

By virtue of the present method, which allows for the taking of records over prolonged periods, the writer has recently been able to investigate the form of response obtained during various stages in the process of dark adaptation. Comparative responses of cone and rod receptors have also been studied. It is possible that measurements of the retinal action potential may turn out to be of clinical significance in distinguishing between defects of the retina and those of higher centers.

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Cardiac Output in Coronary Occlusion Studied by the Wezler-Boeger Physical Method.*

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The gas analytic methods of determining cardiac output, developed particularly by Grollman, cannot be applied in patients with acute conditions, for example, acute coronary occlusion. We have, therefore, used the physical method of Wezler-Boeger,¹ based upon the equation of the distensibility of the aorta estimated from the pulse wave velocity. The output is calculated from the pulse pressure, pulse wave velocity and aortic diameter. The functional length of the elastic reservoir of the aorta is determined from the natural frequency of the femoral pulse.

$$\text{Stroke Volume (V)} = 2Q \frac{\Delta P \times \frac{a \times T}{4}}{s \times a^2} = \frac{Q}{2} \cdot \frac{\Delta P \times T}{s \times a} \text{ cc.}$$

Q = Diameter of the aorta, in the outlined cases obtained from Suter's¹ anatomical tables.

ΔP = Pulse pressure in Dyn/cm².

T = Natural frequency of the aorta, obtained from femoral pulse tracings.

s = Specific gravity of blood = 1.06.

a = Pulse wave velocity of the aorta, obtained from simultaneous carotid and femoral pulse tracings.

Comparative studies of the Grollman acetylene and the physical methods have been carried out in 70 determinations in 8 normal subjects.² Both methods checked within 10%, the absolute values running parallel.

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¹ Wezler, K., and Boeger, A., *Erg. d. Physiol.*, 1939, **41**.

² To be published.

The cardiac output was determined by the Wezler-Boeger method in 5 patients with acute coronary occlusion during the acute and subacute phases of the attack.

Case One. Hypertension for 7 years. In this case the cardiac output was lowest on the second day of the attack (Fig. 1) at

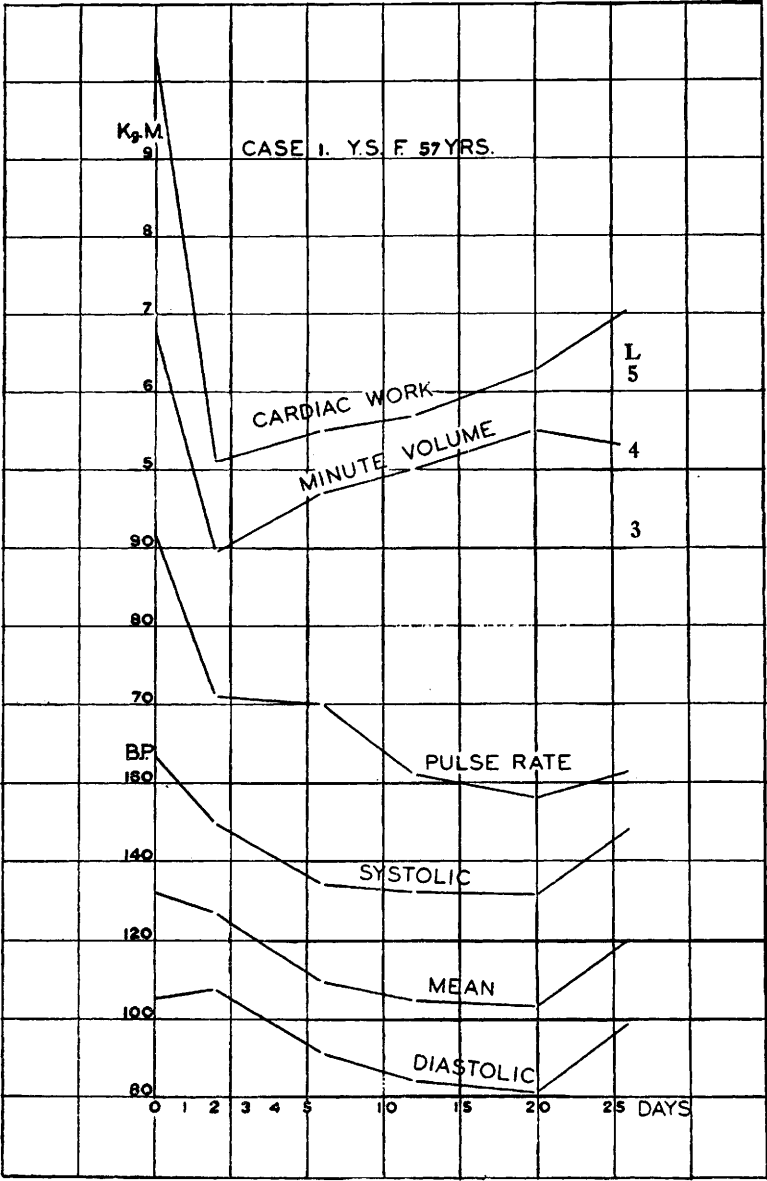


FIG. 1.

which time the systolic, but not the diastolic pressure had fallen, resulting in a diminished pulse pressure. During the following 2 weeks the cardiac output gradually returned to normal. It is im-

TABLE I.

Date	B.P.	P.P*.	Pm	Rate	a in m/sec	S + D	T	V	MVL	Cardiac work KgM/min	
Y.S. f.57. Case I.											
2/8/41—6 hr after onset	168/106	62	132	91.6	9.6	.655	.305	63.9	5.9	5.75	10.32
	166/105	61		91.6	9.6	.635	.295	60.9	5.6		
10	151/111	40	127.0	71.7	8.73	.835	.29	42.3	3.0	2.95	5.1
	148/109	39		70.2	8.73	.855	.285	41.4	2.9		
14	136/92	44	109.5	69.4	7.39	.865	.28	54	3.75	3.7	5.5
	132/90	42		70.3	7.39	.85	.285	52.5	3.7		
20	132/85	47	104.6	60.6	7.39	.995	.31	63.9	3.9	4	5.69
	132/83	49		61.2	7.39	.98	.31	66.7	4.1		
3/4	148/99	49	120	61.2	7.62	.98	.335	70.1	4.3	4.3	7.02
B.G. m. 47. Case II.											
4/14 7P.M. 5 hr after onset	200/124	76	156.7	78.5	17.22	.765	.36	46.75	3.7	3.7	7.88
4/15	136/85	51	106.9	69	8.45	.87	.235	41.6	2.9	2.9	4.215
18	132/97	35	111.7	60.3	8.02	.995	.3	38.3	2.3	2.3	3.495
	132/95	37		62.8	7.75	.955	.265	37.1	2.3		
5/5	136/91	45	111.8	66.8	8.16	.9	.33	53.3	3.6	3.55	5.4
	141/94	47		69.8	8.16	.86	.295	49.7	3.47		
21	168/108	60	131	65.3	9.3	.92	.28	52.9	3.5	3.6	6.42
	163/102	61		68.2	9.49	.88	.285	53.55	3.7		
R.W. f. 59. Case III.											
2/8 8 hr after onset	182/121	61	146.7	76.5	11.71	.785	.305	52.4	4	4	7.98
	181/119	63		73.6	11.71	.815	.305	54.2	4		
2/10	148/102	46	123.8	100.15	9.76	.598	.185	26.7	2.7	2.8	4.73
	152/106	46		100.14	10.0	.598	.185	28.2	2.9		
11	124/101	23	110.9	103.3	9.54	.58	.295	17.1	1.8	1.8	2.73
14	116/96	20	107.6	112.1	9.12	.535	.22	15.9	1.8	1.85	2.71
	122/102	20		111.2	8.73	.54	.22	16.6	1.9		
I.M. m. 53. Case IV.											
12/5/40 12 hr after onset	118/88	30	100.5	49.6	7.67	1.21	.53	62.6	3.1	3.15	4.225
	119/86	30		48.3	7.67	1.24	.51	66.1	3.2		
14	128/89	39	105.8	44.8	8.36	1.34	.61	86	3.8	3.8	5.48
	129/91	38	107.3	45.1	8.36	1.33	.62	85.1	3.8		
20	128/88	40	105.2	44.1	8.36	1.36	.625	90.4	4	4	5.17
	130/91	39	107.8	45.4	8.36	1.32	.620	87.5	4		
27	129/87	42	105.1	43.1	8.36	1.38	.61	92.5	4	4	5.83
	128/86	42	104.1	44.75	8.36	1.34	.615	93.3	4.17		
H.W. m. 35. Case V.											
9/17/40 10 hr after onset	135/92	43	110.45	80.5	7.08	.745	.29	43.2	3.5	3.5	5.26
18	112/78	34	92.6	66.7	6.54	.9	.32	40.85	2.75	2.68	3.26
	104/72	32	85.7	68.2	6.54	.88	.315	37.5	2.6		
25	105/70	35	85	64.8	6.54	.925	.315	41.3	2.7	2.7	3.22
	108/72	36	87.9	63.5	6.54	.945	.325	43.2	2.75		
10/24	109/69	40	86.2	54.3	7.08	1.105	.345	47.9	2.6	2.6	2.97
	106/65	41	82.6	52.4	7.08	1.145	.35	49.7	2.6		
	107/66	41	83.6	52.2	6.85	1.15	.32	49.3	2.6		

*P.P.—pulse pressure; Pm—mean pressure; S + D—duration of heartbeat; M.V.L.—minute volume in liters.

portant to note that the cardiac output and blood pressure did not run parallel. Thus the former returned to normal while the blood pressure remained low and did not change later when the blood pressure rose, although the cardiac work was increased.

Case Two. The course in another hypertensive patient was similar to that just described. (Actual number of determinations was 10.)

Case Three. Hypertension for 20 years. The patient sustained a cerebral hemorrhage on the sixth day, after which our observation was discontinued. The patient died of pneumonia on the fifteenth day.

Case Four. Hypertension for 5 years. Coronary occlusion with heart failure. During the stay of 4 weeks in the hospital the cardiac output gradually rose from 3 to 4 liters.

Case Five. Coronary occlusion with complete heart block just before admission. On the fifteenth day the patient developed renal colic with severe shock, which caused a marked drop in cardiac output from 2.7 to 1.7 liters. After several hours the shock disappeared, and the cardiac output returned to the previous level. (Actual number of determinations was 8.)

Summary. In 4 of 5 cases of coronary occlusion the cardiac output fell rapidly, reaching its lowest level between the second and third day. The output began to rise soon after the third day and maintained a relatively constant level after the tenth day. In the 2 hypertensive patients (Cases 1 and 2) the cardiac output rose in spite of the fact that the blood pressure remained low.

Conclusion. The physical method of Wezler and Boeger of determining cardiac output was applied in 5 cases of acute coronary occlusion. The changes in the circulatory dynamics have been analyzed.