

When a certain lower level of pressure was reached, the normal rate was abruptly resumed. We have made electrocardiographic studies of this effect.

In the experiments reported here the typical slowing was produced by the intravenous injection of 5 cc. of 1-10,000 adrenalin into dogs under morphine-nembutal anesthesia. Electrocardiograms were recorded from lead 1. In several experiments the carotid blood pressure was recorded optically along with the electrocardiogram by a Hürthle manometer.

We find that the appearance of a slower rate is due to a pulsus bigeminus (Figs. 1 and 2), consisting of alternate normal beats and beats in which the ventricular contraction is premature. A pulsus alternans is indicated by the carotid blood pressure tracing (Fig. 2) as recorded by the Hürthle manometer.

A bigeminal rhythm due to pressor agents has been observed by Levy² in cats under chloroform and under ether anesthesia, although irregularities due to acute hypertension in vagotomized animals have not been thoroughly investigated. The work of Higgins, Ewing, and McGuigan must therefore be regarded as a confirmation and extension of the studies of Levy. It appears from our studies that the bigeminal rhythm may be obtained under nembutal anesthesia as well as under chloroform and ether and that the major condition necessary for its appearance is a sharply elevated blood pressure.

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Effect of Rapid Transfusion on Duration of Systolic Ejection of the Left Ventricle.*

EDWARD SIGMAN. (Introduced by Louis N. Katz.)

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Katz¹ and Wiggers² have shown that there is an increase in the duration of the systolic ejection phase following intravenous saline infusion. The increase in duration of this period is independent of any heart rate change and was shown to be due to distention of the

² Levy, A. G., *Heart*, 1914, **5**, 299.

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¹ Katz, L. N., *J. Lab. and Clin. Med.*, 1921, **6**, 291.

² Wiggers, C. J., *Am. J. Phys.*, 1921, **56**, 415, 439.

heart. However, the possibility remains that this lengthening of systolic ejection might possibly be due to a change in the osmotic pressure of the blood or to a specific action of the sodium chloride. The present research was undertaken to investigate these possibilities.

The technique of Wiggers and Katz³ for studying the effects of infusions was followed in essentially all details with the following modifications: (1) defibrinated blood was substituted for the isotonic sodium chloride solution, (2) care was taken to keep the temperature of the infused blood the same as that of the animal. Records on bromide paper by optical means were made of the aortic blood pressure and heart volume (Wiggers and Katz³).

Summary tables in the 4 successful experiments were made of the systolic, diastolic and pulse pressures in the aorta, and the systolic, diastolic and stroke volumes of the heart, together with the duration of the systolic ejection and the cycle. A typical protocol is shown in Table I. It will be seen that as the transfusion was continued and the heart distended the duration of systolic ejection increased, although the cycle length remained practically unchanged.

TABLE I.
Experiment VI. Three curves measured.

Defibrinated blood added, cc.	Time from onset of exp., min.	Cycle length, sec.	Duration of systolic ejection, sec.	Blood Pressure			Heart Volume	
				Systole mm. Hg.	Diastolic mm. Hg.	Pulse mm. Hg.	Diastolic, cc.	Stroke, cc.
Control	0	0.41	0.14	135	127	8	y	7
100	1	0.40	0.14	154	141	13	y+3	13
200	2	0.40	0.14	171	156	15	y+8	20
300	3	0.40	0.15	186	164	22	y+10	25
400	4	0.46	0.16	188	161	27	y+10	27
500	5	0.44	0.17	202	171	31	y+18	43
580	9	0.45	0.19	228	172	56	y+20	47

Conclusion. These experiments show that the increase in systolic ejection following transfusion (and in infusion) is due to the distention of the heart.

³ Wiggers, C. J., and Katz, L. N., *Am. J. Phys.*, 1922, **58**, 439.