplished by increase in the diastolic size of the heart. Removal of a pericardium which limits this normal cardiac dilatation materially lessens the degree of cardiac decompensation.

10. An analogy is drawn between these experimental observations

and the so-called "decompression" of enlarged hearts by removal of the bony framework of the precordial area.

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