

fed upon liver had increased in length 188 mm. or 83.5%, those fed upon the outer, acidophilic portion of anterior pituitary had increased 125 mm. or 55.5%, while those fed upon the inner, basophilic portion had increased in length 24 mm. or 10.6%. Thus the predominatingly acidophilic portion of the pituitary had manifested a growth-promoting power 44.9% greater than that of the predominatingly basophilic portion. Stating these results in terms of averages, they are as follows: At the beginning of the experiment both groups showed means of $7.50 \pm .07$ with standard deviation .548. At the conclusion of the experiment the group fed the basophilic portion showed a mean of $8.31 \pm .11$ and standard deviation .906, while the group fed the acidophilic portion showed a mean of $11.66 \pm .15$ and a standard deviation of 1.392.

It will be observed, however, that the growth-promoting power of liver exceeded the best pituitary growth by 28%. Whether there is inherently greater power in the liver or whether some factor of antagonism is at work in the pituitary tissue we have yet to determine. The work of Evans and Simpson² indicates an antagonism between the 2 hormones of the anterior pituitary, that promoting growth and that affecting the endocrine system.

Our investigation indicates that the predominatingly acidophilic portion of the anterior pituitary possesses a greater growth-promoting power for planarian worms than the predominatingly basophilic portion. This result was obtained by feeding the fresh gland to normal worms.

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Further Observations on Experimental Aortic Insufficiency. III. Factors Accountable for the Systolic Collapse of the Central Pressure Pulse.

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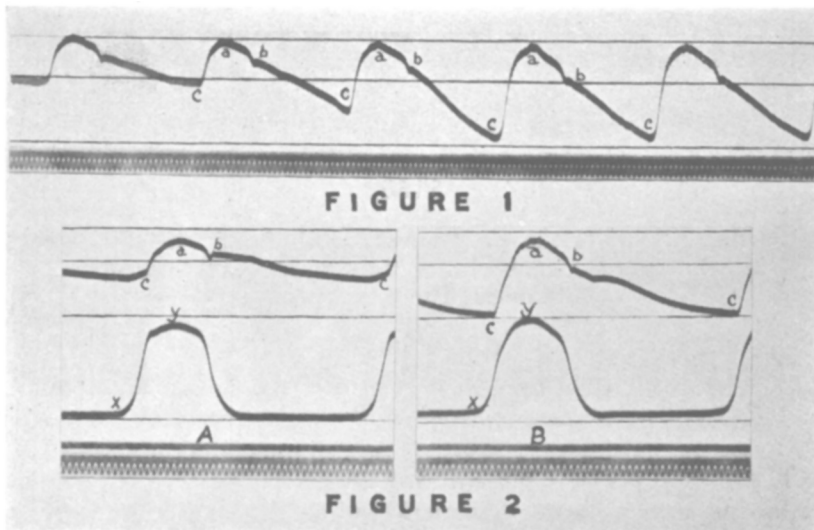
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The arterial pressure pulse during aortic insufficiency is described as having both a "water hammer" and "collapsing" characteristic, the former term referring to the impression given by the abrupt and great systolic rise, the latter, to the tactile and graphic effects of the steep gradient of its decline. Stewart¹ first directed attention to

¹ Stewart, H. A., *Arch. Int. Med.*, 1908, **1**, 102.

the fact that the chief decline of pressure precedes the dicrotic wave in the peripheral pulse and from this drew the conclusion that it must be systolic in time rather than diastolic, as is commonly taught. Subsequent improvements in methods of graphic registration, together with a clearer understanding of the physical changes involved in pulse transmission, have shown that this inference was not justifiable. The writer² found that the diastolic gradient of the central arterial pulse is predominately affected and chiefly concerned in the collapse and lower diastolic pressure. The analyses of Frank³ have shown that the end of systole does not correspond to the rise of the dicrotic wave of the peripheral pulse but coincides with an uncertain and not easily determinable point on that part of the descending slope which precedes the dicrotic wave. Nevertheless, careful inspection of optical records obtained from experimental and clinical valvular insufficiency reveals the fact that the decline of pressure previous to the apex of the V-shaped incisura is also greater (Cf. Figs. 1-2). Though not strictly correct, we may refer to this as the "systolic collapse".

The cause of this phenomenon has not been analyzed. Obviously it can be due to either or both of 2 possible alterations: the systolic pressure maximum may be higher or the incisural apex may occupy a lower position on the pressure curve. A determination of the fac-



² Wiggers, C. J., *Arch. Int. Med.*, 1911, **8**, 17.

³ Frank, O., *Sitzungsberichte der Gesellsch. f. Morphol. u. Physiol. in München*, 1926, **37**, 33.

tor actually concerned is of importance in completing our conceptions of the dynamics of aortic insufficiency; for if the greater systolic pressure drop be due solely to the former it would be adequately accounted for by the increased force of ventricular contraction, but if it be due to an absolute decrease in the position of the incisura a late systolic or very early diastolic loss of pressure must be concerned.

In order to study the question experimentally, intra-aortic and intra-ventricular pressure pulses were recorded optically by means of the author's manometers, and temporary valvular insufficiency was produced by a method previously described.² Sixty-four experiments were made on 5 different dogs, under morphine and barbital anesthesia.

Typical records are reproduced in Figures 1 and 2. A glance at the former establishes the fact that the abrupt post-incisural decline of pressure (b-c) is predominately responsible for the lower diastolic pressure (c) and the larger pulse pressure (c-a). However, the pre-incisural drop (a-b) is also typically greater, due to a lower position of the incisural apex (b). Figure 2 comprises 2 segments of records from another animal. Segment A serves as a normal control, segment B represents the pressure changes shortly after production of an aortic insufficiency. In addition to the features exhibited in Figure 1, this curve shows that the greater pre-incisural drop is due in part to a lower position of the incisural apex (b) and in part to a higher pressure maximum (a). A study of intra-ventricular pressure curves (lower records) shows that this is due to a more vigorous contraction, the initial (x) and the ventricular pressure maximum (y) both being elevated.

An analysis of 64 sets of similar records showed that, as in Figure 1, the greater systolic collapse was due exclusively to a lower position of the incisura in 35 instances. In some of these the systolic pressure maximum remained unaltered, in others it decreased slightly. In 29 experiments, the systolic pressure maximum was elevated in addition, as in Figure 2. In three instances only, the incisural position was unaffected and the greater systolic collapse was due entirely to a higher pressure maximum.

Conclusions. (1) The pronounced systolic fall of pressure which accompanies the steeper diastolic decline characteristic of aortic insufficiency is practically always associated with a lower position of the incisural apex. (2) Therefore, a late systolic or early diastolic loss of pressure from the arterial system must occur as a result of insufficient valves. (3) An elevation of the systolic

pressure maximum may or may not contribute to the greater systolic collapse, depending on the degree to which physiological compensation operates in a particular heart.

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A New Tri-atomic Alcohol from the Urine of Pregnant Women.

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A new alcohol differing from the pregnandiol of Marrian¹ and Butenandt² and also from the crystalline ovarian hormone isolated by Doisy,³ Thayer and Veler, Butenandt,⁴ Laquer⁵ and Marrian⁶ has been obtained from the urine of pregnant women.

This alcohol has been isolated in the form of snow white crystals. The melting point by the open beaker method of 5 different preparations was 273°, 273°, 273°, 272.3°, 272° (uncorrected). The crystals melted sharply without decomposition.

The molecular weight determination by Rast's micro procedure gave an average value of 294. The iodine numbers of 3 different preparations were 85.3, 86.2, and 88.5. The average of these values, 86.7, permits one to calculate a molecular weight of 292.8 if one double bond is assumed.

Determination of the number of hydroxyls in 2 samples by the procedure of Peterson and West⁷ indicates that 3 atoms of oxygen exist in this form. Found 124, 129; theory 129 gms. of $\text{CH}_3\text{C}=\text{O}$ per mole. The average molecular weight of the triacetyl derivative is 410. M. P. 126° uncorrected.

The specific rotation of a 0.322% solution in 95% ethyl alcohol at 28° in a 2 dm. tube with a sodium flame was +68.3°. Another sample (0.148%) gave a value of +72.8°.

¹ Marrian, G. F., *Biochem. J.*, 1929, **23**, 1090.

² Butenandt, A., *Ber. d. deut. chem. Ges.*, 1930, **63**, 659.

³ Doisy, E. A., Thayer, S. A., Veler, C. D., *J. Biol. Chem.*, 1930, **86**, 499.

⁴ Butenandt, A., *Naturwissenschaften*, 1929, **17**, 879.

⁵ Dingemans, E., de Jongh, S. E., Kober, S., and Laquer, E., *Deut. Med. Wochenschr.*, 1930, **56**, 301.

⁶ Marrian, G. F., *Biochem. J.*, 1930, **24**, 435.

⁷ Peterson, V. L., and West, E. S., *J. Biol. Chem.*, 1927, **74**, 379.