THE ROLE OF REFRACTORINESS IN THE COLLIDING IMPULSES METHOD

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By the colliding impulses method, it is possible to estimate the total afferent spike flow in a whole nerve, arising from a large number of receptors. This method is first suggested for qualitative determination of afferent impulses from receptors in the aortic arch and skin [6].

The idea of the method is based on the principle of "extinction" of orthodromic and antidromic impulses, resulting from the fact that immediately after excitation, a period of refractoriness develops in the fibers. According to the change in amplitude of the total antidromic potential, it is possible to determine the type of the fibers, the velocity of conduction of impulses along them, and the relative number of fibers along which the afferent flow is conducted from the receptors.

Later the colliding impulses method was modified in order to estimate the frequency spectrum of afferent impulses [1, 2]. The method has a limitation due to the need to choose long distances from the receptors to the stimulating electrodes and also between the stimulating and recording electrodes [1, 3]. For instance, for fibers with high velocities of spread of impulses, the distances from the stimulating electrodes to the receptors required by the method must exceed the real lengths of the nerves.

To clarify and to evaluate essential limitations of the colliding impulses method, we undertook special model experiments.

EXPERIMENTAL METHOD

Experiments were carried out on adult cats anesthetized intramuscularly with hexobarbital (250 mg/kg). The cats were placed on a heated operating table. The cutaneous n. saphenus was dissected at three sites in the hind limb: in the groin, in the region of the knee joint, and in the leg. One pair of stimulating electrodes was placed on the nerve in the region of the inguinal ligament. The nerve was ligated and divided proximally. The second pair of stimulating electrodes was placed on the nerve in the leg. Distally to these electrodes the nerve was again ligated and divided. In the region of the knee joint, recording electrodes were applied to the nerve and connected to a UBP2-03 amplifier (Fig. 1). The stimulating electrodes were connected through isolating attachments to an ÉSU-1 square-pulse generator. The voltage and duration of the stimulating pulses were chosen to be optimal for obtaining evoked potentials of model groups of A_β, A_δ, and C cutaneous nerve fibers of maximal amplitude.

The total number of experiments was 11.

EXPERIMENTAL RESULTS

The number of fibers excited by proximal stimulating electrodes was greater than in the case of stimulation by distal electrodes. The reason is that in the interval between these pairs of electrodes, various nerve branches leave the common nerve trunk, thus correspondingly reducing the number of fibers in the direction from center to periphery. Consequently, the total evoked potential recorded to stimulation by the distal electrodes was lower in amplitude than the potential obtained by excitation of the nerve with the proximal electrodes (Fig. 1a).

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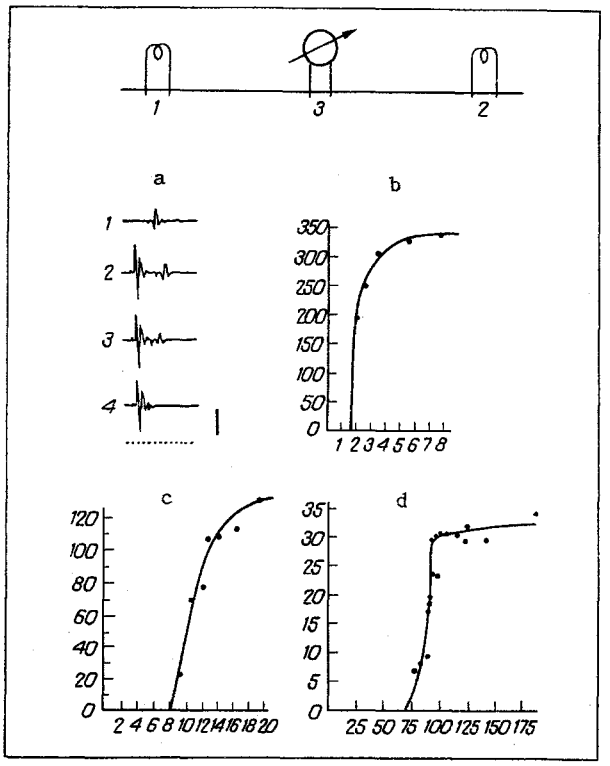

Fig. 1. Arrangement of stimulating (1, 2) and recording (3) electrodes on nerve and change in amplitude of total action potential (AP) of cutaneous nerve fibers as a result of collision between spike trains in a model experiment. a: 1) Total AP of A_β fibers to stimulation of distal end of nerve, 2, 3, 4) total AP of A_β fibers to stimulation of proximal and distal ends of nerve. Stimulation of distal region of nerve delayed relative to stimulation of proximal region by different time intervals (described in text). Calibration: 500 μsec, 500 μV; b) curve showing change in amplitude of total AP of nerve fibers of A_β type depending on time delay between stimulation of nerve by distal and proximal electrodes. Abscissa, duration of delay (in msec); ordinate, amplitude of AP (in μV); c) the same as in b, but for A_δ fibers; d) the same as in b, but for C fibers.

Stimulation applied to the nerve from different pairs of electrodes was retarded relative to one another in order not to allow collision between the evoked approaching impulses. Two total evoked potentials were recorded on the oscilloscope screen: the first from stimulation of the proximal region of the nerve, the second from stimulation of the distal region. The interval between stimulation of the nerve from the different electrode pairs was then shortened so that impulses travelling along the nerve from the stimulating electrodes collided in the region between the distal stimulating and the recording electrodes. If the impulses collided in all fibers excited by the distal electrodes, the amplitude of the total potential fell to zero.

Nerve fibers of different types have different conduction velocities of nervous impulses. Correspondingly, so that impulses in different fibers would collide, with equal distances between the electrodes, different lengths of delay were required between the stimulation. For instance, in our experiments for A_β-fibers, with a distance of 104 mm between the stimulating electrodes, the amplitude of the delayed potential began to decrease after collision with impulses in the opposite direction when the delay time was 3.4 msec, and it reached zero with a delay of 1.7 msec (Fig. 1a). For type A_δ fibers, with the same distance between the stimulating electrodes, reduction of the amplitude of the potential during collisions began with a delay of 16 msec, and with a delay of 8 msec the amplitude of the delayed potential fell to zero (Fig. 1c). For unmyelinated C fibers these delays amounted to 95 and 70 msec, respectively (Fig. 1d). The distance between the stimulating electrodes in the last case was 96 mm.

Depending on the values of the delays and distances between the electrodes which we obtained, the dimensions of the nerve regions in which the impulses could collide were calculated.

For example, in the experiment illustrated in Fig. 1b, the A_β evoked potential travels from the stimulating distal electrodes to the recording electrodes for a distance of L_1 = 54 mm, in 0.73 msec. The conduction velocity v is given by:

$$v = \frac{L_1}{T} = \frac{54}{0{,}73} = 73{,}9 \frac{\text{мм}}{\text{msec}}$$

The absolute refractory phase τ in fibers of this type lasts 0.5 msec on average [4, 5]. The distance which it occupies on the nerve (ΔL) will be:

$$\Delta L = v \cdot T = 73{,}9 \cdot 0{,}5 = 36{,}9 \text{ мм}.$$

The total time in which an evoked potential cannot be obtained from the distal electrodes (T_0) will be:

$$T_0 = T + \tau = 0{,}73 + 0{,}5 = 1{,}23 \text{ msec}$$

In our experiments this time was 1.7 msec (we shall call it T_{01}). Thus the period of inexcitability in the experiment (τ_a) lasted, not 0.5 msec, but longer:

$$\tau_a = T_{01} - T = 1{,}7 - 0{,}73 = 0{,}97 \text{ msec}$$

This period occupies a distance of ΔL_a on the nerve. This is given by: $\Delta La_a = v \cdot \tau_a = 73.9 \cdot 0.97 = 071.68$ mm. For A_δ fibers (see Fig. 1c):

$$L_1 = 54 \text{ мм}, \quad T = 2{,}2 \text{ msec},$$

$$v = \frac{L_1}{T} = \frac{54}{2{,}2} = 24{,}5 \frac{\text{мм}}{\text{msec}}$$

The absolute refractory phase in fibers of this type lasts 1.0 msec on average [4],

$$\Delta L = v \cdot \tau = 24{,}5 \cdot 1 = 24{,}5 \text{ мм},$$

and in that case

$$T_0 = T + \tau = 2{,}2 + 1 = 3{,}2 \text{ msec}$$

In our experiments T_{01} was 8 msec. Hence

$$\tau_a = T_{01} - T = 8 - 2{,}2 = 5{,}8 \text{ msec},$$

but

$$\Delta L_a = v \cdot \tau_a = 24{,}5 \cdot 5{,}8 = 143{,}1 \text{ мм}$$

For C fibers (Fig. 1d):

$$L_1 = 41 \text{ мм}, \quad T = 33 \text{ msec},$$

$$v = \frac{L_1}{T} = \frac{41}{33} = 1{,}2 \frac{\text{мм}}{\text{msec}}$$

The absolute refractory phase in fibers of this type lasts 2 msec on average [4], and in that case

$$\Delta L = v \cdot \tau = 1{,}2 \cdot 2 = 2{,}4 \text{ мм},$$

but

$$T_0 = T + \tau = 33 + 2 = 35 \text{ msec}.$$

In our experiments T_{01} was 70 msec. Hence

$$\tau_a = T_{01} - T = 70 - 33 = 37 \text{ msec}$$
$$\Delta L_a = v \cdot \tau_a = 1{,}2 \cdot 37 = 44{,}4 \text{ мм}$$

The results show that a decrease in the amplitude of the evoked potential as a result of collision takes place not only in the region between the electrodes (L_1). A decrease in amplitude can also be observed when the colliding impulse has already passed the stimulating electrodes for A_β-fibers by 71.7 mm and for A_δ-fibers by 143.1 mm, and for C-fibers by 44.4 mm. This fact is very important for the colliding impulses method, for it increases the probability of collisions between antidromic and orthodromic impulses and increases the distances allowable between stimulating and recording electrodes. In this way the limitations on the use of the colliding impulses method can be reduced. The previous theoretical calculations [1, 3] did not take into account the duration of the refractory state of impulses spreading along the nerve, and considered the impulse to be single point.

Thus model experiments and calculations based on them have widened the scope for use of the method by reducing the limitations imposed by the length of the interelectrode segment and of the whole nerve.

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DISTRIBUTION OF LABELED AMINO ACIDS AND DELTA SLEEP-INDUCING PEPTIDE IN THE BODY AFTER INSTILLATION INTO THE RABBIT CONJUNCTIVA

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Our previous investigations [1, 3] showed that instillation of the regulatory peptides angiotensin-II and delta sleep-inducing peptide (DSIP) into the conjunctival sac of rabbits gives rise to physiological effects similar to those observed when they are administered by the intragastric and intravenous routes. These observations were confirmed by other investigators [2], but the problem of the pathways of spread of oligopeptides in the body and their effects on the various physiological functions when applied to the conjunctiva of the eye remained unsolved.

The aim of this investigation was to study the distribution of individual amino acids and of DSIP, labeled with tritium, when instilled into the conjunctival sac of the rabbit's eye.

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