THE PULSE PRESSURE AS AN INDEX OF THE SYSTOLIC OUTPUT.[1]

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PLATES XXIX AND XXX.

In a communication made some time ago, one of us (Dawson)[2] called attention to the propriety of considering the pulse pressure (that is the difference between the systolic and diastolic pressure) as an index of the systolic output. In this connection it was stated that ". . . experiments on animals, although not yet completed, have been carried far enough to show" that "the pulse pressure varies with the systolic output. In this series of experiments the pulse pressure as determined with the Hürthle manometer was compared with the systolic output as determined with the cardiac plethysmograph of Henderson, and it was found that on stimulation of the peripheral end of the vagus, in asphyxia, hemorrhage, infusion and so forth the pulse pressure and the systolic output vary together." These experiments have now been completed and the results are presented in the present communication.

METHOD.

Anesthesia.—Each dog received from 0.6 to 1.2 grm. of morphia hypodermically, and an hour later was anesthetized with ether, administered at first with a cone, but later through a tracheal cannula.

Recording.—Simultaneous tracings were obtained as follows:

1. The mean blood pressure in the femoral artery by means of a mercury manometer.
2. The systolic output by means of Henderson's cardiac plethysmograph.

[1] Received for publication March 20, 1908.

[2] *British Med. Jour.*, 1900, ii, 996.

3. The blood pressures in the carotid and femoral arteries by means of two[3] Hürthle manometers.

The recording surface was a Hürthle kymographion and the general character of the tracings obtained is shown in Fig. 3 (Plate XXIX) and Fig. 4 (Plate XXX).

RESULTS.

The results of the experiments are so concordant that it has been deemed admissible to present only the protocols of two typical

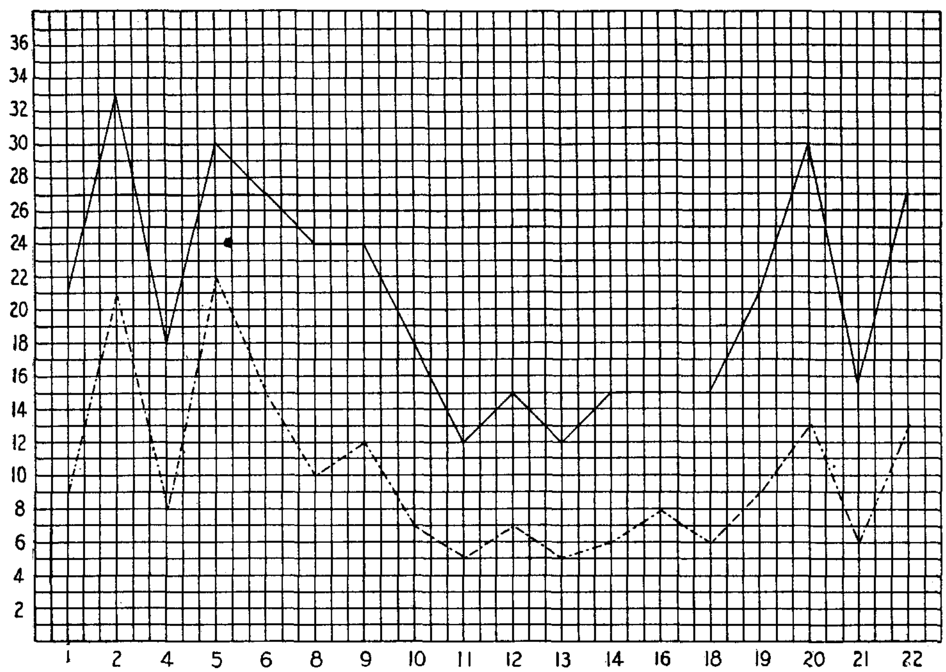

FIG. 1.

experiments. These are given in the form of two tables, two charts and two tracings.

[3] By using two arteries and two manometers we doubled the chance of obtaining an accurate record of the pulse pressure. In Fig. 3 (Plate XXIX) and Fig. 4 (Plate XXX), for example, the record obtained from the carotid was not to be relied upon, for after the completion of the experiment it was found on careful examination of the record, that with high pressures the lever pressed too tightly against the paper, while with lower pressures the lower part of the curve was spoiled by an obstruction to the movement of the lever.

Tables.—The numbers in Column I correspond to those which appear running horizontally at the bottoms of Figs. 1 and 2. The figures in Column II have only a relative value. The figures in Column III denote in cubic centimeters the change in the volume of both ventricles. Since most persons conceive of changes occur-

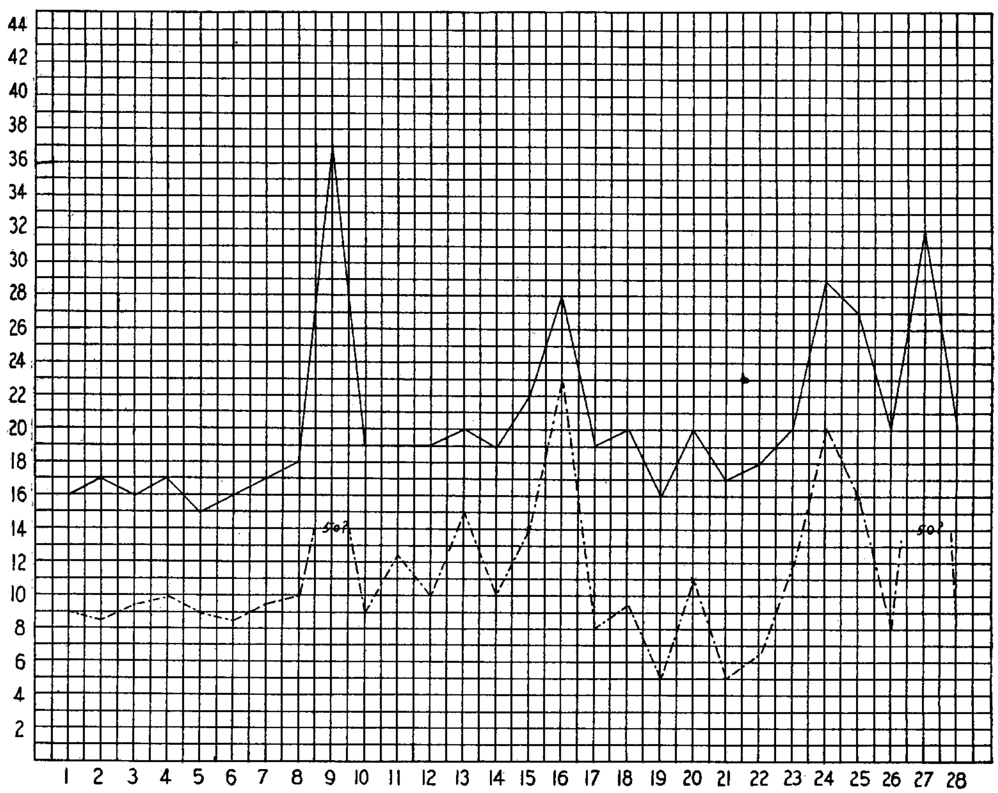

Fig. 2.

ring in the circulation in terms of variations in the mean blood pressure, Column IV has been added to complete the picture. The results given in the tables are also shown in Figs. 1 and 2, which correspond to Tables I and II respectively.

Other explanatory data are the following: asphyxia was produced by stopping the artificial respiration; stimulations were made with the faradic current; the stimulations of the annulus Vieussentis occurred in the course of an ineffectual attempt to reach the accelerators. The carbonate solution had the composition: Sodium carbonate, 7.1 per cent. and sodium bicarbonate, 4.6 per cent.

TABLE I.

Experiment I.

I. Numbers Correspond to Those in the Figure.	II. Pulse Pressure (Length of Stroke of Hürtle Lever) from Carotid.	III. Systolic Output (Change in Vol. of Heart in c.c.).	IV. Mean Blood Pressure (Femoral) in mm. Hg.	
1	7	9	158	After section of both vagi.
2	11	21	60	During stimulation of peripheral end of vagus.
4	6	8	110	After recovery.
5	10	22	54	During stimulation of peripheral end of vagus.
6	9	15	66	During escape from vagus inhibition.
8	8	10	98	Normal.
9	8	12	148	During asphyxia.
10	6	7	100	During stimulation of the central end of the vagus.
11	4	5	54	The same.
12	5	7	78	The same.
13	4	5	136	During stimulation of the annulus Vieussentis.
14	5	6	84	After recovery.
16	5	8	80	After intra-arterial infusion of strong sodium carbonate and bicarbonate.
18	5	6	64	During apnea following the infusion.
19	7	9	90	After a second infusion.
20	10	13	78	After a third infusion.
21	5	6	32	After bleeding.
22	9	13	30	After a fourth infusion.

In this experiment (Experiment I), the record obtained by the Hürthle manometer connected with the carotid artery was the more satisfactory and consequently the values relating to this artery only have been presented. Strong carbonate (see No. 18) usually causes very violent respirations followed by a respiratory pause which usually continues until death unless the animal is kept alive for some time with artificial respiration so that the effect of the carbonate has had time to disappear.

On perusal of the tables, it is seen that the pulse pressure (Column II) and the systolic output (Column III) vary together. This is shown even more clearly in the figures, where the lines which represent the quantitative variations in these two phenomena are seen to be almost exactly parallel.

TABLE II.

Experiment II.

I. Numbers Correspond to Those in the Figure.	II. Pulse Pressure (Length of Stroke of Hürthle Lever) from Femoral.	III. Systolic Output (Change in Vol. of Heart in c.c.).	IV. Mean Blood Pressure (Femoral) in mm. Hg.	
1	16	9	97	Normal.
2	17	8	148	After section of vagus.
3	16	9	139	
4	17	10	228	Stimulation of central end of vagus.
5	15	9	126	
6	16	8	130	
7	17	9	228	Stimulation of central end of vagus.
8	18	10	144	
9	36	50?	52	Stimulation of peripheral end of vagus.
10	19	9	134	
11	19	13	164	Stimulation of the anterior crural.
12	19	10	140	
13	20	17	166	During stimulation of annulus Vieussentis.
14	19	10	130	
15	22	14	220+	Asphyxia.
16	28	28	190+	Asphyxia.
17	18	8	148	
18	19	9	168	
19	16	5	100	Bleeding.
20	20	11	182	Transfusion (intravenous) of 0.7 % sodium chloride solution.
21	17	5	124	Bleeding.
22	18	6	84	
23	21	12	64	Intra-arterial infusion of strong sodium carbonate and bicarbonate.
24	29	21	82	Same.
25	27	16	106	Same.
26	20	8	58	
27	32	50?	54	Intra-arterial infusion of strong sodium carbonate and bicarbonate.
28	20	9	58	

In this experiment (Experiment II), the record obtained by the Hürthle manometer connected with the femoral artery was the more satisfactory and consequently the values relating to this artery only have been given. The very great excursions of the plethysmographic lever (see Nos. 9, 16 and 27) was probably due to the inertia of this part of the apparatus. The curve of the mercury manometer (Nos. 15 and 16) ran into the plethysmographic curve so that the height of the mean blood pressure could not be measured.

CONCLUSIONS.

On the basis of these facts we feel justified in making the following assertion:

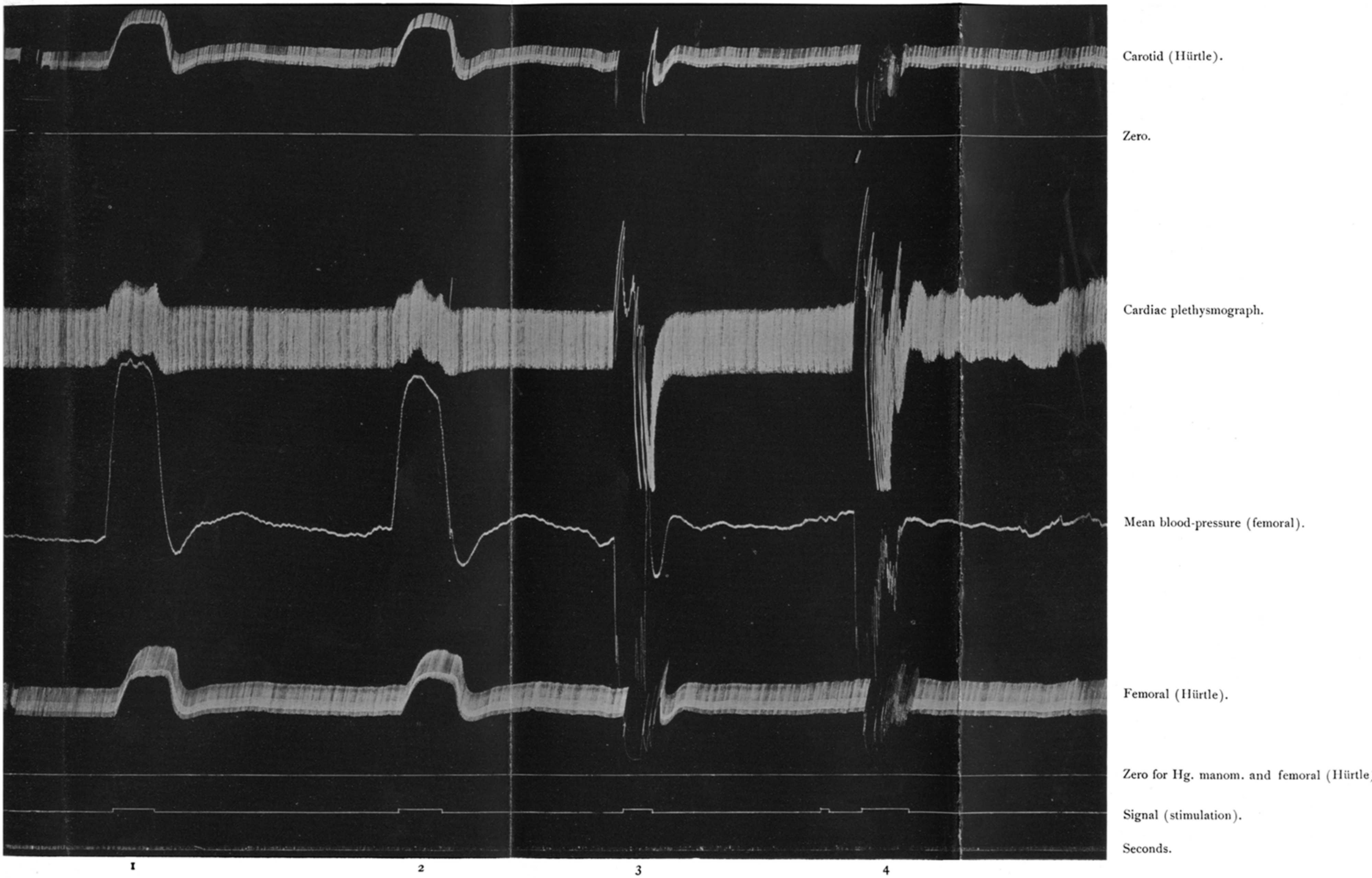
Carotid (Hürtle).
Zero.
Cardiac plethysmograph.
Mean blood-pressure (femoral).
Femoral (Hürtle).
Zero for Hg. manom. and femoral (Hürtle).
Signal (stimulation).
Seconds.
1
2
3
4

FIG. 3.

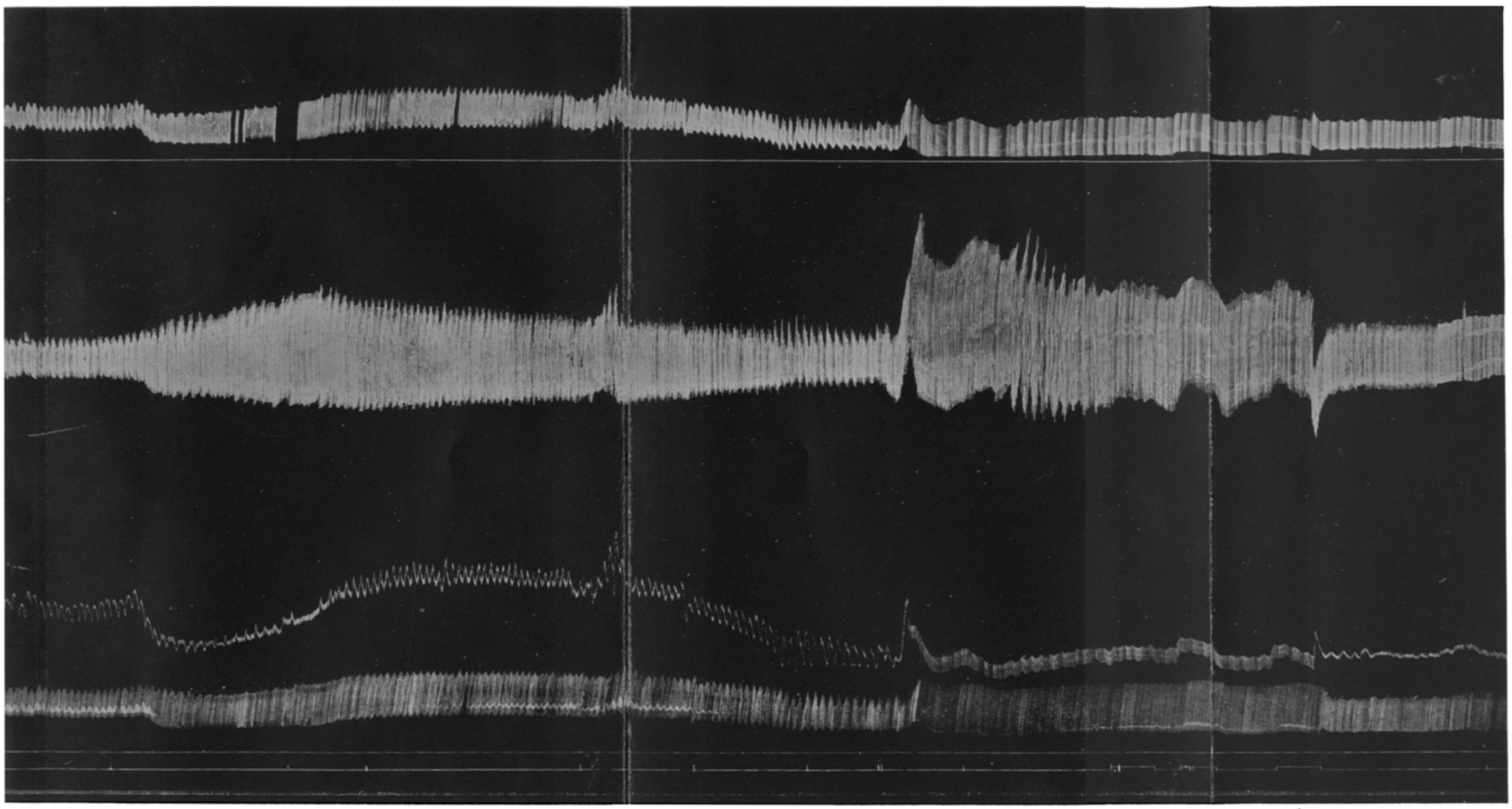
Carotid (Hürtle).
Zero.
Cardiac plethysmograph.
Mean blood-pressure (femoral).
Femoral (Hürtle).
Zero for Hg. manom. and femoral (Hürtle).
1
2
3
4
5
6

Fig. 4.

Under normal conditions and during various procedures (namely, stimulation of the vagus centrally and peripherally, of the saphenus nerve centrally, and of the annulus Vieussentis, intravenous transfusion of 0.7 per cent. sodium chloride solution, intra-arterial transfusion of strong carbonate, bleeding and asphyxia) the pulse pressure is a reliable index of the systolic output.

Explanation of Figures.

Figs. 1 and 2 are diagrammatic presentations of Tables I and II respectively. The solid line represents pulse pressure; the broken line, systolic output. Fig. 3 (Plate XXIX) and Fig. 4 (Plate XXX), are parts of the tracing obtained in Experiment II.

Plate XXIX.

1 and 2. Stimulation of central end of vagus.
3 and 4. Stimulation of peripheral end of vagus.

Plate XXX.

1 and 3. Intra-arterial transfusion of strong carbonate.
2. Bleeding.
4, 5 and 6. Stimulation of central end of vagus.