RECOVERY CYCLE OF CAPSULO-CORTICAL RESPONSES IN THE SOMATOSENSORY AREA IN CATS

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The recovery cycle of the various components of capsulo-cortical responses in somatosensory cortical area I was investigated in acute and chronic experiments on waking cats and on cats anesthetized with ether. A method of computation is suggested which enables the true amplitude of the fast components of the response to be assessed, which is impossible by direct measurement becasuse of their summation with the slow component of the response. The results showed that the recovery cycle of the 3rd and 4th initial fast waves in waking cats differs from the recovery cycle of the first fast wave and the integral positive wave. Ether completely blocks the 3rd and 4th fast waves by stage III_1. The results demonstrate primary inhibition of cortical neurons by ether and they support the hypothesis of the postsynaptic nature of the 3rd and 4th fast waves.

In response to application of a single electrical stimulus to the thalamic relay nucleus or axons of thalamo-cortical neurons an evoked potential on whose initial positive wave several fast waves are superposed arises in the corresponding projection area of the cortex [4, 5, 9, 11, 12]. The nature of these waves is not fully understood. Some workers associate them with the activity of thalamo-cortical axons [5, 9, 11, 12]. According to other workers these waves, except the first, reflect spike discharges of cortical neurons [4, 8, 10, 14, 17]. Investigations of the recovery cycle of the fast waves in waking animals by different workers have yielded somewhat inconsistent results [8, 10, 17].

The object of the present investigation was to study the nature of the fast waves and to make a more detailed analysis of the recovery cycle of the various components of the capsulo-cortical response.

EXPERIMENTAL METHOD

Altogether 15 experiments were carried out on 10 cats under acute and chronic conditions. The experimental method was fully described earlier [2]. The internal capsule was stimulated through a double electrode by square pulses of electric current, 0.1 msec in duration and twice the threshold level of intensity. Evoked potentials were recorded from the screen of a VC-7 oscilloscope by means of a PC-2A (Nihon Kohden) camera. Five responses were superposed on each frame. In 3 experiments under halothane anesthesia the cortex was isolated from the subcortical structures [6] and, in addition, the anterior part of the corpus callosum was divided. After the division the stimulating electrode was inserted horizontally into the white matter above the incision and 3-4 mm below the boundary of the gray matter beneath somatosensory area I. The stages of ether anesthesia were estimated from the EEG [13]. The experimental results were analyzed by statistical methods. To obtain a clearer picture of the results the weighted sliding mean method was used for each interval between stimuli in each stage of anesthesia separately, after which the means were obtained for each point on the graph [3]. The calculations were carried out on the M-220 computer.

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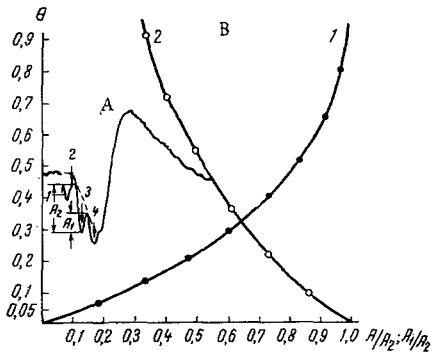

Fig. 1. Capsulo-cortical response (A) and graph for calculating true amplitude of fast waves of the capsulo-cortical response (B). A: arrows 1-4 indicate corresponding fast waves (1-4) of the capsulo-cortical response; broken line denotes slow positive wave of response (after subtraction of the fast waves from the integral response); using the third fast wave as the example the method used for the necessary measurements for subsequent calculation of the true amplitude of the fast wave is illustrated; A_1) amplitude of end of wave (minimum); A_2) amplitude of beginning of wave (maximum); B explanation in text.

Method of Measuring the Amplitude of the Fast Waves*

Since the fast components of the capsulo-cortical response, i.e., the 3rd and 4th fast positive waves (using the nomenclature in [8]), are usually superposed on the slower integral positive wave (Fig. 1A), it is impossible to determine the true amplitude of these fast waves by direct measurement. Accordingly, additional calculations were necessary in order to determine the real amplitude of the fast waves. Let it be assumed that the slow wave $U_1(t)$, where the indentations are located on it, has the form of the straight line:

$$U_1(t) = kt,$$

where t is the time from the beginning of the indentation and k the coefficient of proportionality. Let it further be assumed that in the absence of a slow wave the fast wave $U_2(t)$ is sinusoidal in form, i.e., that

$$U_2(t) = A \sin\left(\frac{\pi}{\tau} t\right), \qquad (a)$$

where A is the true amplitude of the indentation and τ its duration. The equation for the curve recorded by superposition of the fast wave $U_2(t)$ on the slow wave $U_1(t)$ thus has the form

$$U(t) = U_1(t) + U_2(t) = kt + A \sin\left(\frac{\pi}{\tau} t\right). \qquad (b)$$

Let the value t_{max} at which U(t) reaches its maximum be found. By differentiating the equation for U(t) and equating it to zero the following equation is obtained for t_{max}:

$$k + \frac{A\pi}{\tau} \cos\left(\frac{\pi}{\tau} t_{max}\right) = 0, \qquad (c)$$

whence

$$t_{max} = \frac{\tau}{\pi} \arccos\left(-\frac{k\tau}{A\pi}\right). \qquad (d)$$

Next, let an equation be found for A_2 (see Fig. 1A) – the ordinate of the maximum of U(t) measured from the level of the origin of the fast wave. It is evident that

$$A_2 = U(t_{max}) = \frac{k\tau}{\pi} \arccos\left(-\frac{k\tau}{A\pi}\right) + A\sqrt{1 - \left(\frac{k\tau^2}{A\pi}\right)}. \qquad (e)$$

*The method of calculation was suggested by K. Yu. Bogdanov.

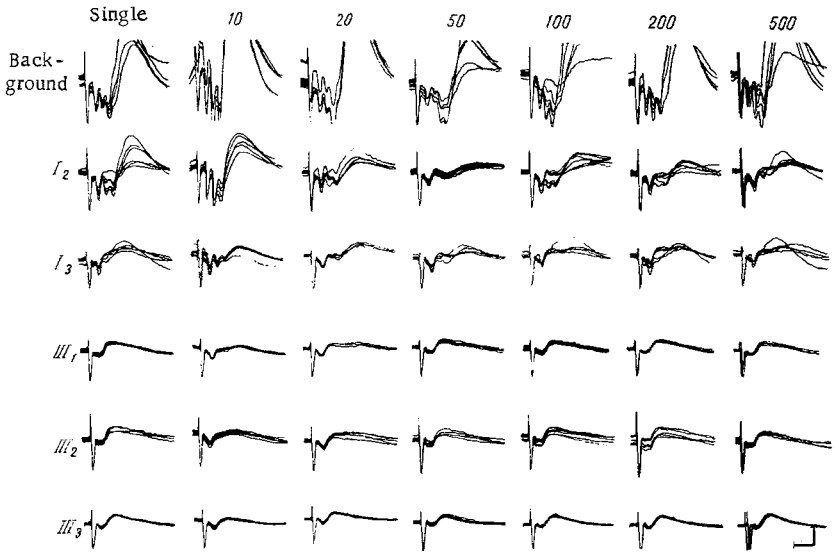

Fig. 2. Effect of ether on evoked potentials in somatosensory cortical area I in response to electrical stimulation of the internal capsule of twice the threshold strength. Horizontal rows represent stages of anesthesia (I_1, I_2, I_3, III_1, III_2, III_3); left vertical row denotes response to single stimulus, other vertical rows denote responses to second stimulus following first after interval indicated by numbers above (in msec). Time marker 5 msec, calibration 100μV. Downward deflection corresponds to positivity.

Consequently,

$$\frac{A_2}{A} = \theta \arccos(-\theta) + \sqrt{1-\theta^2}, \text{ where } \theta = \frac{k\tau}{\pi A} \tag{1}$$

Evidently A_1, the ordinate of the end of the fast wave in Fig. 1A measured from the level of its origin, is given by $A_1 = k\tau = \pi A\theta$, and accordingly

$$\frac{A_1}{A_2} = \frac{\pi\theta}{\theta \arccos(-\theta) + \sqrt{1-\theta^2}}. \tag{2}$$

A convenient method of calculating the real amplitude of A thus follows from Eqs. (1) and (2). Actually, by determining the value of A_1/A_2 from the experimental curve, it is possible to find the value of θ from Eq. (2) or from the corresponding graduated curve 1 in Fig. 1B, and by substituting it into Eq. (1) (or transferring this value to curve 2), the ratio A/A_2 can be found along the abscissa. Knowing the value of A_2 the true amplitude A of the fast wave can be found.

The calculation of the real amplitude of the fast wave superposed on the slower wave is thus reduced to the following stages: a) determination of the values of A_1 and A_2 (Fig. 1A); b) obtaining the ratio A_1/A_2; c) from the value of A_1/A_2 the corresponding value of the parameter θ is determined by curve 1 (Fig. 1B); d) from the value of θ thus found, the value of A/A_2 is obtained from the abscissa of curve 2; e) the required amplitude of the fast wave A is equal to the ratio A/A_2 thus found, multiplied by A_2.

EXPERIMENTAL RESULTS AND DISCUSSION

The characteristics of the response in somatosensory cortical area I of the waking cat to single electrical stimulation of the internal capsule are illustrated in Fig. 1A and Fig. 2 and they do not differ from those described in the literature. Changes in the capsulo-cortical response to double stimulation in the waking animal and at different stages of ether anesthesia are shown in Figs. 2 and 3. The data for the dy-

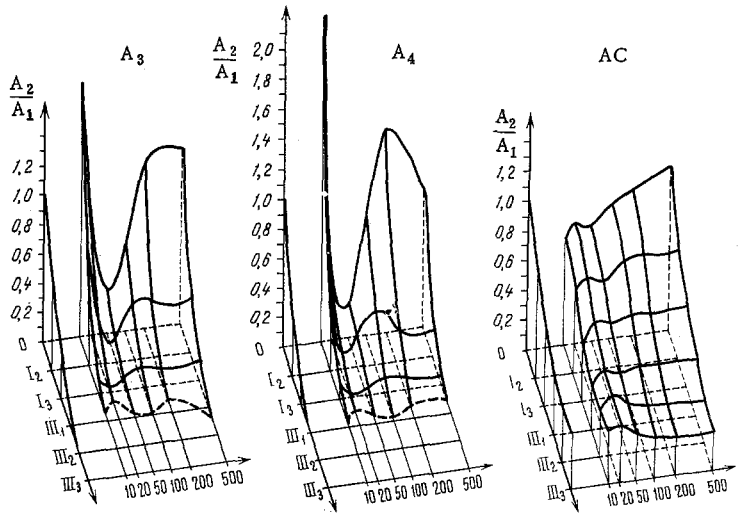

Fig. 3. Effect of ether on amplitude of integral positive wave of the capsulo-cortical response (AC) and on amplitude of fast – 3rd (A_3) and 4th (A_4) – waves of response (stimulation of twice threshold strength). Along the three axes: amplitude of response (relative to amplitude of response to single stimulus in waking animal), stages of anesthesia, and intervals between stimuli (in msec).

namics of the first fast wave are not shown on the graph because of the very small changes in this component, and the second wave was not analyzed because of its inconstancy and low amplitude. The sharp increase in amplitude of the third and, in particular, the fourth fast waves of the waking animal corresponding to an interval of 10 msec, namely by 69% ($P > 0.99$) and by 161% ($P > 0.99$) respectively, is most interesting and has not been described by other authors. A similar increase was found in the experiments on the cortex with its connections with the subcortical structures severed. It must be emphasized that there was no change in the form of the response under these circumstances. Although, because of the small number of experiments with cortical isolation, the results were not subjected to statistical analysis the impression was obtained that facilitation for short intervals between stimuli was more marked in the isolated hemisphere than in the insect cortex. This increase, which evidently originated in the cortex itself, could be due either to an increase in the amplitude of responses in the presynaptic terminals [5, 9, 11, 12] or to facilitation of synaptic transmission and an increase in the number of synchronously discharging cortical neurons [4, 8, 10, 14, 17].

There are several arguments in support of the second hypothesis. First, the amplitude of the first fast wave, which undoubtedly reflects activity of presynaptic axons, was not appreciably changed. Second, ether anesthesia leads to rapid inhibition of the 3rd and 4th fast waves but does not affect the first fast wave and the relatively small changes in the slow components of the response reflecting the appearance of a post-synaptic potential (Fig. 3). It is unlikely that the slow components of the response remain after conduction in the presynaptic terminals is blocked (assuming that the 3rd and 4th waves reflect the activity of these terminals). Third, the high sensitivity of the 3rd and 4th waves to the action of ether is in good agreement with the high sensitivity of cortical unit activity to the action of this substance described previously [16]. Fourth, if the 3rd and 4th fast waves reflected the activity of slowly conducting (unmyelinized) fibers their inhibition ought to be accompanied by an increase in the latent period of these waves, which was not found in the present experiments.

It can be concluded from the above considerations that the 3rd and 4th fast waves, in agreement with views expressed by other workers [4, 8, 10, 14, 17], reflect synchronous discharges of cortical neurons. The character of the effect of ether on the capsulo-cortical responses suggests that from the beginning of its inhalation, even before the appearance of visible behavorial changes (as we found in the chronic experiments) or of changes in the EEG, ether inhibits excitability of the cortical neurons. This effect is evidently not mediated through other structures for, in particular, the excitability of the reticular formation is not notably disturbed during ether anesthesia [1], and stages I and II are actually accompanied by strengthening of ascending reticular influences [15]. It can be postulated that the concrete mechanism of the effect of ether

on cortical neurons is an increase in the critical level of membrane depolarization, for the presynaptic terminals and the mechanism of generation of the excitatory postsynaptic potential are probably less sensitive to the action of this substance. Support for this view is given by the absence of change in amplitude of the first fast wave and the comparatively slight degree of inhibition of the integral positive wave of the response during ether anesthesia.

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