

for five-day periods for comparison with the controls of the same periods, and all experiments were continued for at least ten days.

The results show that amino acids and their derivatives have but slight effect on the division rate at any period of vitality. The purins and their derivatives have but a slight effect on the division rate when vitality is very low or very high, but a marked effect of increasing the rate when vitality is decreasing (allantoin) or increasing (hypoxanthin, xanthin).

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The first onflow and diastolic waves in the venous pulse.

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Simultaneous records of the contractions of the auricle and ventricle, intraventricular pressure, arterial pulse, and the pulse of the superior vena cava were made.

Previous investigators have not agreed as to the time relations of the 3d positive (Mackenzie's "v") wave, some placing its appearance during ventricular systole, and others believing that it occurs in diastole. This confusion has arisen from the fact that Mackenzie's "v" wave in reality consists of two positive waves which are separate both in time and origin. (Bard described two such waves, but was not definite concerning the time relations.)

The first of these waves has been called the "onflow" wave, and the second, the "diastolic" wave. They are preceded, of course, by the auricular and systolic ("c") waves.

In a series of some fifty dogs, the onflow wave has always appeared just at the end of auricular relaxation, and therefore, during the first half of ventricular systole. The wave is terminated at the very beginning of ventricular diastole. The origin of the wave cannot be ascribed to the passive auricle, nor to the ventricle, the base of which is still moving downward, and which would tend, therefore, to produce a negative, rather than a positive wave. The wave must simply represent the increased pressure

resulting from the onflowing blood, which can now no longer enter the completely relaxed auricle, and must necessarily "back up" into the great veins. The instant ventricular relaxation commences, the base of the ventricle pushes up against the column of blood in the auricle and veins, and thus produces a still greater pressure—*i. e.*, the diastolic rise. In all of the experiments this diastolic rise has occurred synchronous with the beginning of ventricular relaxation, *before* the closure of the semilunar valves. The auricle, of course, is still passive.

The diastolic rise continues until the ventricle has relaxed sufficiently to allow the a-v valves to open, when the increased pressure in the auricle is terminated by the rapid outrush of blood. The resulting diastolic fall then continues until the end of ventricular relaxation, or, in other words, until the ventricle ceases to enlarge and receive the onflowing blood. The blood which cannot enter the ventricle, "backs up" into the auricle and veins, and thus produces the 2d onflow wave (Hirschfelder's "h"), in a manner similar to that in which the 1st onflow wave was originated.

The reason for the non-appearance of the 1st onflow wave in many jugular pulse tracings is that it is partially or completely fused with the diastolic wave. (The point of separation is indicated by a notch in many of Mackenzie's records.) The "a," "s," and "d" waves are essentially impact waves, and therefore travel faster than the onflow wave, which is dependent, for its rate of propagation, upon the rapidity of the onflow of blood from the periphery. The onflow wave appears just after the "s" wave in the lower part of the sup. vena cava, but by the time the effect of the "backing up" of blood has been felt in the veins of the neck, the onflow wave has been overtaken and obscured by the "d" wave.

The above conclusion is supported by figures from several experiments, showing that the onflow wave appeared in the jugular vein .05 second later than in the vena cava. The relative positions of the "a," "s," and "d" waves do not vary in the different regions.