THE MECHANICAL EFFECT OF FLUID IN THE PERICARDIUM ON THE FUNCTION OF THE HEART.

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THE fact that a rise of pressure in the pericardial cavity produces a diminution in aortic pressure is well known. Experiments on this point were carried out by Cohnheim(1), François-Franck(2), Lewis(3) and others and it was found that an intrapericardial pressure which is a little above the venous pressure may hinder the diastole of the auricle and obstruct the venous supply to the heart. Lewis used in his researches a rather small intrapericardial pressure and found that a slight rise of intrapericardial pressure was accompanied by a distinct fall of systemic pressure. In order to throw more light on this relation, and also, if possible, to find some method to recover the heart's action under a high intrapericardial pressure, I have made the experiments given below.

Method. Most of the experiments were carried out on the heart-lung preparation on dogs. A small T-tube of metal was made for the special purpose of introducing the fluid into the pericardial cavity. This tube has a length of 3·5 cm. and a bore of 2 mm. One end of this tube is furnished with a small thin oval flange with a diameter of about 8 mm. A disc which is of just the same size as the flange moves round the tube and may be pressed against the flange by means of a screw. A small hole was made on the front aspect of the pericardium. Through this hole the flange was introduced, the disc was then pressed against the flange so that the pericardium was fixed water-tight between these two discs. One of the ends of the tube was connected to a mercury manometer and the other end to a glass T-tube by which this end is connected with an injection syringe of 10 c.c. and a vessel filled with Ringer's solution at 37° C. Ringer's solution was first sucked up into the injection syringe, then allowed to flow into the pericardium under pressure. Instead of the syringe

I sometimes used a vessel filled with Ringer's solution hanging from the ceiling. The head of water could be changed by pulling the vessel up or down. The pressure in the pericardial cavity, in the aorta, and in the vena cava were recorded simultaneously. The output was measured in the ordinary way.

1. *The effect of fluid in the pericardium on the output, the arterial pressure, and the venous pressure.*

In the pericardium there is normally a certain amount of fluid. The intrapericardial pressure is in the heart-lung preparation almost zero when the venous supply to the heart is moderate. It becomes however distinctly positive when the venous supply is large. In my experiments I started usually with pressure at zero. It was sometimes necessary to suck away a small amount of fluid (1–2 c.c.) in order to bring the pressure to zero. Any inflow of fluid, even so small an amount as 3–5 c.c., causes more or less increase of intrapericardial pressure, and at the same time an increase of venous pressure, and a decrease of the output of the left ventricle. With small amounts, the arterial pressure changes hardly at all or only slightly, but a remarkable decrease appears when the fluid allowed to flow into the

TABLE I. Weight of heart 42 gms. Pulse rate 130–142 per minute.

Amount of fluid in pericardium (c.c)	Intra-pericardial pressure (mm. Hg)	Output per minute (c.c.)	Arterial pressure (mm. Hg)
I. Height of venous reservoir 9 cms.			
0	0	507	74
5	1·0	422	74
10	2·4	320	73
15	2·8	253	71
20	3·6	163	67
25	4·1	65	60
30	6·1	12·8	33
35	6·6	0	21
II. Height of venous reservoir 24 cms.			
0	0	867	75
2	7·2	843	75
5	11·1	800	73
15	11·6	592	69
25	13·0	395	65
35	15·6	136	62
40	17·5	41	50
45	18·2	0	27

pericardial cavity reaches a rather large amount. With continued increase of fluid in the pericardium all these effects become more conspicuous, and at last the heart ceases to expel any blood at all.

A. *Effect on the output.* The rate of decrease of output by introducing fluid into the pericardium is very regular. Table I and Fig. 1 show one example of such experiments.

As I showed in my previous experiments(4), the existence of the pericardium cannot be without influence on the function of the heart

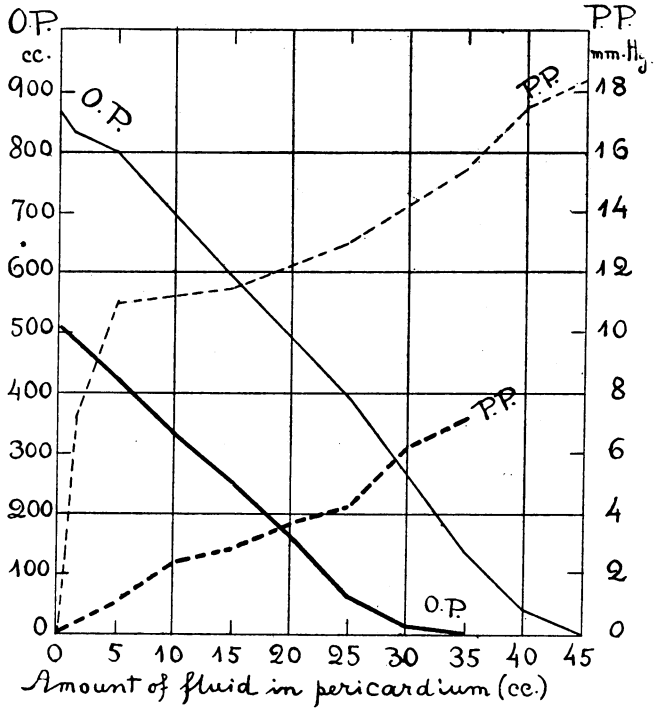

Fig. 1. (Table I) Thick lines: height of venous reservoir 9 cm. Thin lines: 24 cm. P.P.: Intrapericardial pressure. O.P.: output of the left ventricle.

although the venous supply to the heart be moderate so that the heart works with a rather small volume. Thus it is no wonder that a small amount of fluid in the pericardium affects the output. This decrease of output by inflow of fluid may be due to the increased resistance to diastole of the heart.

B. *Effect on the arterial pressure.* It is worthy of note that the arterial pressure falls only slightly by introducing fluid into the pericardium until the fluid reaches a certain amount. Any further addition

beyond this amount, however, causes a remarkable fall in arterial pressure. Table II and Fig. 2 *a* and *b* which were obtained from a heart of 50 gms. show this relation. In Fig. 2*a* (height of venous reservoir 13·5 cm., output 810 c.c. per minute), the arterial pressure fell only from 87 to 73 mm. Hg by an increase in the amount of fluid in the pericardium from 0 to 40 c.c. By a further addition of 5 c.c. of fluid the arterial pressure fell suddenly from 73 to 53 mm. Hg, and by another addition of 5 c.c. of fluid to 36·2 mm. and at last to 27·5 mm. through another 5 c.c. of fluid. The heart now ceased altogether to expel blood. This was also the case in Fig. 2*b* (height of venous reservoir 27 cm.). The increase of fluid in the pericardium from 0 to 50 c.c. caused a fall of arterial pressure from 95·2 to 76·7 mm. Hg. By further additions of 5 c.c. of fluid at a time the arterial pressure changed remarkably, namely to 67·5, 44, and 27 mm. Hg. The arterial resistance was placed at a height of about 45 cm. above the heart so that a column of blood of just the same height was always pressing on the aorta. The recorded arterial pressure therefore remained at a height of about 27 mm. Hg when the heart ceased its action.

TABLE II. Weight of heart 50 gms. Arterial resistance 45 mm. Hg.

Amount of fluid in pericardium (c.c.)	Intra-pericardial pressure (mm. Hg)	Arterial pressure (mm. Hg)	Venous pressure (mm. H_2O)
I. Height of venous reservoir 13·5 cms.			
0	0	87·0	35
5	1·1	85·3	36
10	1·6	83·4	39
20	2·8	81·5	54
30	4·1	80·0	56
40	6·5	73·0	89
45	7·3	53·0	99
50	8·5	36·2	115
55	9·8	27·5	136
II. Height of venous reservoir 27 cms.			
0	0	95·2	150
5	8·9	96·0	150
10	8·9	94·2	156
20	12·2	89·8	170
30	13·9	85·2	189
35	15·0	80·0	198
40	15·5	77·5	208
50	16·4	76·7	225
55	17·5	67·5	240
60	17·7	44·0	255
65	19·5	27·0	275

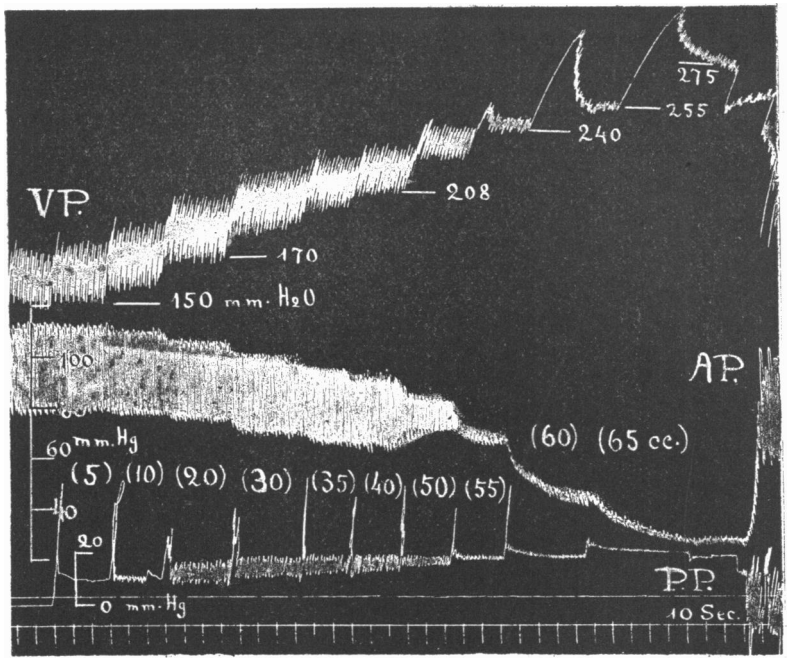

Fig. 2*a*. (Table II) Height of venous reservoir 13·5 cm. V.P.: venous pressure. A.P.: arterial pressure. P.P.: intrapericardial pressure. Figures in brackets indicate the amount of fluid in the pericardium.

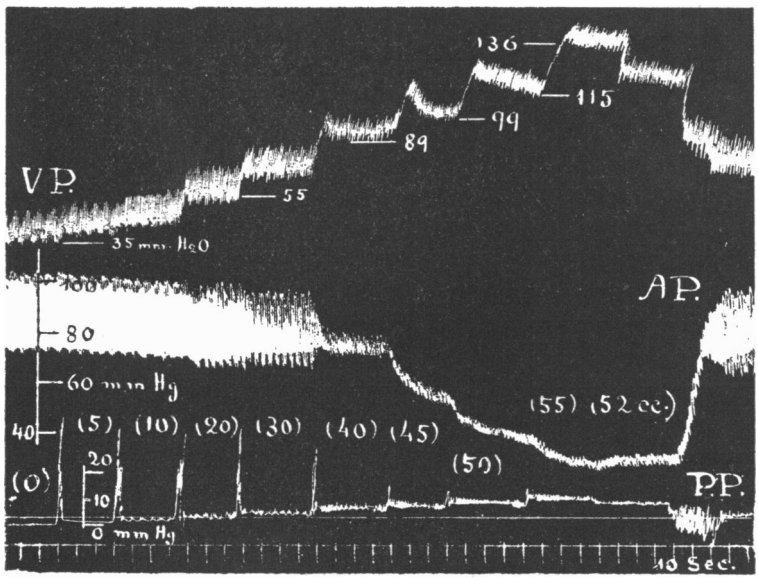

Fig. 2*b*. (Table II) Height of venous reservoir 27 cm.

The amount of fluid beyond which any addition causes a remarkable fall of arterial pressure, and also the amount by which the heart ceases to expel any more blood, are synoptically shown in Table III.

TABLE III.

Weight of heart (gms.)	Height of venous reservoir (cms.)	Amount of fluid in pericardium (c.c.) causing standstill of blood circulation	Amount of fluid in pericardium (c.c.) effecting slightly blood pressure
42	9	35	25
42	24	45	35
50	13·5	55	40
50	27	65	50
39	13	30	22
39	27	35	25
43	6	35	25
63	17	85	65
37	5	40	28
37	15	60	45

Using the numbers in this table, we may conclude that the fall of arterial pressure is rather small until the fluid in the pericardium reaches about 70 % of the amount by which the heart ceases to expel any more blood. Any further increase of fluid exerts a very remarkable influence on the arterial pressure which may be dangerous during normal life.

The relation between the amount of the output and the height of the arterial pressure depends on the distensibility of the vessels although all other important conditions should be eliminated. It is thus of course incorrect to estimate changes in arterial pressure from the results of experiments in the heart-lung preparation in which the blood flows through arterial rubber tubes. I therefore carried out some experiments on animals with open thorax. Fig. 3 shows one example. This figure was obtained from a dog of 4·8 kilos (weight of heart 31 gm.). On account of the thorax being open the arterial pressure was rather low. The rate of decrement of arterial pressure is shown in Table IV. The relations are on the whole the same as those in the heart-lung preparation although the effects of a moderate amount of fluid are more distinct in the normal animal than in the heart-lung preparation.

In the researches in which he only used low air pressure for increasing the intrapericardial pressure, Lewis determined that on an average a rise of the pressure of 1 mm. Hg was accompanied by a fall of systemic pressure of 8 mm. Hg. He also noticed that this ratio often tends to

increase slightly when relatively low pressures were employed. As is obvious from Tables, I, II and IV, the ratio of fall of arterial pressure to increase of intrapericardial pressure is, in my experiments, certainly

TABLE IV.

Amount of fluid in pericardium (c.c.)	Intra-pericardial pressure (mm. Hg)	Arterial blood-pressure (mm. Hg)
0	0	60·4
5	1·2	57·0
10	2·0	55·5
15	2·8	48·0
20	3·7	36·5
25	5·7	20·0
30	7·9	11·8

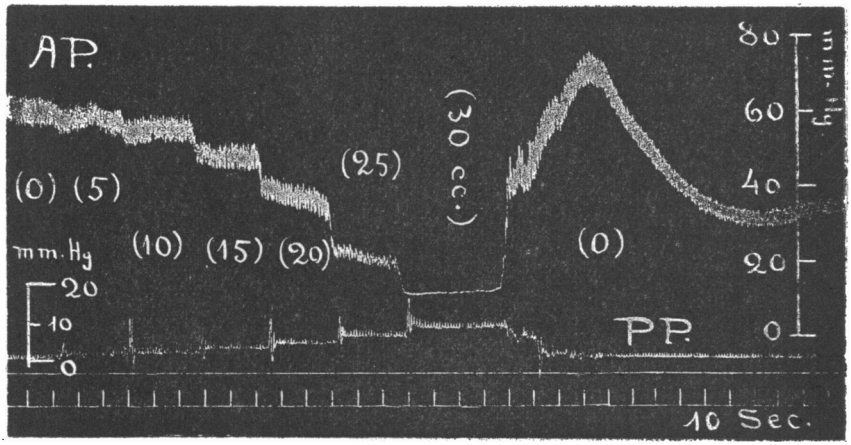

Fig. 3. (Table IV) Experiment on the intact animal with open thorax. The figures in brackets indicate the amount of fluid in the pericardium.

not so large when relatively low pressures were employed, namely a rise of intrapericardial pressure of 1 mm. Hg was accompanied by a fall of arterial pressure of 1·5–4 mm. Hg. This ratio becomes distinctly larger when high pressures are used, namely by a rise of intrapericardial pressure of 1 mm. Hg the arterial pressure fell 20–30 mm. Hg.

2. *The amount and the pressure of fluid in the pericardial cavity which may stop the circulation of the blood.*

When the venous supply to the heart is moderate the rise of intrapericardial pressure by addition of fluid into the pericardial cavity is

at first small but later on it becomes large. In case of the venous supply being large, the inflow of even a very small amount of fluid such as 3–4 c.c. causes already a distinct increase in the pressure and further increases are almost the same as with the moderate venous supply. These relations can be found in Fig. 1 and also in Table II.

The height of intrapericardial pressure at which the heart ceases to expel blood varies within very wide limits and corresponds to the height of the venous reservoir. The height of intrapericardial pressure, of venous pressure, and of venous reservoir, were all three almost the same when the blood circulation was brought to a standstill. For

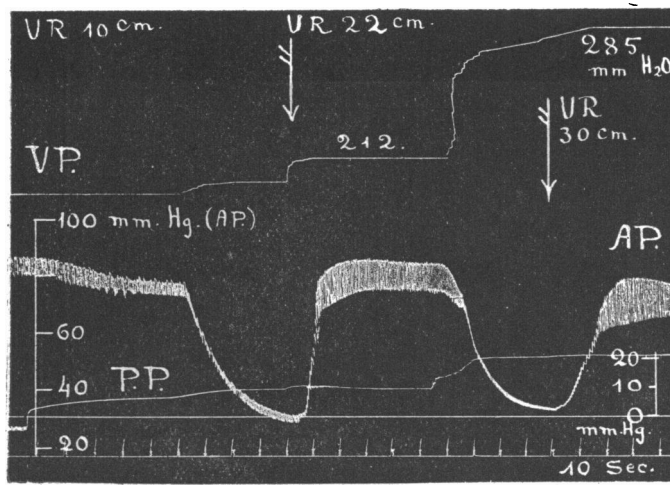

Fig. 4. Influence of increasing venous pressure. V.R.: venous reservoir.

example in Fig. 2*a*, height of venous reservoir 13·5 cm., venous pressure 136 mm. water, and intrapericardial pressure 9·8 mm. Hg.

If the circulation be first brought to a standstill through the introduction of a certain amount of fluid into the pericardium and then the venous reservoir be raised so that the blood will be driven to the heart under increased pressure, the heart begins to act again and the arterial pressure rises to about normal height. Fig. 4 shows these facts. The figure was obtained from a heart of 45 gm. At the beginning of the experiment the venous reservoir was kept at a height of 10 cm. The fluid was run into the pericardium. When the intrapericardial pressure reached 8·5 mm. Hg the arterial pressure fell rapidly. At the first arrow, the venous reservoir being raised to a height of 22 cm., the

arterial pressure began at once to increase to 80 mm. Hg. The intra-pericardial pressure was now raised to 20 mm. Hg by addition of fluid and the arterial pressure fell to 37 mm. Hg. But from this decrement the arterial pressure was again restored by raising the venous reservoir to 30 cm. (at the second arrow).

From the above results it is evident that the cause of the arrest of the circulation is the hindrance to the venous inflow, which the fluid surrounding the heart causes. The amount of fluid in the pericardium when the circulation ceases varies according to the size of heart. Even in the same heart this amount is not fixed but varies according to circumstances and especially depends on the venous supply. If the venous supply to the heart is large the heart receives more blood during its diastole. The volume of the heart therefore increases and the pericardial cavity must be diminished. In spite of these facts a greater amount of fluid may be introduced into the pericardium without stopping the circulation when the venous supply is large than when it is small. In Fig. 1 at the standstill of the circulation, the amount of fluid in the pericardium was, at a height of venous reservoir 9 cm., 35 c.c. and at that of 24 cm., 45 c.c. This relation will be found also in Tables II and III.

3. *Effects of adrenaline.*

According to the results above described, the arterial pressure may remain rather high although a certain amount of fluid exists in the pericardium. If the amount of fluid increases beyond a certain limit its remarkable influence on the arterial pressure occurs. The arterial pressure is then no longer high enough to produce a normal circulation. In order to restore the blood circulation from this condition, apart from removing the fluid, there are two ways, *i.e.* a rise of venous pressure or enlargement of the pericardial cavity. If this cavity be enlarged a little the pressure in it will fall and the arterial pressure may rise appreciably.

It is well known that the adrenalinised heart may act with a very much increased frequency and expel more blood. The volume of the heart is thereby much diminished. It is possible that this diminution in volume may enlarge the pericardial cavity and lower the pressure in it. I have therefore carried out experiments to compare the influence of fluid in the pericardium on the function of the heart before and after application of adrenaline. The following protocol shows the results of one experiment:

Dog, weight of heart 17 gm., height of venous reservoir 24 cm. Output 900 c.c. per minute. By inflow of 22 c.c. of fluid into the pericardium the heart ceased to expel blood. The intrapericardial pressure thereby was 22·5 mm. Hg. 0·1 mg. of adrenaline was added into the venous reservoir. The output now increased to 1040 c.c. per minute. After allowing 22 c.c. of fluid to flow into the pericardium the arterial pressure sank from 112 to 84 mm. Hg. By increasing the amount of fluid to 28 c.c. the circulation was stopped. The intrapericardial pressure thereby was just the same as before, namely 22·5 mm. Hg.

If a certain amount of fluid was introduced into the pericardium, so that the arterial pressure was lowered considerably, and then adrenaline was given to the heart, the heart expelled more blood than before

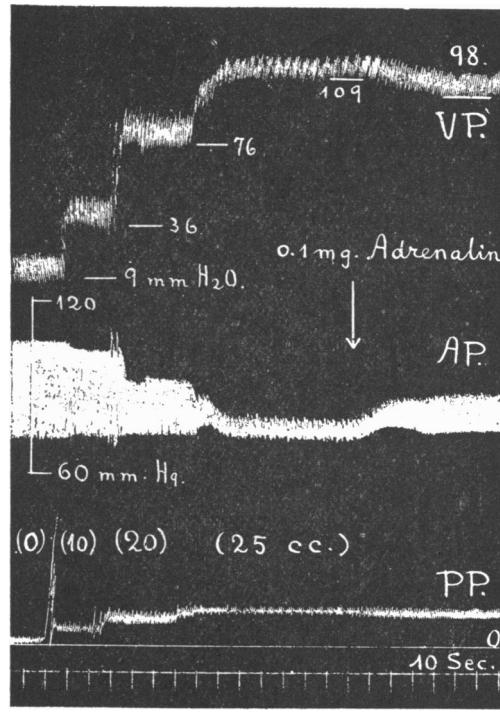

Fig. 5. Influence of adrenaline.

and the blood circulation became more vigorous. Fig. 5 shows one example of such experiments. This figure was obtained from a heart of 26 gm. The venous reservoir was kept at a height of 11 cm. By inflow of 25 c.c. of fluid into the pericardium the arterial pressure fell from 89 to 75·5 mm. Hg, the output was decreased from 810 to 56 c.c. per minute. At the arrow 0·1 mg. of adrenaline was added into the

venous reservoir. The arterial pressure now rose to 82 mm. Hg and the output to 205 c.c. per minute. The venous pressure and the intra-pericardial pressure fell slightly.

4. *Experiments on animals with open thorax.*

The results of the above experiments on the heart-lung preparation show that either the increase of venous supply or the application of adrenaline may restore the blood circulation which was hindered through the fluid present in the pericardial cavity. We shall see now whether these results are applicable to intact animals.

The venous pressure in the heart-lung preparation corresponds to the distending force of the blood on the auriculo-ventricular walls in intact animals. This force is caused by the difference between the pressure in the thorax and that in the other parts in the body, *e.g.* the

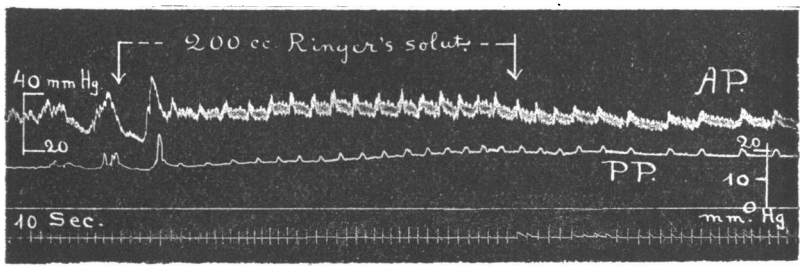

Fig. 6. Influence of infusion.

abdomen where the great veins lie. Although this force may be somewhat changed by several conditions, the chief one is the negative pressure in the thorax, and it is therefore hardly possible to raise this difference between the pressures much beyond the normal limit. It is also difficult to raise the pressure in the veins appreciably by any artificial means. Although a large amount of blood be infused into the vein the pressure in it undergoes no change or rises only temporarily. There is thus less hope of recovering the blood circulation interrupted by fluid surrounding the heart by raising the venous pressure in intact animals. Fig. 6 which was obtained from a dog with open thorax shows one example of infusion. The height of arterial pressure in this animal was 103 mm. Hg. By allowing 45 c.c. of fluid to flow into the pericardium it sank to 32·5 mm. Hg. At the arrow 200 c.c. of Ringer's solution were injected into the femoral vein. The arterial

pressure became regular but did not rise whereas the intrapericardial pressure rose considerably. This means that the volume of the heart increased by the injection of Ringer's solution, but the distending force of the blood flow on the heart did not rise high enough to exceed the pressure surrounding the heart.

Adrenaline, if the dose is not very large, diminishes the volume of the heart in intact animals as in the heart-lung preparation. As a rule, when there is a rise in the arterial pressure there is a fall in the venous pressure. As regards the effect of adrenaline, however, this is not the case. The contraction of the blood vessels evoked by adrenaline is most distinct in the arterial system so that a large amount of blood might escape into the veins. The pressure in the veins does not therefore fall, on the contrary it rises more or less during action of the adrenaline although the heart works extremely energetically. Fig. 7 shows one example. In this experiment the pressures in the carotid, the vena cava superior, and the pericardial cavity were recorded. At the arrow 0·1 mg. of adrenaline was injected into the femoral vein. The venous pressure rose from 8 to 10 mm. water during the increase of arterial pressure. The intrapericardial pressure fell slightly.

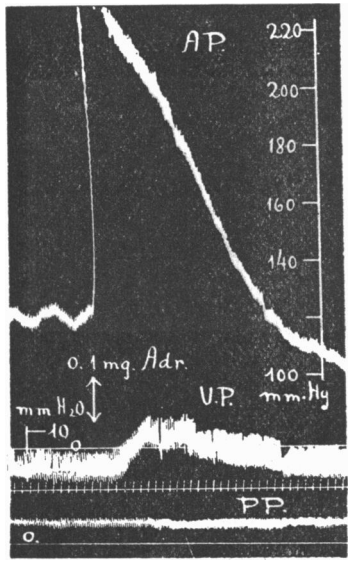

Fig. 7. Influence of adrenaline in the intact animal.

The diminution in the volume of the heart and the increase of venous pressure may both favour the action of a heart compressed by surrounding fluid. We may therefore expect that the injection of adrenaline in the intact animal may recover the blood circulation which was weakened through the pressure of fluid in the pericardium. In order to examine this possibility, I allowed the fluid to flow into the pericardium so far that the circulation was not yet stopped but continued with a much impaired vigour. The arterial pressure sank generally to $\frac{1}{4}$–$\frac{1}{2}$ of the normal height. Adrenaline was then injected into the femoral vein. One example of such experiments is given in Fig. 8. This figure was obtained from the same heart as Fig. 6. The figures above the curves indicate the amount of

fluid present in the pericardial cavity. At the first arrow 0·1 mg. of adrenaline was injected. The arterial pressure rose considerably and the heartbeats became regular. At the second arrow more fluid was run into the pericardium so that the arterial pressure sank again. We observe some difference in the amounts of fluid by which the blood circulation was stopped, before and after application of adrenaline, namely, before adrenaline 45 c.c. and after adrenaline 50 c.c. From this result we may conclude that the injection of adrenaline has a noticeable effect in such cases.

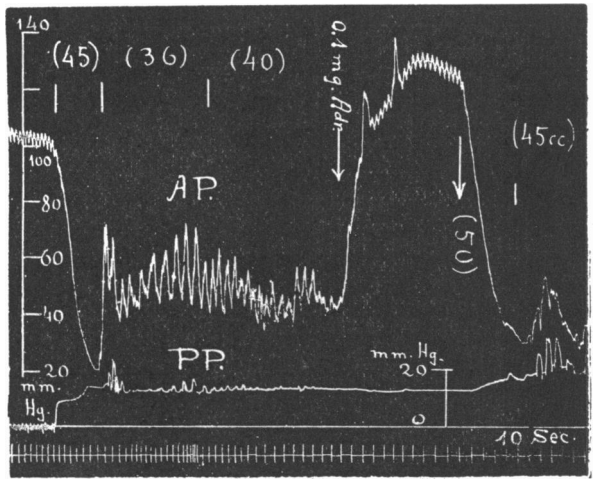

Fig. 8. Influence of adrenaline in the intact animal with fluid in the pericardium. Figures in brackets indicate the amount of fluid in the pericardium.

Summary.

In the heart-lung preparation, and also in animals with open thorax, Ringer's solution was allowed to flow into the pericardial cavity under pressure.

1. A very small amount of fluid in the pericardium causes diminution in the output of the heart. By further addition of fluid the output diminishes regularly.

2. The arterial pressure changes only slightly until the amount of fluid in the pericardium reaches a certain limit. Beyond this limit the addition of a small amount of fluid causes a distinct fall of arterial pressure.

3. The height of intrapericardial pressure which brings the circulation of blood to a standstill is just the same as that of venous pressure. If the venous pressure be raised during the standstill of the circulation, the blood-flow is restored.

4. In the intact animal, it is very difficult to raise the venous pressure by injection into the veins. An infusion of a large amount of blood causes no rise of venous pressure nor dilatation of the heart, or only a brief one. Such an infusion is therefore of no advantage to the circulation when this is hindered by pressure of fluid surrounding the heart.

5. Adrenaline diminishes the volume of the heart; the intrapericardial pressure may therefore be decreased. In the intact animal, it causes also a slight rise of venous pressure. It therefore affects very favourably the heart when compressed by fluid surrounding it.

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