350, and 300 cc and the pH 3.9. This patient also has been relieved of his epigastric distress and has experienced no difficulty in eating.

The data obtained from these 2 patients indicates that the excessive night secretion of gastric juice in ulcer patients is probably neurogenic in origin and may be markedly reduced by supra-diaphragmatic section of the vagus nerves. Although both of these patients appear to have been benefitted by the operation, it is obvious that the period of observation is too short and the number of patients too small to warrant recommendation of this procedure at this time for the treatment of duodenal ulcer.

14228 P

Evaluation of the Ratio of Aortic Rigidity to Peripheral Resistance.

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The expressiveness of pulse rate and blood pressure measurements may be enhanced by introducing the values obtained into the formula described below. This formula helps to differentiate between the effects which conditions in different parts of the cardiovascular system produce upon the blood pressure by offering a number which expresses the ratio of aortic rigidity (or capacity) to the peripheral resistance.

Various investigators (most recently Apéria[1]) in the field of vascular dynamics (comprehensively reviewed by Wezler and Böger[2]) have evaluated the E and W functions, and the E/W ratio. E is the volume-elasticity coefficient and is a measure of the rigidity (or capacity) of the aorta and other large arteries (*windkessel*) and W is a measure of the peripheral resistance. (These symbols are adopted from Wezler and Böger). The evaluations of this ratio published hitherto have been based on data obtained by technics which are not readily available in most laboratories and clinics, as for instance measurements of cardiac output. It is the purpose of this note to point out that the E/W ratio is independent of the stroke volume of the heart, and that this ratio may be determined from heart rate and blood pressure measurements alone.

Development of the Formula. E (volume-elasticity coefficient) is defined as $\frac{\Delta P}{\Delta V}$, where ΔP is the pulse pressure, and ΔV is the change in volume of the *windkessel* corresponding to the given ΔP. Wezler and Böger have equated ΔV to $\frac{V_s}{2}$, where V_s is the stroke volume of the heart. E then becomes $\frac{\Delta P}{\frac{1}{2}V_s}$. In absolute units, E has the dimensions $\frac{\text{dynes}}{\text{cm}^5}$.

W (peripheral resistance) is defined as $\frac{P_m{}^*}{V_s \cdot r/60}$, where P_m is the mean arterial pressure, V_s is the stroke volume as before, and r is the heart rate per minute. The denominator of the ratio is the second-volume of the heart. In absolute units, W has the dimensions $\frac{\text{dynes} \cdot \text{seconds}}{\text{cm}^5}$.

The ratio E/W therefore is equal to $\frac{\Delta P}{\frac{1}{2}V_s} \div \frac{P_m}{V_s \cdot r/60}$, which simplifies to $\frac{r \cdot \Delta P}{30 P_m}$. It should be noticed that V_s cancels out. The constant (30) depends upon the use of Wezler

1 Apéria, A., *Acta Physiologica Scandinavica*, 1941, **2**, 64.

2 Wezler, K., and Böger, A., *Ergeb. Physiol.*, 1939, **41**, 292.

* The venous pressure theoretically should be subtracted from P_m in the numerator of the ratio, but ordinarily may be neglected because of its small magnitude compared with P_m.

TABLE I.
The Effects of Epinephrine upon the Value of E/W. (Data from Grollman,[3] after Euler and Liljestrand.)

	Pulse rate	Blood pressure	Pulse pressure	Mean pressure	E/W
Before injection	60	103/68	35	86	0.82
After inj. of 0.7 cc 1:1000 epinephrine subcut.	66	130/64	56	92	1.34

TABLE II.
The Effects of Pitressin upon the Value of E/W. (Data from Grollman,[3] p. 196.)

	Pulse rate	Blood pressure	Pulse pressure	Mean pressure	E/W
Before injection	67	102/70	32	86	0.83
After inj. of 0.4 cc pitressin intramusc.	64	107/81	26	94	0.59

TABLE III.
The Effects of Pyrotherapy upon the Value of E/W.

Time (min)	Oral temp.	Pulse rate	Blood pressure	Pulse pressure	Mean pressure	E/W
0	36.0	80	162/120	42	141	0.79
40	36.7	88	168/120	48	144	0.98
95	36.8	80	166/120	46	143	0.86
180	38.2	132	170/120	50	145	1.52
210	39.0	144	190/110	80	150	2.56

and Böger's estimate of the relationship $\frac{\Delta V}{V_s} = 0.5$ and will be proportionately altered if other estimates are used. E/W has the dimensions seconds^{-1}, and its value in the normal young adult will approximate unity.

The clinical values of ΔP and P_m in mm mercury may be used directly in the formula instead of the absolute values in dynes/cm^2, since E/W contains the ratio of the two pressures. Thus, in a subject with heart rate of 70, and arterial blood pressure of 110/70, E/W may be computed as follows:

$$r = 70,$$
$$\Delta P = 110 - 70 = 40,$$
$$P_m = \frac{110 + 70}{2} = 90,$$
$$E/W = \frac{r \cdot \Delta P}{30\, P_m} = \frac{70 \cdot 40}{30 \cdot 90} = 1.04.$$

The procedure described involves certain approximations and assumptions, the validity of which seems to be borne out by the observation that E/W, thus computed, in a variety of states varies in the direction which might be expected on the basis of generally accepted physiological concepts, as illustrated below.

[3] Grollman, A., *The Cardiac Output of Man in Health and Disease*, 1932, 179.

1. *The Effects of Epinephrine.* (Table I.)

The data included measurements of cardiac output, which showed a substantial increase after the injection of epinephrine. Grollman, in commenting on the data, said: "The increased cardiac output with the maintenance of a constant mean blood pressure must obviously indicate a decrease in the peripheral resistance. Hence despite its local vasoconstricting effect on arterioles epinephrine apparently causes vasodilatation when administered subcutaneously in man." The rise in the value of E/W fully supports Grollman's inferential reasoning.

2. *The Effects of Pitressin.* (Table II.)

The sharp drop in the value of E/W is consonant with the known vasoconstricting properties of pitressin, *i.e.*, the peripheral resistance is increased.

3. *The Effects of Pyrotherapy.* (Table III.)

The subject was a white woman *aet.* 34, suffering from benign hypertension of more than two years duration. There were minimal fundus changes and negative urine findings.

The marked increase in the value of E/W parallels the vasodilatation caused by the heat therapy, with the resultant decrease in peripheral resistance.

TABLE IV.†
The Change in the Value of E/W with Time Following a Period of Maximal Muscular Exercise.

Time after exercise (min)	Pulse rate	Blood pressure	Pulse pressure	Mean pressure	E/W
0.75	140	151/60	91	105	4.04
1.0	124	149/58	91	103	3.64
1.75	123	150/59	91	104	3.58
2.5	100	152/55	97	103	3.14
3.25	91	141/64	77	102	2.29
4.5	80	130/62	68	96	1.89
6.0	82	122/68	54	95	1.55
10.3	83	121/71	50	96	1.44
13.0	74	119/73	46	96	1.18
16.0	78	119/73	46	96	1.25
22.75	80	114/74	40	94	1.13
31.0	75	118/78	40	98	1.12
44.75	72	115/74	41	94	1.05
54.75	74	117/75	42	96	1.08

4. *The Effects of Muscular Exercise.* (Table IV.)

The subject, a normal young male, climbed 19 meters of stairs as rapidly as possible (about 25 seconds).

The high value of E/W immediately after exercise reveals the extremely low peripheral resistance, and hence the high degree of peripheral vasodilatation (confirming Ogden and Shock†).

The foregoing cases have all been analyzed on the implicit assumption that the value of E was not materially changed. It is clear that the value of E/W will be dependent on the values of both E and W. The exact analysis of cardiovascular dynamics requires determination of the absolute values of E and W, but in many cases, as in the above, it may reasonably be assumed that one of the variables is relatively stable compared with the other.

Summary. 1. A method of computing the ratio of aortic rigidity (or capacity) to peripheral resistance is described, using clinical values of heart rate and blood pressure. 2. The assumptions implicit in the method are discussed. 3. Several types of cardiovascular events are analyzed in terms of this ratio.

† Compiled from data collected by one of us in connection with the Adolescent Study of the Institute of Child Welfare, University of California. The facilities afforded are acknowledged with thanks. PROC. SOC. EXP. BIOL. AND MED., 1935, **33**, 5.