THE SIGNIFICANCE OF THE PERICARDIUM.

By YAS KUNO (*Mukden*).

(*From the Institute of Physiology, University College, London.*)

CONTENTS.

THE view generally taken of the function of the pericardium is that it prevents over-distension of the heart, but the only experimental work on its mechanical effect with which I am acquainted is that of Barnard(1). He showed that the pericardium is inextensible and that it can bear a pressure of $1\frac{1}{4}$ to $1\frac{3}{4}$ of an atmosphere, whereas the heart ruptures at a pressure of $\frac{3}{4}$ to 1 atmosphere. He also found that a heart without the pericardium is able to receive twice as much fluid as a heart with pericardium intact.

Recently an investigation of the effect of opening the pericardium on the gaseous exchanges of the heart has been made by Evans and Matsuoka(2). Their results afford an interesting corroboration from the chemical standpoint of the conclusions I have arrived at from a study of the purely mechanical results.

The experiments of Patterson and Starling(3) and of Patterson,

Piper and Starling(4) from this Institute, have proved the following facts. The work of the heart depends on the length of its muscle fibres at the beginning of systole. The length is therefore determined by the quantity of blood flowing into the heart during diastole. The output increases with the inflow within very wide limits, and a heart of 50 grms. is able to expel from 100 c.c. to 3000 c.c. of blood, according to the conditions of inflow. In these experiments the pericardium was always opened. In view of these results and those of Barnard, the question arises as to whether such a great variation in volume is possible in the heart with pericardium intact. I have therefore carried out the following experiments with a view to determining what, if any, difference exists in the function of the heart with and without pericardium, and in what way the pericardium acts as a protective organ to the heart.

Method. Nearly all the experiments were carried out on the heart-lung preparation on dogs, and the preparation was made in the same way as that used in the experiments of Patterson and Starling, though with the following alteration. In their experiments the venous reservoir was usually fixed at a height of 35 cms. The venous pressure could be altered to any desired extent by screwing or unscrewing the clip on the rubber tube connecting the reservoir with the venous cannula. In this way the venous pressure is determined by regulating the blood supply from the reservoir. So long as the heart is working under constant conditions, the venous pressure will also remain constant. If however the heart's action for any reason becomes more powerful, so that the heart drives out more blood and can deal with a larger inflow, the conditions become somewhat abnormal, since the inflow is limited by the screw clip on the supply tube. The venous pressure measured near the right auricle therefore sinks and may become abnormally low for the heart. If the heart's action is weakened, the result is exactly the opposite. This arrangement is therefore not well adapted for experiments such as those contained in the present paper, in which we expect changes in the efficacy of the heart's action. We may probably assume that in the normal animal the venous supply to the heart is always adequate and we must endeavour to imitate this condition in any artificial schema. For this purpose I have connected the blood reservoir with the venous cannula by means of a very wide rubber tube, and, omitting the screw clip, have used changes in the height of the reservoir to bring about alterations in the venous pressure. In this arrangement there is always an ample supply of blood

to the heart whatever changes may have occurred in the action of this organ.

Unfortunately the advantages of this arrangement were not apparent to me at the beginning of the investigation. A large number of my experiments were made with the first arrangement, and I have only been able to repeat a certain proportion of them with the improved method.

In order to determine the function of the pericardium I have carried out experiments in two ways. In some experiments I have first made a series of determinations with the pericardium intact, then opened the latter and repeated the experiments, comparing the results in the two series. In most cases however I have arranged to open or close the pericardium at will, so that each set of conditions could be repeated first with closed and secondly with opened pericardium. For this purpose the pericardium was slit up in the middle line in front. A narrow strip of celluloid of the same length as the wound in the pericardium, provided with three or four holes, was then laid on each side of the wound and threads passed through the holes in the celluloid and the corresponding edge of the wound. On drawing these threads together and clamping them with small spring forceps, the pericardium can be closed almost as effectively as in the normal condition.

In my experiments the pressures in the aorta and in the vena cava, the output of the left ventricle, and in many cases the pressure in the pulmonary artery, were simultaneously determined.

1. *The influence of opening the pericardium on the arterial pressure, the venous pressure, and the output of the left ventricle.*

A. *The influence on the venous pressure.* So long as the action of the heart remains constant, the height of the venous pressure is the chief factor determining the volume of the heart, which, as we have seen, is so important for its activity. If however the rapidity of diastole, or in other words the resistance to the dilatation of the heart, is altered for any cause, the heart under the same conditions will receive a correspondingly altered amount of blood during the diastolic period, so that if the inflow from the venous reservoir is not altered, the venous pressure must rise or fall. Any condition therefore which aids the diastolic relaxation of the heart must lower the venous pressure.

We may make the following assumptions: when the inflow into the normal heart is very large, its volume becomes also large, so that

the pericardium must be strongly stretched. In this case the diastole of the heart must be interfered with to some extent by the presence of the pericardium. The venous pressure therefore rises considerably, but in spite of this rise the heart cannot receive as much blood during diastole as could enter it from the venous reservoir. If now the pericardium be opened, the heart must dilate under the high venous pressure. It will therefore receive more blood during diastole and expel more during systole. At the same time the venous pressure must fall and the arterial pressure rise. This assumption however is only justified if the heart under physiological conditions is able to dilate to a volume considerably greater than that of the pericardial cavity. We have therefore to decide in the first place whether this assumption is really justified by the results of experiment,—whether in fact the heart is influenced by the pericardium only when its volume is largely increased or whether the effect of the pericardium on the heart is appreciable under all conditions. The answer to this question is given to a large extent by the observation of changes in the venous pressure.

In all my experiments the venous pressure was determined in the vena cava by means of a cannula which was passed into this vessel from the abdomen and was connected with a water manometer. The effect of alterations in the inflow was observed with pericardium alternately closed and opened. It was found that in all cases when the inflow of blood was not excessive, the venous pressure sank each time the pericardium was opened. This fall of pressure was noticeable even when the inflow was so small, that the output of the heart amounted only to 190 c.c. of blood per minute. The extent of the fall of venous pressure varied considerably according to the amount of inflow, and also according as the inflow was varied by means of the spring clip or by alterations of the reservoir connected with the venous cannula by a wide rubber tube. We may speak of these two methods as methods 1 and 2.

When method 1 was employed the lowering of the venous pressure on opening the pericardium was much more striking than when the second method was used. Thus in the former case the venous pressure might sink to a fifth of its previous height on opening the pericardium, whereas with the second method it might fall only to a half. The reason for this difference I have already discussed. That the second method is more nearly analogous to the conditions which exist in the normal animal is shown by the results I have obtained on animals with opened thorax which will be discussed in Section D. In both methods

however the results are in the same direction and show that on opening the pericardium there is a fall of venous pressure which is very small

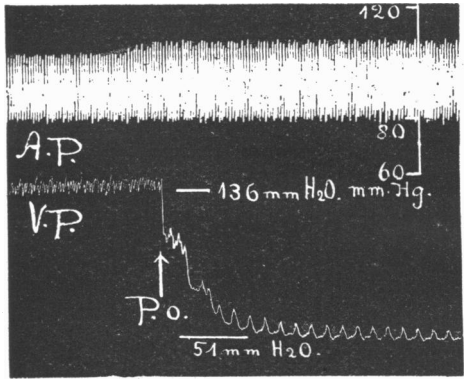

Fig. 1 (Tab. 1). Influence of opening the pericardium. Venous inflow moderate. (Method 1.) P.o. Pericardium opened.

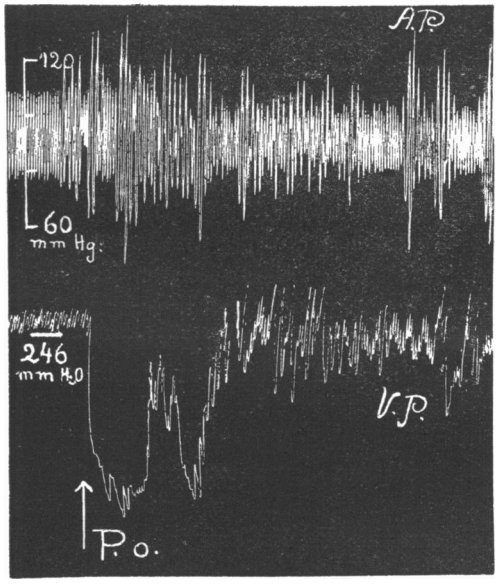

Fig. 2. (Tab. 1). Influence of opening the pericardium. Very large inflow. (Method 1.)

when the inflow is small and becomes more marked when the inflow is increased up to a certain limit. If the venous inflow is increased beyond

this limit, the fall of venous pressure becomes less marked, so that, finally, opening the pericardium causes no further fall of venous pressure. In many cases in fact there may be a distinct rise, associated with irregularity or failure of the heart's action. In both cases the venous pressure rose, as one would expect, with increase in the venous inflow. The rate at which this increase occurs is however much slower when the pericardium is opened than when it is shut, until the amount of inflow becomes excessive, when the venous pressure rises in both cases to an almost equal height. Two examples of such observations are illustrated by the following tables (Tables 1–2, cp. also Tables 4 and 7 and the Figs. 1, 2, 7, 8, 9, 12, 17, 18).

Figs. 1 and 2 were obtained from Exp. 1. In Fig. 1 the output before the opening of the pericardium was 1200 c.c., in Fig. 2, 2000 c.c. per minute. In Fig. 1 the venous pressure seemed to sink considerably, while in Fig. 2 it actually rises after a temporary fall and is very irregular.

It is easy to understand the reason for the fall of venous pressure which occurs on opening the pericardium when there is a considerable inflow into the heart. Since the heart cannot dilate to its full extent while constrained by the pericardium, each heart beat is not sufficiently extensive to deal with the whole of the blood which could enter it from the venous reservoir during the time of diastole. When the pericardium is opened the heart can dilate, so that the energy of its contraction is increased. It can thus take up more blood and send out more blood than it did before, bringing about in this way a fall in the venous pressure and a rise in the output. The effect of opening the pericardium when the inflow is minimal is a little more difficult to understand, since in this case the heart cannot dilate to the limit set it by the pericardium. One must remember however that when the thorax is opened the whole weight of the heart is sustained by the pericardium. When this is widely opened, the heart tends to fall during diastole into the back of the chest. It seems possible that when the inflow is small and the distending pressure acting on the auricles is also minimal, the weight of the ventricles may drag upon the pericardium as this lies on the auricles, and so impede the dilatation of these cavities and the inflow of blood into the heart. It is doubtful whether this effect of the pericardium on the limitation of inflow with a very low venous pressure would be appreciable in the unopened chest. Under the conditions of experiment however it seems that the so-called after-loading effect of the venous supply to the heart does not occur when

the pericardium is intact and that there is always, with the chest opened, a certain positive pressure exerted on the walls of the heart during diastole by the inflowing blood. This resistance of the pericardium to the diastolic expansion must increase with any increase in the heart volume, so that the fall of venous pressure observed on opening the pericardium must be greater the larger the venous inflow. On the other hand the rise and irregularity of the venous pressure, which may be observed on opening the pericardium when the venous inflow is excessive, are due to failure of the heart, and will have to be discussed later.

I have often noticed that on opening the pericardium the venous

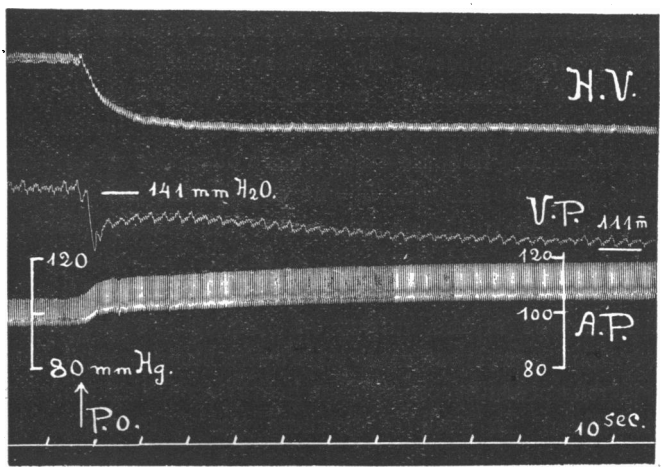

Fig. 3 (Tab. 2). Influence of opening the pericardium. Inflow large. (Method 2.) H.V. heart volume; V.P. venous pressure; A.P. arterial pressure.

pressure sinks at once to a certain extent and then continues to sink slowly for one to two minutes afterwards. (See Fig. 3.) In this experiment (Tab. 2) the output of the heart before opening the pericardium was 1480 c.c., after opening 2080 c.c. per minute. After this initial fall the venous pressure continued to sink during the next two minutes from 121 to 111 mm. H_2O, while the arterial pressure during the same period rose from 107 to 111 mm. Hg, pointing to a gradual increase in the efficiency of the heart's action. I am inclined to think that this slow improvement may be due to an improvement in the circulation through the coronary vessels, and that the pressure of the pericardium when marked may actually impede the flow through these

vessels. Further experiments however will be required before this can be regarded as established.

B. *The influence on the output.* It has been shown that the rate of diastolic filling is increased in every case by opening the pericardium. If the height of the venous reservoir and the diameter of the connecting tubes remain unaltered, this quickening of diastole always brings about an increase in the heart volume and therefore an increased output at each beat, since increased volume of the heart means increased functional capacity. All the experiments which were carried out by method 2 confirm this conclusion as to output, which was found to increase *pari passu* with the lowering of the venous pressure (cp. Tables 2, 7, and Figs. 7, 8, 17). On the other hand somewhat different results were obtained in the experiments in which the venous inflow was controlled by altering the screw clip. When the inflow was considerable, the outflow was increased by opening the pericardium. If however the inflow was small, the output was hardly altered, if at all, by opening the pericardium, and in some cases I have observed a slight diminution in the output. For example, in one experiment (heart 64 grms.) the output fell from 455 to 428 c.c. per minute; in another (heart 58 grms.) from 600 to 566 c.c. and from 365 to 323 c.c. per minute. In every case however where the inflow was over 600 c.c. per minute I obtained a definite increase in the output on opening the pericardium. The only exceptions were those cases where the blood supply was so great that the venous pressure rose and the heart became irregular on opening the pericardium. In such cases there was a corresponding fall of output.

The explanation of these results presents no difficulty. If method 1 is used with a minimal inflow which is insufficient to dilate the heart during diastole to the limits set by the pericardium, the heart is already putting out practically the whole of the blood which it receives and keeping the pressure low in the great veins. Removal of the pericardium therefore, although diminishing the pressure on the right auricle and ventricle and therefore causing a still lower venous pressure, does not alter the inflow and therefore cannot alter the output. The small diminution in output occasionally observed may be due to the increased fraction of the blood which may pass through the coronary vessels after removal of the constraint of the pericardium. On the other hand if the second method be used, *i.e.* a plentiful available supply under a very low reservoir pressure, the small difference in venous pressure caused by opening the pericardium is sufficient to affect materially

the head of pressure which is responsible for the filling of the heart during diastole. In this case therefore opening the pericardium causes a distinct increase in output (cp. Table 2).

C. *The influence on the arterial pressure.* In the heart-lung preparation the resistance in the artificial circulation is maintained constant, so that the height of the arterial pressure is dependent on the output of the heart. The influence of the pericardium on the arterial pressure therefore depends on its influence on the output. Thus with small venous inflow (method 1) opening the pericardium had practically no influence on the arterial pressure, whereas with an adequate venous supply a marked rise of arterial pressure accompanied the increased output. The increased output at each beat is shown on the arterial pressure tracing by the larger oscillations of the manometer float with each beat. The varying influence of opening the pericardium on the arterial pressure according to the method adopted for regulating the venous inflow is shown in Figs. 4 and 5. Fig. 4 is derived from the same animal as Fig. 3 (Tab. 2). The weight of the heart was 61 grms. and the venous inflow only 355 c.c. per minute. The second method was however adopted so that there was a potential supply of blood to the heart. We see in this figure a definite rise of arterial pressure after opening the pericardium. In Fig. 5 from a heart of 50 grms. the venous supply was 695 c.c. per minute and the venous inflow was controlled by a screw clip. In this case hardly any change in the blood-pressure followed the opening of the pericardium although there was a definite fall of venous pressure from 53 to 25 mm. H_2O. On the other hand, if the blood supply is so large that there is no fall of venous pressure on opening the pericardium, the arterial pressure also fails to rise and generally becomes irregular, as we see in Fig. 2.

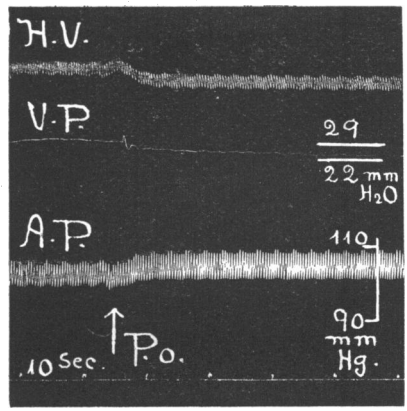

Fig. 4 (Tab. 2). Influence of opening the pericardium. Inflow very small. (Method 2.)

D. *Experiments on the whole animal with open thorax.* In order to investigate the condition of the venous supply to the heart in animals in a more normal condition, I have carried out some experiments on

dogs, anæsthetised with chloroform ether, in which the chest was opened in the middle line. The pericardium was opened as usual and fitted with the celluloid and threads so that it could be closed at will. The arterial pressure was measured in the carotid artery and the venous pressure in the superior vena cava. In this condition the heart is at atmospheric pressure so that the dilating force acting on the heart wall during diastole must be about 30 mm. H_2O lower than normal, corresponding to the negative pressure of the thoracic cavity. The condition of such an animal is therefore comparable with those in the heart-lung preparation in which the venous pressure is maintained low.

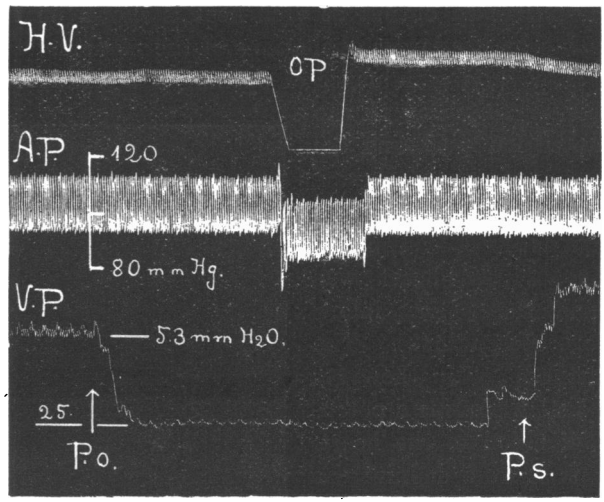

Fig. 5. Influence of opening the pericardium. Inflow moderate. (Method 1.) P.s. pericardium closed. O.P. output measured.

In the three experiments which I carried out in this way I found a definite rise of arterial pressure and fall of venous pressure on opening the pericardium. An example is given in Fig. 6, which is derived from an animal of 6·25 kilos. The pericardium was opened for a period of $1\frac{2}{3}$ minutes. The blood-pressure rose from 123 mm. Hg to 138–130 mm. Hg, while the amplitude of its oscillations increased from 8·5 to 13 mm., and the venous pressure sank from 40 to 19 mm. H_2O.

These results agree with those of the experiments on the heart-lung preparation made by method 2 with an adequate venous supply of blood, and we may therefore conclude that this method corresponds

more nearly to natural conditions than the one in which the flow is controlled by the screw clip. The same results of opening the pericardium were observed when the pressure was raised about 20 mm. Hg by compression of the abdominal aorta.

In order to make the results clearer as to the influence of the pericardium on the arterial pressure, venous pressure and the output of the heart, I have represented the results of Table 2 schematically in the accompanying figure (Fig. 7)[1].

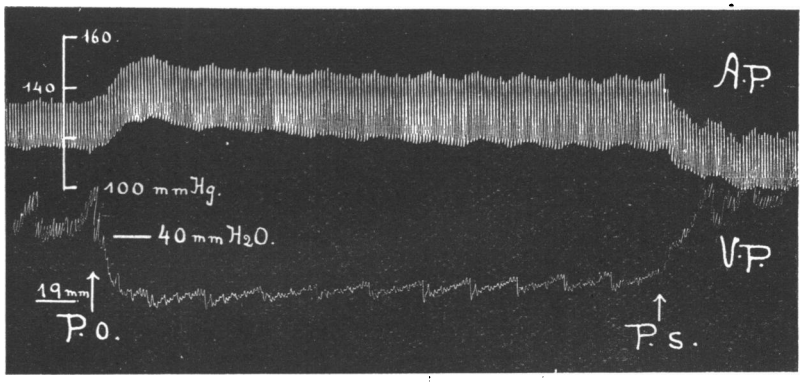

Fig. 6. Experiment on the intact animal with open thorax. A.P. blood-pressure in carotid artery; V.P. pressure in superior vena cava.

2. *The relation between venous pressure and output of the heart when the venous inflow is altered.*

It is evident from the preceding section that in the heart free from the pericardium a smaller venous pressure is necessary to maintain a given output of blood than in the normal heart. It becomes interesting to enquire whether this apparently favourable influence of opening the pericardium on the output applies under all conditions, and especially whether the maximal output of which the heart is capable is much greater in the heart without pericardium than in the normal heart.

Considerable difficulties arise in endeavouring to investigate the influence of the pericardium on the heart's action when using a large venous inflow. Under these conditions the heart, on opening the

[1] In this and all the following figures the continuous lines represent the condition by intact or closed pericardium, while the dotted lines represent the condition after opening the pericardium.

pericardium, enlarges considerably and it may become difficult or even impossible to close the pericardium again. Moreover, we find that the heart is very often permanently damaged as a result of the dilatation, so that after closing the pericardium it no longer functions normally. Even the method which I have often adopted, of carrying out a series of experiments on the heart, first with the pericardium intact and then after opening the latter, presents disadvantages, since the heart in

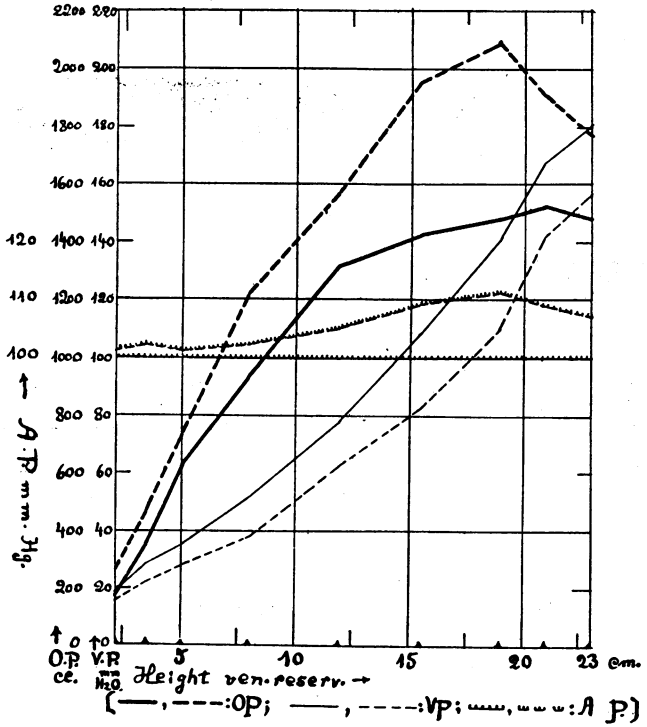

Fig. 7 (Tab. 2). Schematic representation of the influence of the pericardium on the arterial pressure, venous pressure and output with different venous supplies. The A.P. was kept constant by altering the resistance. The figures represent the percentage value of A.P.

many cases is fatigued or even damaged in the first series of experiments and its action in the second series is thus permanently weakened. This permanent weakening is shown by the fact that in the second series, with open pericardium, a higher venous pressure may be necessary than in the first series to maintain the same output of blood. My results therefore as to the connection between the venous inflow and

the maximal output under these two conditions must be regarded as nearly approximate. The connection between venous pressure and output is shown in Fig. 8, drawn from the results of three experiments, and is also evident in Figs. 12, 17, 18, and Tables 5, 6 and 7.

In Table 5 the pericardium was first maintained intact through a series of observations, and then opened and the series repeated. In Tables 1, 2 and 7, each condition of blood flow was repeated first with open and then with closed pericardium. In every case the venous pressure rose as the venous inflow was increased, but the rise was much steeper with closed than with open pericardium. In every case too the output was increased with rise of venous pressure up to a certain limit, above which the output not only did not increase but actually diminished as the venous pressure rose still higher. The maximal output of the heart without pericardium was in some experiments smaller than with closed pericardium. This might however have been the result of fatigue since in the other experiments the relationship was the reverse of this. In Table 2 the increase in the maximal output resulting from the opening of the pericardium was extremely pronounced. These results may be seen from the following table.

No.	Weight of heart gms.	Maximal output c.c. per min.		Venous pressure mm. H_2O	
		Pericardium closed	Pericardium open	Pericardium closed	Pericardium open
1	48	1945	1750	132	62
2	52	1750	1310	284	86
3	50	1530	1780	241	196
4	44	1460	2000	292	180
5	61	1530	2080	169	111

In this table only those cases are given in which the maximal output could be determined satisfactorily under both conditions of the heart.

There can be no doubt that the factor limiting the maximal output under normal conditions must be the capacity of the pericardium. When the pericardium is absent, other limiting factors come in, *e.g.* the optimum dilatation of the heart with increased length of the muscle fibres, incompetence of the valves or actual injury of the heart muscle. As we shall see, hæmorrhages are constantly found in any heart which has had to deal with a large venous inflow in the absence of a pericardium. In no case have these hæmorrhages been absent where the output of the heart was raised to a maximum.

It is evident from the above table that the maximum output is obtained in both conditions of the pericardium by very varying venous pressures, although always much lower with open than with closed pericardium. In Nos. 3 and 4 the venous pressures were much higher

than in the other experiments, but in these two cases the heart suddenly became injured, and the optimum venous pressure for a heart without pericardium must be much lower than these figures. We can at any rate say that the maximum distending force which may occur in the normal animal, namely, 35 mm. H_2O, is above the optimum for the heart freed from the pericardium. We may therefore conclude that so long as the venous pressure is normal, removal of the pericardium only improves the functional capacity of the heart when the blood supply during diastole is maintained low. As soon as the blood supply is large enough to raise the venous pressure in the great veins to a height which represents only one-third to one-half of the maximal distending

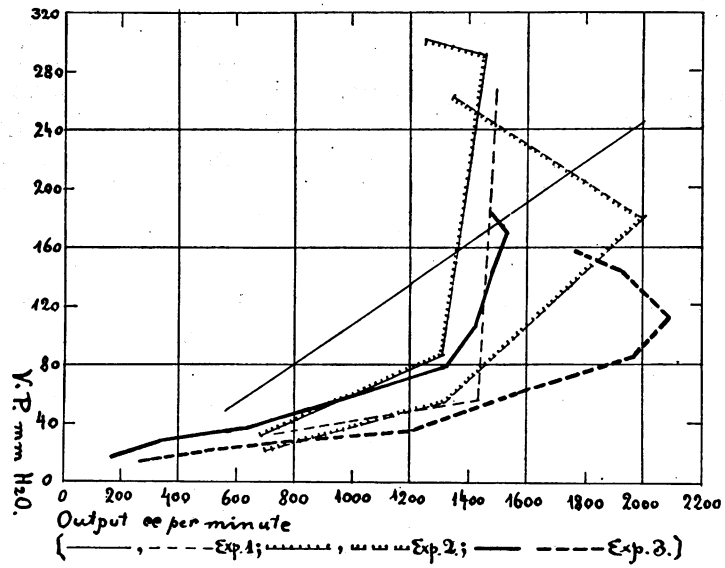

Fig. 8. Effect of increased inflow on the output and venous pressure.

force in the normal animal, the condition becomes dangerous for a heart deprived of its pericardium. The maximal functional capacity of the heart without pericardium cannot be raised over that of the heart with pericardium without considerable danger. On the other hand, so long as the pericardium is intact, a high venous pressure presents very little risk to the heart. It is worth noting too that in experiments on the heart-lung preparation, if the pericardium be removed, it is not safe to put the blood reservoir at a height of more than about 15 cms. above the level of the heart. The mechanical conditions therefore produced are closely similar to those present in the heart in the normal animal.

3. *Influences of changes in arterial pressure on venous pressure and output.*

Markwalder and Starling(5) have shown that the heart expels a constant quantity of blood under very varying arterial pressures, provided only that the venous supply remains constant. Each rise in arterial pressure however is attended with a corresponding increase in the volume of the heart, and it is the increase in length of the muscle fibres thereby induced which is responsible for the increased functional

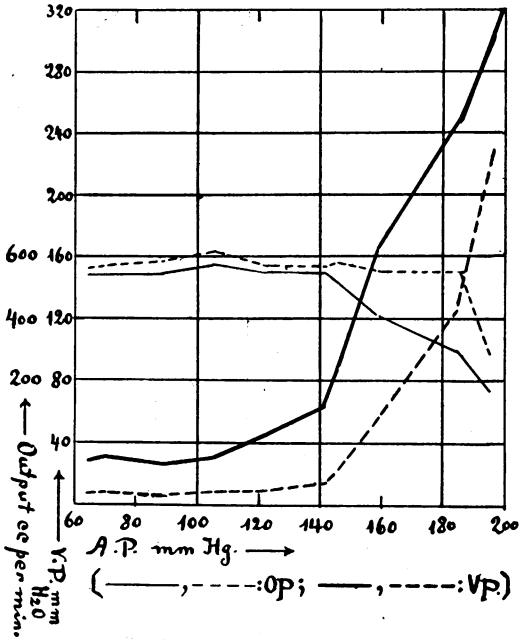

Fig. 9 (Tab. 3). Influence of increase in the arterial pressure on the output and venous pressure.

capacity of the heart. These experiments were carried out on hearts in which the influence of the pericardium had been removed, and it becomes interesting to see how far they apply to the heart still contained within its pericardium. I repeated six such experiments, one example of them is given in Table 3 and in Fig. 9, and another in Fig. 10. It will be noticed that there is a great difference in the effect of rise of arterial resistance on the output under these two conditions. Where the pericardium was opened, the output only diminished slightly as the arterial

pressure was raised, and this slight diminution may be ascribed, as shown by Markwalder and Starling, to the increased flow through the coronary vessels. With the pericardium intact, the pressure could be raised to 130–140 mm. Hg without producing greater alterations than those obtained in the heart without pericardium. A further rise of arterial pressure however brought about a very considerable diminution in the output. Thus in an experiment (Table 3) at a pressure of

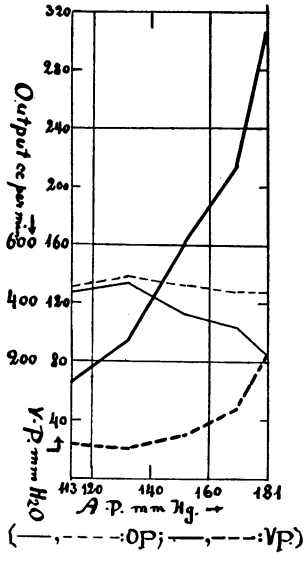

Fig. 10. Influence of increase in the arterial pressure on the output and venous pressure.

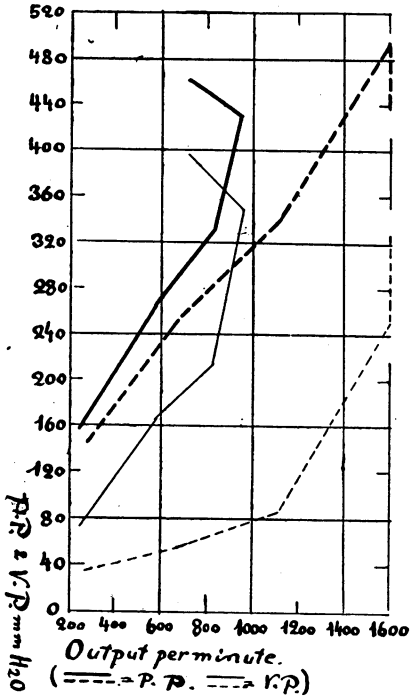

Fig. 11 (Tab. 4). Effect of increased inflow on the pulmonary pressure and venous pressure. Pericardium opened and closed alternately.

140 mm. Hg, the difference between the output of the left heart under the two conditions of the pericardium was only 12 c.c. per minute, whereas when the arterial pressure was raised to 186 mm. the output from the heart with pericardium intact was 245 c.c. less than in the heart without pericardium. In another experiment (Fig. 10) the difference with a pressure of 132 mm. Hg was only 27 c.c. per minute; with a pressure of 181 mm. Hg 209 c.c. per minute. In another

experiment at a pressure of 114 mm. Hg the difference was practically nothing, but with a pressure of 148 mm. Hg the difference amounted to 250 c.c.

It is impossible to explain these differences by alterations in the fraction of the blood passing through the coronary vessels. Indeed, we should expect the circulation through these vessels to be accelerated rather than hindered by opening the pericardium. The results as far as the venous pressure is concerned, agree well with those on the output. The venous pressure rose constantly with the rise of the arterial pressure, but with closed pericardium the rise was much steeper, and the difference between the two conditions of the pericardium was especially marked at high arterial pressures. Thus in Tab. 3 the rise of venous pressure with an alteration of arterial pressure from 65 to 122 mm. Hg was not very marked under either condition of the pericardium. But on raising the arterial pressure still further, there was a very rapid rise of venous pressure when the pericardium was closed, and the point at which this rapid rise occurs is the same at which the output begins to fall off, showing that the rise of venous pressure depends on a failure of the left ventricle to pass on the blood coming to it (Fig. 9).

4. *The influence of the pericardium on the pulmonary circulation.*

The effect of opening the pericardium on the right ventricle varies according to the venous inflow. If the latter is small the intracardial pressure sinks when the pericardium is opened, but there is no increase in the inflow, so that the output of the right ventricle is either unaltered or even smaller, as has been shown in Section 1 (Method 1). The pressure in the pulmonary artery also remains constant or sinks slightly. If the venous inflow is large, the augmentation of the beat of the heart which follows opening the pericardium, causes an increase in the output of the right ventricle, and the pulmonary arterial pressure rises. If the venous inflow becomes too large for the right ventricle the pulmonary pressure ceases to rise and indeed falls, although the left ventricle may be acting quite well.

I have carried out four experiments on the pressure in the pulmonary artery. To measure this a T-cannula was introduced into the pulmonary artery, as described by Führer and Starling(6), and connected with a water manometer, the excursion of which was registered by connecting the top of the tube with a piston recorder. In order to introduce the

cannula the pericardium had to be slit up for a certain distance, but the wound thus made was carefully sewn up again after the introduction of the cannula, and the pericardium incised in the middle line, so that it could be opened or closed in the usual way. Two examples of these experiments are contained in Tables 4, 5, and in Figs. 11 and 12.

It must be mentioned that they were all carried out with the blood reservoir at a constant height, in which method the venous inflow on

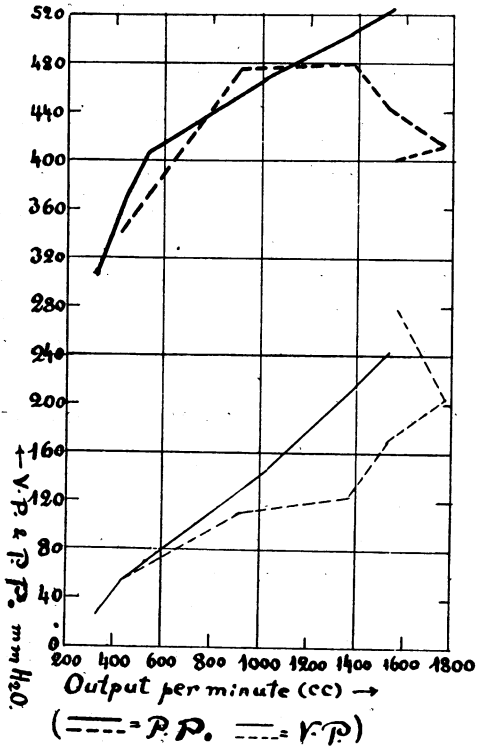

Fig. 12 (Tab. 5). Effect of increased inflow on the pulmonary pressure and venous pressure. Pericardium first intact, then opened.

opening the pericardium must be regarded as inadequate so long as the venous pressure is low. As a result in an experiment (Table 4, Fig. 11) we see that the pulmonary pressure sank when a small venous inflow was diminished by opening the pericardium. It is doubtful whether such a result would be obtained in experiments carried out with an adequate venous supply. In this experiment we see that the pulmonary pressure could be altered in three different ways by the

opening of the pericardium. 1. With a small venous inflow of 254 or 580 c.c. per minute the pulmonary pressure sank on opening the pericardium, namely, from 158 to 144 mm. H_2O and from 262 to 250 mm. H_2O respectively. It is noteworthy that the output of the left ventricle in both these cases increased on opening the pericardium, showing that the supply to this ventricle was adequate. 2. With a moderate inflow of 830 or 962 c.c. per minute the pulmonary pressure, after a transitory fall, rose gradually. 3. On further increasing the venous inflow the

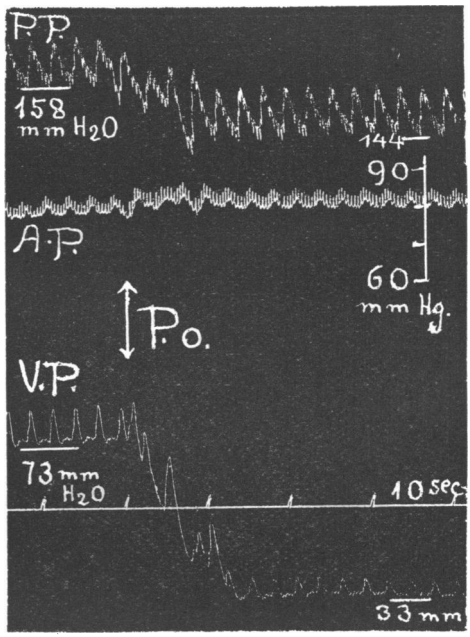

Fig. 13 (Tab. 4). Influence of the pericardium on the pulmonary pressure, arterial pressure and venous pressure. Venous supply small.

venous pressure rose, but the pulmonary pressure sank on opening the pericardium[1]. Figs. 13 and 14 are examples of cases 1 and 2. In Fig. 13 the pulmonary pressure before opening the pericardium was 158 mm. H_2O and the venous pressure 73 mm. H_2O; after opening the pericardium the pulmonary pressure sank to 144 mm. H_2O and the venous pressure to 33 mm. H_2O. In Fig. 14 before opening the

[1] In this experiment the pericardium was too much stretched as a result of the introduction of the cannula. This must be regarded as the reason for the very small output obtained with closed pericardium.

pericardium the pulmonary pressure was 326 mm. H_2O and the venous pressure 232 mm. H_2O; on opening the pericardium the pulmonary pressure rose to 340 mm. H_2O, while the venous pressure gradually sank to 84 mm. H_2O.

In all these experiments the pericardium was alternately closed and opened. In Tab. 5 the pericardium was first intact and was then opened before the second series of observations was taken. The results however were almost the same. The maximal pulmonary

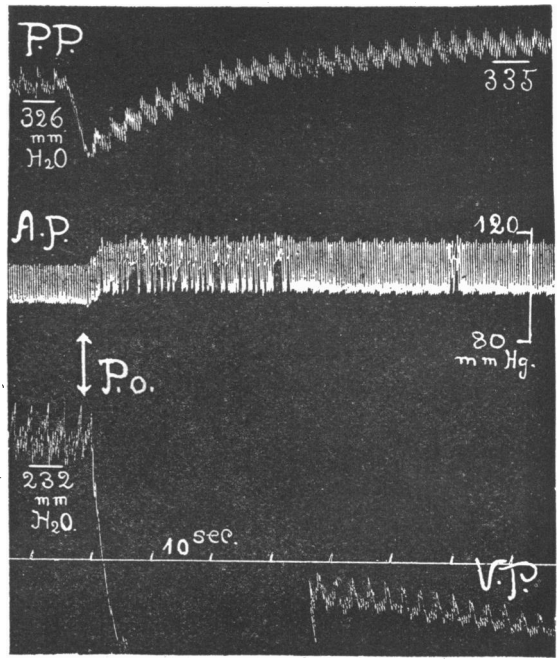

Fig. 14 (Tab. 4). Influence of the pericardium on the pulmonary, arterial and venous pressures. Venous supply moderate.

pressure after opening the pericardium never reached the height of that observed with the pericardium closed, and the pulmonary pressure sank with a further increase in the venous inflow. It seems therefore that the functions of the right ventricle are very easily disturbed by opening the pericardium if the venous inflow is considerable. In an experiment especially, the height of the venous pressure with a big venous inflow rose considerably above that of the pulmonary artery

(pulm. pres. 276, ven. pres. 346 mm. H_2O) showing considerable interference with the efficiency of the right ventricle.

Figs. 15 and 16 are taken from the same heart (Table 5), the pericardium being intact in 15 and open in 16. At the arrow the venous inflow was increased. In Fig. 15 with an increase from 1380 to 1530 c.c. per minute, the venous pressure rose from 215 to 241 mm. H_2O. In Fig. 16 with an increase of venous inflow from 1520 to 1780 c.c. per minute, the venous pressure rose from 169 to 201 mm. H_2O. The pulmonary pressure was however differently affected in the two cases.

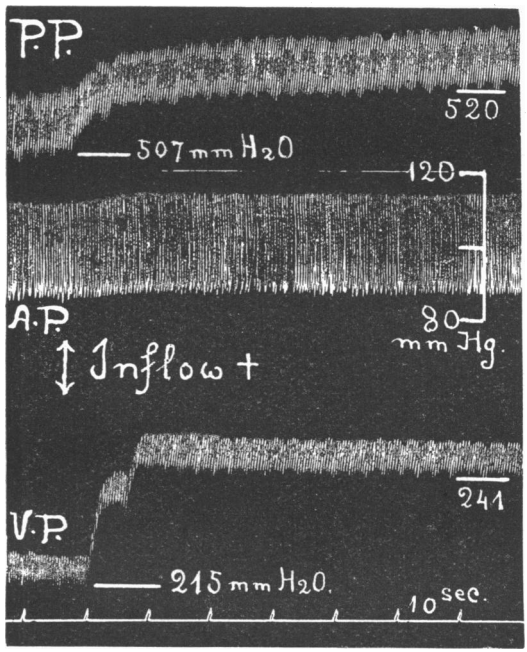

Fig. 15 (Tab. 5). Effect of increased inflow. Pericardium intact.

In Fig. 15 it rose from 507 to 521 mm. H_2O, while in Fig. 16 it fell from 443 to 415 mm. H_2O.

In one experiment the maximal output of the left ventricle with open pericardium coincided with the highest point of the pulmonary pressure, showing that the maximal efficiency of the left ventricle is dependent on the state of activity of the right ventricle. A falling off in the output of the left ventricle with increasing venous inflow may be, and often is, due to the fact that the right ventricle becomes incompetent with

increasing venous inflow before there is any failure on the part of the left ventricle.

In one experiment the pulmonary cannula was tied in too near the heart so that it interfered with the closure of the semilunar valves. As is seen from Table 6 the output of this heart was very small in spite of a high venous pressure, though it increased slightly on opening the pericardium. This slight increase was I believe due to the effect of opening the pericardium on the left ventricle, since the pulmonary pressure with open pericardium did not rise higher than 300 mm. H_2O,

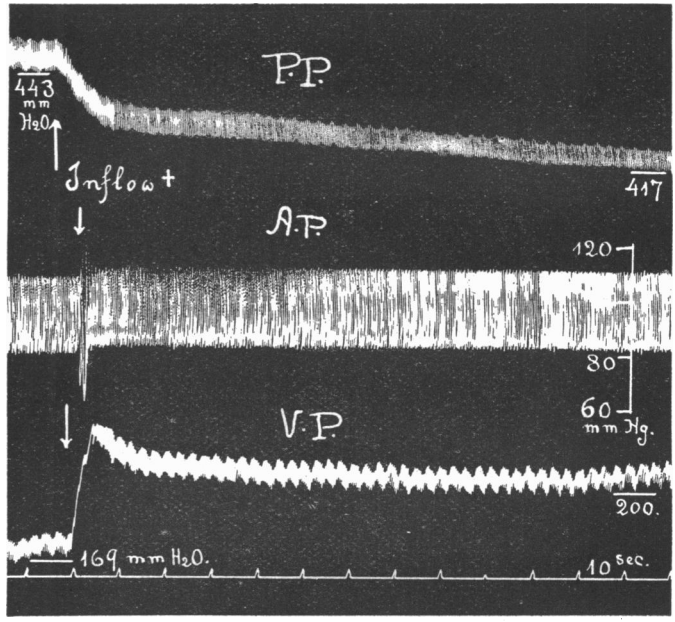

Fig. 16 (Tab. 5). Effect of increased inflow. Pericardium open.

although it reached 474 mm. H_2O when the pericardium was closed. The difference between the two conditions of the pericardium is thus much greater than we generally observe in normal hearts. As we should expect, the absence of the support afforded by the pericardium to the thin-walled right ventricle has greater effects in this case, where an abnormal strain is put upon this side of the heart.

In a number of experiments I have closed the blood reservoir with a rubber cork, as in Führer and Starling's experiments, and connected the air-space over the blood with a volume recorder. When

the amount of blood in the reservoir increases, the volume recorder marks a rise, so that its excursions were taken as indicating a diminution in volume of the heart, causing increased volume of blood in the reservoir and a rise of the volume recorder. My experiments have shown me that such a record is fallacious and cannot be regarded as indicating the average volume of the heart. There is no question that on opening the pericardium the systolic and diastolic volumes of the heart increase, and such an increase is also indicated by the volume recorder provided that the venous inflow into the heart is considerable. With a small venous inflow however, we see that the average volume of the heart apparently gets smaller on opening the pericardium since the volume of blood in the reservoir increases (cp. Fig. 5). This paradoxical result must be ascribed to the influence of opening and closing the pericardium on the pulmonary circulation. When the pericardium is opened, the left ventricle dilates and acts more strongly, so that it tends to empty the pulmonary veins. On the other hand, the right ventricle tends to become over-dilated and the pulmonary arterial pressure remains the same or falls slightly. The lungs therefore contain on the average less blood than they did when the pericardium was closed, and the amount of blood which is pumped out of the lung vessels more than counterbalances the increase of blood contained in the cavities of the heart, so that the residual blood in the venous reservoir increases although the average volume of the heart is also increased.

5. *The influence of change of rate of heart beat.*

Markwalder and Starling(7) have shown that provided the conditions of inflow remain the same, the output of a heart is within wide limits independent of the frequency of beat. With a slower rate each beat is more filled and sends out more blood than when the heart is beating at a greater rate, this increased diastole following at the slow rate being associated with a rise of venous pressure. Although the rate of relaxation of the heart becomes slower with diminution in frequency of beat, the absolute length of the diastole must be longer with diminshed rate, and this increase of the diastolic period is still more marked when the slowing is brought about, not, as in Markwalder and Starling's experiments, by cooling, but, as in the normal animal, in consequence of tonic inhibitory influence exerted by the vagi. The assumption of Yandell Henderson(8) therefore that by a greater frequency of heart beat the heart is in a systolic condition during a

greater length of time, must be regarded as justified. We have seen that the pericardium exercises under every condition of venous inflow a certain amount of resistance to the diastolic expansion of the heart, which increases with any increase in the heart volume. As the frequency of the heart beat diminishes, this influence of the pericardium must therefore increase, an increase which is borne out by the following experiments. In these experiments the frequency of heart beat was altered by changing the temperature of the inflowing blood, the other conditions of inflow being maintained constant. The effect of the

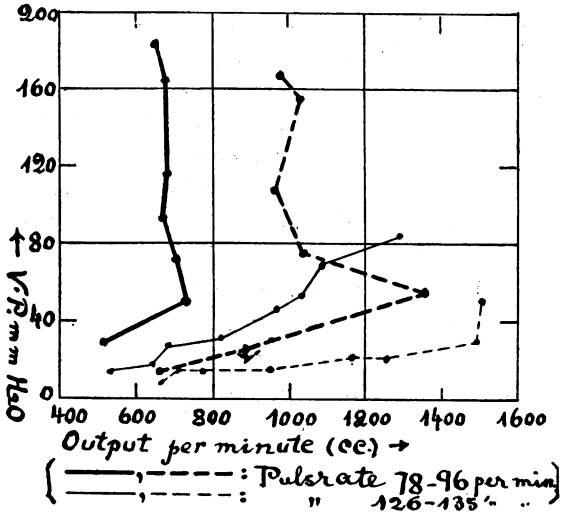

Fig. 17 (Tab. 7). Influence of the pulse rate on the function of the heart.

change in rate was observed both with open and with closed pericardium.

One example of such observation is shown in Table 7 and Fig. 17, and another one in Fig. 18[1].

It is quite evident from these results that the pericardium has much greater significance for the action of the heart with a slow than with a rapid rate of beat. Especially marked is the influence of the pericardium on the maximal output of the heart, which was almost identical at all rates in the absence of pericardium, whereas with intact pericardium the maximal output was much greater at the higher

[1] In this experiment the blood reservoir was not sufficient to bring about the rise in venous pressure necessary with the slower rate. This probably accounts for the great difference.

frequencies. The influence of rate on the maximal output at different frequencies is shown in the following table:

No.	Rate	Pericardium closed	Pericardium open
		Output in c.c. per minute	
1	Fast	1300	1360
	Slow	725	1520
2	Fast	2400	2600
	Slow	1340	2060
3	Fast	1965	1760
	Slow	950	1930

The statement of Markwalder and Starling that the output of the heart is independent of the rate only holds good therefore in the case of the heart without pericardium and does not apply to the heart under normal conditions. This result is especially interesting since it is well known that under normal conditions the pulse frequency is always increased when there is any increase in the velocity of the circulation.

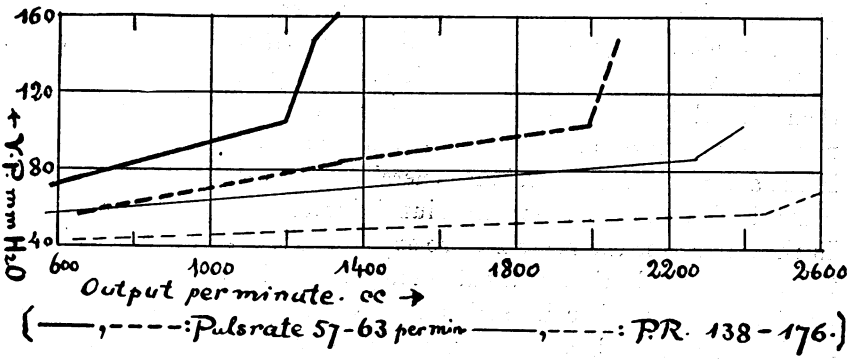

Fig. 18. Influence of the pulse rate on the function of the heart.

The influence of frequency on output, the importance of which has been especially insisted upon by Yandell Henderson, tends to be obscured in experiments on the heart-lung preparation on account of the very high frequency with which the heart, freed from all nervous connections, beats at the normal temperature. In the dog's heart this is generally about 160 per minute, whereas in the intact animal the rate would probably vary between 70 and 90 per min. So that in order to appreciate the influence of frequency on the mechanical performance of the heart, we have to compare the action of the normal heart with that of the heart-lung preparation beating at a temperature of 28°–30° C. Even then although the rate per min. approximates the normal rate,

the condition of the heart is not strictly comparable with that of the normal animal, since the process of cooling not only diminishes the rhythm but slows the rate of rise of contractile stress and prolongs the duration of the excitatory condition. By working with the heart at this temperature we learn however that at normal rates of beat it is impossible while the pericardium is intact to increase the total output to more than a moderate extent, however much we may raise the venous pressure or rate of venous inflow. There is only one way to increase the output further and that is by increasing the frequency of the heart beat.

6. *The importance of the pericardium as a protective mechanism for the heart.*

I have already shown in Section 1 that if the venous inflow be very large the effect of opening the pericardium may be to cause not a fall but an actual rise of venous pressure, the heart beat becoming at the same time irregular. As regards the maximal output which can be evoked by increase in the venous inflow, this is in many cases larger with open than with closed pericardium. A still further increase in venous inflow may however cause in both cases a diminution of output. This diminution in the absence of a pericardium generally occurs suddenly and is very marked, whereas when the pericardium is intact its onset is more gradual. In the latter case the heart may be exposed to too large a venous inflow for a considerable time without undergoing serious damage, the only effect being to weaken its action somewhat. In the absence of pericardium however when the heart has been once over-distended by too large a venous inflow, its insufficiency not only rapidly declines but it shows signs of permanent damage, so that its maximal performance can never again be brought to the same level. The first sign of this permanent damage is generally the irregularity of heart beat which may occur in various forms, of all of which I give examples in Fig. 2, and another more marked instance in Fig. 19.

The functional disturbance of the heart in the absence of the pericardium must therefore be ascribed not only to mechanical or physiological factors but to pathological changes wrought by the over-distension. These are of two kinds, namely, rupture of the heart muscle and insufficiency of the valves.

(*a*) *Valvular insufficiency.* We might expect to find insufficiency of the valves, especially of the auriculo-ventricular valves, when the

heart undergoes extreme distension. I have often seen in my experiments a sudden change in the curve of venous pressure on opening the pericardium. Its height rose considerably, the oscillations were increased and became synchronous with those of the arterial pressure curve, the same irregularities being present on both curves. In some cases this evidence of valvular incompetence appeared directly the pericardium was opened and later on disappeared, pointing to a recovery in the action of the heart, the venous pressure at the same time sinking to a lower level.

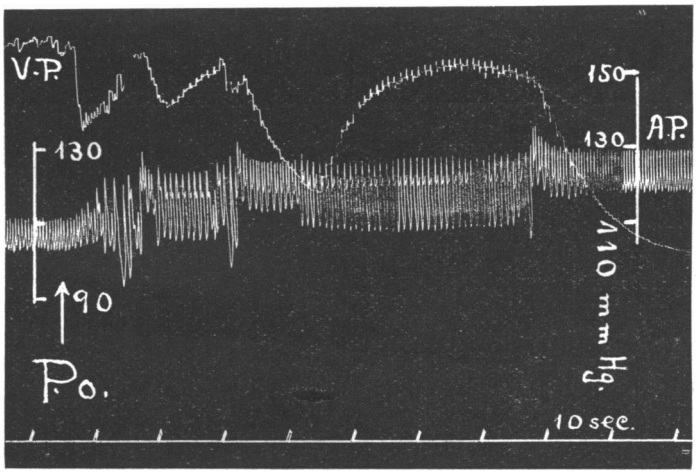

Fig. 19. Irregularity in the arterial and venous pressure on opening the pericardium. Venous supply fairly large.

A very marked case of this nature is shown in Fig. 20 which is derived from an experiment on a heart of 44 grms. In this experiment the output was 1260 c.c. per min. On opening the pericardium the venous pressure rose from 302 to 332 mm. H_2O, the amplitude of the oscillations was much increased, and the arterial pressure sank. After a minute the heart showed signs of recovery, the venous pressure sinking gradually to 264 mm. H_2O, its oscillations becoming smaller, while the arterial pressure rose above the original rate. The power of the heart muscle to recover its functional capacity under these conditions of strain must be ascribed to the same conditions as those which bring about the gradual increase of functional capacity of the heart often observed after opening the pericardium (cp. Sect. 1).

(*b*) *Rupture of the heart muscle.* In all my experiments I have examined the heart at the end for evidence of injury. Without exception I have found that whenever the heart had been subjected to a fairly large venous inflow with the pericardium opened, hæmorrhages were present in the outer and inner layers of the heart wall (cp. Sect. 2). These hæmorrhages were especially frequent in the inner wall of the left ventricle, over the whole surface of the heart, and in the septum of the auricles. In the inner wall of the right ventricle they were not always present, and I was never able to find them in the middle layer of the heart muscle. The hæmorrhages were generally small petechiæ but sometimes formed large hæmatones. These hæmorrhages were generally in the capillary region of the coronary vessels even lying

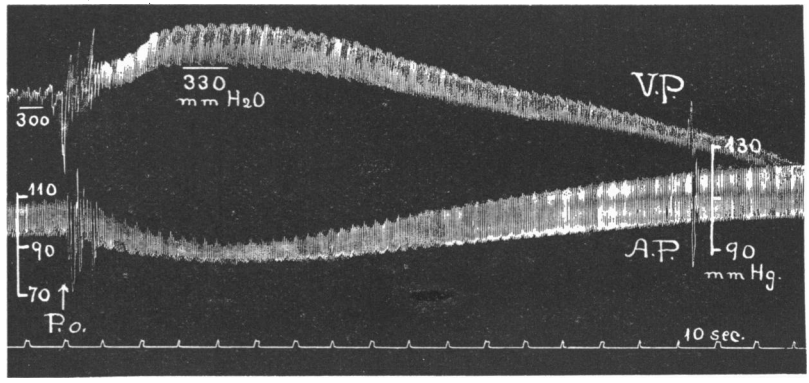

Fig. 20. Recovery of the heart's action after valvular insufficiency brought about by opening the pericardium.

near the outer face. I have tried to determine what was the limit of venous pressure or venous inflow required to produce these hæmorrhages but could not find any definite limit. In one experiment (heart weight 52 grms.) the maximum venous inflow employed during the experiment was 1360 c.c. per minute and the venous pressure 127 mm. H_2O. In another experiment (heart weight 97 grms.) the venous inflow was 2060 c.c. per minute and the venous pressure 153 mm. H_2O. Since in both these experiments hæmorrhages in the heart muscle were produced, it is evident that the level of venous pressure or venous inflow necessary to produce them must be lower than these figures. In one experiment the venous inflow was kept small,—500–600 c.c. per minute—while the arterial pressure was raised only to 138 mm. Hg,

that is to a height which must be often attained in normal life. In this case the venous pressure on opening the pericardium was only 18 mm. H_2O, yet even here hæmorrhages were easily seen on the surface of the heart and on the inner wall of the left ventricle, and there were minute petechiæ in the inner wall of the right ventricle. In two animals in which both chest and pericardium were opened, I injected adrenalin, producing a rise of blood-pressure to double its previous height. In one of these cases there were hæmorrhages in the left ventricle and on the surface of the heart; in the other case the heart was intact. In one dog in which the action of the heart was very weakened on account of clotting of the blood, I left the pericardium intact and raised the blood reservoir to a height of 40 cm., opening the screw clip to the extreme limit. I allowed the heart to work with this huge inflow for six minutes before stopping the experiment. In this case there were minute hæmorrhages on the inner wall of the left ventricle but nowhere else. As a rule, the hæmorrhages make their appearance as the strain of the heart is approaching the maximal capacity of working.

Conclusions.

As a result of the experiments above described, most of which were carried out on the heart-lung preparation, we may come to the following conclusions:

1. Opening the pericardium causes a fall of venous pressure, a rise of arterial pressure, and an increase of output. The extent to which these results are observed varies with the condition of the heart and is dependent on the volume of the heart. They therefore increase with increase in the amount of venous inflow. If however the venous inflow becomes very large, the effect of opening the pericardium may become smaller, and by too great an increase of venous inflow, the venous pressure instead of sinking may rise on opening the pericardium. In this case the output is also diminished and the action of the heart becomes generally irregular.

2. The maximal performance of the heart as measured by the output of the left ventricle varies according as the pericardium is opened or closed. With the pericardium open the output is generally larger than when the pericardium is closed, though the increase in output is in favourable cases only 25 % of the normal amount. When the work of the heart is raised to a maximum, this organ, if the pericardium is

absent, always suffers injury, generally in the form of hæmorrhage. The optimum venous pressure is much lower for the heart without than for the heart with pericardium. In the first case it is certainly not higher than 190 mm. H_2O, and in most cases considerably lower.

3. As the arterial pressure is raised, the heart in its normal state fails to drive out the blood it receives much sooner than when the pericardium is opened. The marked diminution in the output of the normal heart is observed even with an arterial pressure of about 130 mm. Hg, and with further rise of arterial pressure, the output rapidly diminishes. If on the other hand the pericardium be opened, the heart can drive out blood against an arterial pressure of 185 mm. Hg as well as it did against a lower pressure. It appears to me therefore that the increase in the maximal performance of the heart caused by removal of the pericardium is more marked when this is measured by alterations in the arterial resistance than when it is brought about by increase in the venous inflow.

4. The pressure in the pulmonary artery rises on opening the pericardium so long as the venous inflow is moderate. With a large venous inflow however, opening the pericardium may cause a fall of pulmonary pressure. It seems that in the absence of the pericardium, the right ventricle fails in its expulsive powers sooner than the left ventricle when the venous inflow is large. This relative failure of the right ventricle is probably the reason for the different effect of opening the pericardium on the maximal performance of the heart with rising arterial pressure and rising venous inflow respectively. When the mechanical work of the heart is increased by raising the arterial resistance, the output is dependent only, or at any rate in the first place, on the functional capacity of the left ventricle, whereas when the venous inflow is increased, the failure of the right ventricle limits the possibility of increase in the output of the left ventricle.

5. The influence of the pericardium is much more marked with a slowly than with a rapidly beating heart. The maximal output of the heart in the absence of the pericardium is certainly increased by increased frequency though the extent of the difference is not very great[1]. When however the pericardium is intact, the maximal output of the slowly beating heart is only one-half that which may be obtained with the heart beating at its maximum rate, however high the venous

[1] Patterson and Starling have shown that provided the venous pressure remains constant, the output of the heart is greater with a rapidly beating heart than with a slowly beating heart.

pressure rises. What I have here called the slow beat of the heart, corresponds roughly to the frequency of the heart beat in the normal animal. It is evident therefore that under normal circumstances the heart cannot put out more than a moderate amount of blood unless its frequency is increased. Thus not only an increase of venous inflow but also a rise in the frequency of the heart beat are necessary conditions for the maximal performance of the heart. Under conditions of ordinary life this rise in pulse rate in times of stress is provided for by amongst other factors a discharge of adrenalin into the circulation.

6. Whenever the heart without pericardium is submitted to a large venous inflow or has to beat against a high arterial pressure, hæmorrhages occur in the heart wall and frequently also valvular insufficiency. The hæmorrhages occur both in the outer and inner layers of the heart wall and are specially frequent in the inner wall of the left ventricle. This injury of the heart muscle may possibly play a part in limiting the functional maximal performance of the heart without pericardium.

We may conclude that so long as the venous pressure remains low, the function of the heart is improved by opening the pericardium. In the absence of the pericardium it is difficult however to increase the work of the heart above its normal level without danger to the organ, which may occur in the form of hæmorrhages or of valvular insufficiency. Thus a heart in the absence of pericardium is already in danger when it is submitted during diastole to a dilating force or venous pressure which is only one-third to one-half of that found under normal conditions. When the pericardium is intact, the heart, in order to perform a certain amount of work, requires a higher venous pressure than when the pericardium is opened. When however the venous pressure is at its normal height, it can perform as much work as the heart without a pericardium, and it does this without any danger to itself. The existence of the pericardium is thus necessary for the unimpaired working of the heart in normal life.

I desire to thank Prof. Starling for his suggestions, advice and help throughout the course of this research.

Protocols.

The following protocols give the details of seven out of the 30 experiments which were made.

Table 1 (Exp. 3). Dog. 7·5 kilos. Heart 64 grms.

Venous supply	Arterial B. P. (mm. Hg)			Pressure V. C. (mm. H_2O)			Output (c.c. per minute)			Pulse rate per minute	Temp. °C.
	Pericard. shut	Pericard. opened	Difference	Pericard. shut	Pericard. open	Difference	Pericard. shut	Pericard. opened	Difference		
Moderate	85	85	0	50	32	− 18	575	730	+ 155	105	34·5
+	91	93	+ 2	136	51	− 85	1200	1430	+ 230	105	34·5
+ (full)	92	?	?	246	270	+ 24	2000	1500	− 500	105	34·5

Table 2 (Exp. 30). Dog. 7·5 kilos. Heart 61 grms.

Height of venous reservoir (cms.)											
2·3	101	102	+ 1	19	16	− 3	182	270	+ 88	132	33·5
3·5	102	104	+ 2	29	22	− 7	355	475	+ 120	158	34·0
5·0	98	99	+ 1	35	28	− 7	630	730	+ 100	130	34·0
8·0	96	98	+ 2	52	38	− 14	940	1210	+ 270	138	35·0
12·0	98	103	+ 5	79	64	− 15	1320	1570	+ 250	146	35·2
15·5	100	109	+ 9	109	83	− 26	1420	1960	+ 540	140	35·2
19·0	100	111	+ 11	141	111	− 30	1480	2080	+ 600	143	35·0
21·0	109	118	+ 9	169	142	− 27	1530	1920	+ 390	132	34·0
23·0	101	108	+ 7	181	157	− 24	1480	1780	+ 300	127	34·0

TABLE 3 (Exp. 6). Dog. 4·25 kilos. Heart 58 grms.

Arterial B. P. (mm. Hg)	Pressure V. C. (mm. H_2O)			Output (c.c. per min.)			Pulse rate per minute	Temp. °C.
	Pericard. shut	Pericard. opened	Difference	Pericard. shut	Pericard. opened	Difference		
65	31	7	− 24	555	565	+ 10	144	36
70	33	7	− 24	556	575	+ 19	147	36
89	25	6	− 19	555	589	+ 34	143	36
105	32	8	− 24	580	610	+ 30	162	37
122	46	8	− 38	550	562	+ 12	161	37
141	64	14	− 50	550	562	+ 12	168	37
146	87	19	− 68	535	577	+ 42	147	36
159	167	58	− 109	410	545	+ 135	150	36
186	254	125	− 129	300	545	+ 245	150	36
195	327	231	− 96	182	273	+ 91	150	36

TABLE 4 (Exp. 22). Dog. 5 kilos. Heart 52 grms.

Venous supply	Pressure V. C. (mm. H_2O)			Pulm. art. pres. (mm. H_2O)			Arterial B. P. (mm. Hg)			Output (c.c. per min.)			Pulse rate per minute	Temp. °C.
	Peric. shut	Peric. opened	Diff.	Peric. shut	Peric. opened	Diff.	Peric. shut	Peric. opened	Diff.	Peric. shut	Peric. opened	Diff.		
Small	73	33	− 40	158	144	− 14	81	81	—	254	260	+ 6	156	36·5
+	164	55	− 109	262	250	− 12	92	95	+ 3	580	665	+ 85	168	37
+	232	84	− 148	326	340	+ 20	94	106	+ 12	830	1120	+ 290	156	36·5
+	348	250	− 98	430	494	+ 64	99	108	+ 9	962	1600	+ 638	159	36·5
+ (full)	392	324	− 68	460	440	− 20	95	103	+ 8	710	1600	+ 890	156	36·5

TABLE 5 (Exp. 23). Dog. 5·5 kilos. Heart 50 grms.

Venous supply	Pressure V. C. (mm. H_2O)	Pulm. arter. pres. (mm. H_2O)	Arterial B. P. (mm. Hg)	Output per minute (c.c.)	Pulse rate per minute	Temp. °C.
Pericardium intact						
Small	35	315	58	307	132	37
+	55	367	68	413	335	37
+	69	405	76	533	135	37
+	147	469	93	1020	134	37
+	215	507	97	1380	141	37
+ (full)	241	521	99	1530	135	37
Pericardium opened						
Moderate	55	341	68	418	135	37
+	111	475	85	920	141	37
+	123	479	94	1360	150	37·5
+	121 – 169	459 – 443	96	1520	150	37·5
+	199 – 201	447 – 415	96	1780	153	37·5
+ (full)	279	403	95	1560	156	37·5

TABLE 6 (Exp. 24). Dog. 5 kilos. Heart 48 grms. Pulmonary insufficiency.

Pericardium intact						
Moderate	121	260	71	450	93	31·5
+	291	398	86	715	93	31·5
+	405	474	86	750	96	31·5
+ (full)	415	474	86	750	96	31·5
Pericardium opened						
Moderate	63	170	71	475	90	31·5
+	225	285	90	850	90	31·5
+	305	302	94	728	84	31·0
+ (full)	315	266	82	800	81	31·0

TABLE 7 (Exp. 29). Dog. 3·5 kilos. Heart 36 grms.

Height of venous reserv. (cms.)	Pressure V. C. (mm. H_2O)			Arterial B. P. (mm. Hg)			Output per minute (c.c.)			Output per beat (c.c.)			Pulse rate per minute	Temp. °C.
	Peric. shut	Peric. opened	Diff.	Peric. shut	Peric. opened	Diff.	Peric. shut	Peric. opened	Diff.	Peric. shut	Peric. opened	Diff.		
6	16	9	− 7	83	85	+ 2	540	675	+ 135	4·0	5·0	+ 1·0	135	35
8	19	15	− 4	86	87	+ 1	645	705	+ 60	4·9	5·3	+ 0·4	132	35
10	27	14	− 13	86	88	+ 2	680	780	+ 100	5·0	5·8	+ 0·8	135	35
11	30	15	− 15	89	91	+ 2	820	930	+ 110	6·2	7·0	+ 0·8	132	35
13	47	23	− 24	91	95	+ 4	955	1160	+ 205	7·1	8·6	+ 1·5	135	35
14	52	22	− 30	88	91	+ 3	1030	1240	+ 210	7·6	9·2	+ 1·6	135	35
17	69	29	− 40	91	99	+ 8	1080	1500	+ 420	8·4	11·6	+ 3·2	129	35
19·5	85	50	− 35	99	105	+ 6	1300	1520	+ 220	10·3	12·1	+ 1·8	126	35
7	29	16	− 13	87	89	+ 2	530	660	+ 130	5·4	6·7	+ 1·3	99	31·5
9·5	51	27	− 24	96	103	+ 7	725	880	+ 155	7·6	9·2	+ 1·6	96	31·5
13	73	55	− 18	89	101	+ 12	700	1360	+ 660	7·8	15·1	+ 7·3	90	30·5
15	91	76	− 15	88	94	+ 6	665	1040	+ 375	8·5	13·4	+ 4·9	78	30
18	111	107	− 4	89	93	+ 3	680	965	+ 285	8·7	12·4	+ 3·9	78	29
23	165	157	− 8	89	91	+ 2	680	1030	+ 350	8·7	13·2	+ 4·5	78	29
26	185	165	− 20	89	102	+ 13	650	985	+ 335	8·0	12·2	+ 4·2	81	29

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