seem to indicate that the theory of isochronism is not valid for the retrolingual preparation.

## 5921

Afferent Impulses from Single End Organs in the Carotid Sinus.\*

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Previous experiments revealed the general character of the discharge in the carotid sinus nerve and its relation to the pulse curve. They showed a large burst of impulses resulting from the rapid rise in pressure at the beginning of systole followed by comparative inactivity during diastole. As the pressure increases the impulses become more continuous, extending throughout the heart cycle.

This work has been much extended and many observations have been made on the discharge of impulses from single end organs in the carotid sinus of the rabbit. Electrodes lead from the exposed nerve to a vacuum tube amplifier and thence to either a loud speaker or a Matthew's oscillograph for photographic registration. The procedure has been to divide the nerve in such a way that only one of the remaining fibers is in connection with a functionally active end organ. It is easy to decide when this stage in the dissection has been reached for either the sound furnished by the loud speaker or the photographic record from the oscillograph reveals a single series of regularly spaced impulses. The pulse curve has been recorded simultaneously by means of a Wiggers' manometer.

A typical record, Fig. 1, shows that with the beginning of the rapid rise in pressure the end organ starts to discharge impulses at a rate of about 55 a second, which then decreases as the pressure falls. The duration of this discharge in the heart cycle varies largely from one preparation to another and is obviously a function of the threshold of the end organ, the mean blood pressure and the form of the pulse curve. The first condition is well illustrated by preparations in which there are 2 or 3 active fibres remaining. In such cases one commonly finds one of the end organs continuing to function later in the cycle than the others.

<sup>\*</sup> The expenses of the research have been in part defrayed by a grant from the Committee on Scientific Research of the American Medical Association.

<sup>&</sup>lt;sup>1</sup> Bronk, D. W., PROC. Soc. EXP. BIOL. AND MED., 1931, 28, 1014.

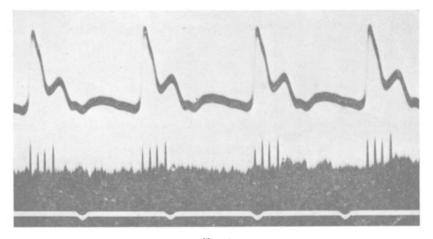


Fig. 1.

Nerve impulses from a single end organ in carotid sinus. Upper record: Blood pressure (mean 63 mm.); middle record: Oscillograph record of impulses; lower record: Time in 1/5 sees.

The effect of increased blood pressure is very definitely to increase the duration of the activity per heart cycle of a single end organ. From experiments in which the mean blood pressure ranged from about 40 mm. Hg. (amyl nitrite) to 150 mm. Hg. (adrenalin) we have obtained records of the corresponding nerve discharges showing no impulses at all, one per systole, then 2, 3, and more as the pressure increased until they were continuous with only slight variations in frequency corresponding to systole and diastole.

In preparations in which several fibres are active this increase in the level of blood pressure shows not only an increase in the frequency and duration of impulses from the individual end organ but also an increase in the number of end organs responding. Starting with a subthreshold pressure at which no endings are stimulated, first one and then another comes into action during systole as their several thresholds are reached. In the normal animal, therefore, as the blood pressure rises more impulses reach the centers in the medulla each heart cycle—as a result of an increased number of functionally active end organs, a longer duration of discharge from each end organ per cycle, and a higher frequency of discharge from the several endings.

Preliminary tests have been made to determine the effect of CO<sub>2</sub> on these receptors. Although the animals have rebreathed 10% CO<sub>2</sub> for over 3 minutes we have only observed a variation in frequency or duration of impulse discharge that could be accounted for merely by the accompanying change in level of blood pressure. This point,

however, as well as the effect of other chemical agents is being more extensively investigated.

## 5922

## Influence of the Hypersensitive State in Experimental Streptococcus Viridans Bacteremia.

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While studying the localization of Streptococcus viridans at sites of local antigen-antibody reactions in rabbits sensitized to horse serum, it was observed that there was a difference in the rate of disappearance of these organisms from the blood stream when injected intravenously into normal animals and animals sensitized to horse serum. Bull¹ observed the fate of bacteria injected into normal dogs and rabbits. Bail² made similar observations, and Hopkins and Parker³ and Wright⁴ studied the fate of bacteria in normal and immunized animals. More recently Boone, Chase and Brink⁵ made some similar observations working with intestinal absorption of B. prodigiosus in dogs during acute anaphylactic shock. There has not been found so far in the literature any work concerning the rate of disappearance of Streptococcus viridans from the bloodstream of hypersensitive rabbits.

A strain of Streptococcus viridans was isolated from an apical abscess of a tooth at necropsy. The hourly growth rate in broth was established. A determination of the quantity (pour plates) of organisms injected was made at the time of inoculation. The volume of the suspension in saline was kept constant at 1 cc. Inoculations were made into the marginal ear vein of the rabbits. Blood cultures, both pour plates and broth, were taken from the femoral vein at intervals of 15 minutes for the first hour, then half hourly for 3 hours, then hourly for 8 hours, and finally every 24 hours until the animal died or until the cultures remained negative.

Three groups of animals were used. Group A, normal animals, received injections of organisms only. Group B, normal animals,

<sup>&</sup>lt;sup>1</sup> Bull, J. Exp. Med., 1914, 20, 237.

<sup>&</sup>lt;sup>2</sup> Bail, Arch. f. Hyg., 1905, 52, 272.

<sup>&</sup>lt;sup>3</sup> Hopkins and Parker, J. Exp. Med., 1918, 27, 1.

<sup>4</sup> Wright, J. Exp. Path. and Bact., 1927, 30, 185.

<sup>&</sup>lt;sup>5</sup> Boone, Chase and Brink, Proc. Soc. Exp. Biol. and Med., 1931, 29, 1.