Effect of Antibrain Synaptosomal Fraction Serum and Complement on Evoked Potentials and Impedance

Antibrain antibodies modify the ultrastructure and the electrical properties of the nervous tissue cells[1-6]. In the present note, we report the effect of anti-synaptosomal fraction serum and 'complement' (guinea-pig serum) on the EEG, flash evoked potential (EP) and impedance. The study was performed in 15 alert cats with chronically implanted cannulae and bipolar concentric electrodes in the lateral geniculate body (LGB). The electrodes and 22-gauge stainless steel cannulae were attached together so that their tips were at the same height, but 1–2 mm apart (as described previously[7]). The EEG samples were subjected to spectral analysis and groups of 50 EP were averaged. Impedance was measured at 1 kHz by a Wheatstone bridge technique using current levels of 10^{-13} A/μ^2 at the electrode surfaces[8].

Antisera were prepared to rat cerebral synaptosomal fractions isolated from iso-osmotic Ficoll-sucrose gradients[9]. When assayed with homologous antigen by microcomplement fixation[10], the antiserum used in these experiments had a titer of 1:8000. At this dilution the antiserum reacted specifically with rat cerebral synaptosomal membranes, but not with particulate preparations of other rat organs, or with rat brain nuclei, mitochondria, soluble protein, or synaptic vesicles[11]. The antiserum to rat synaptosomes cross reacted extensively with cat synaptosomal preparations. An 'index of dissimilarity'[12] of 2.12 was determined for this cross reaction[11]. The chemical nature of the complement fixing antigens has not yet been determined.

Figure 1 shows the time course of the effects of anti-synaptosomal fraction serum on the impedance and on the EP when 1 µl was introduced through the cannula into the LGB. A small increase in the EP and in the impedance was observed within 10 min. After 1–2 days the values return to the control. Similar results were obtained in 5 other experiments. Control experiments with an absorbed anti-synaptosomal fraction, an antimyosin serum or with artificial cerebrospinal fluid do not show any change in the EP or impedance.

Figure 2 shows the effect of the complement on the impedance and EP. The EP dropped quickly reaching the minimum value within 10 min. The impedance also dropped reaching a minimum value in 24 h. An almost complete recovery was observed for impedance and a partial one for EP after 3 to 4 days. Similar results were obtained in 7 out of 8 experiments.

Following the introduction of the complement into the LG, changes of the spontaneous EEG also appeared. Figure 3 shows that 1 µl of guinea-pig serum (complement) induces a diminution of the spectral density of the EEG from LG at 1–2 Hz and 6–14 Hz. Those changes appeared within 10 min and lasted for 3–4 days. Introduction of heat-denatured guinea-pig serum was not followed by any change in EEG, EP or impedance.

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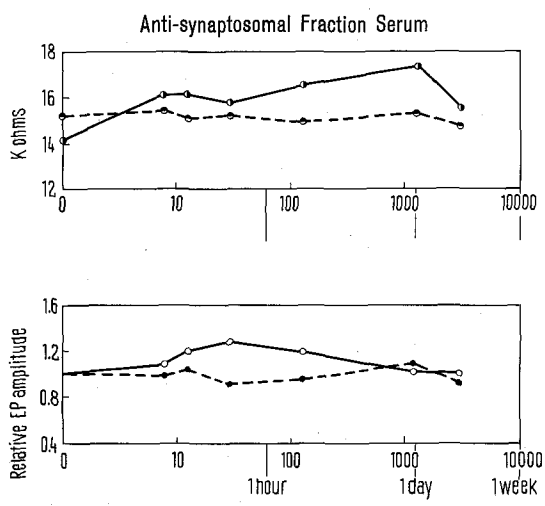

Fig. 1. Effect of anti-synaptosomal fraction serum on impedance (upper part) and flash evoked potential amplitude (lower part) after 1 µl anti-synaptosomal fraction serum was introduced into one lateral geniculate body of a cat (open symbols, solid line). The other lateral geniculate body was not disturbed and was used as a control (filled symbols, dashed line).

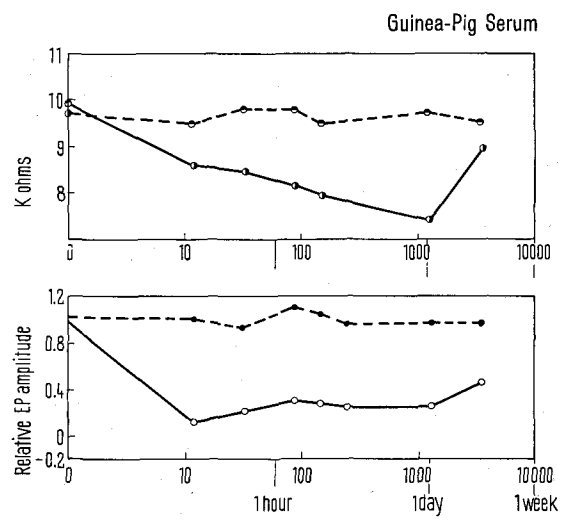

Fig. 2. Effect of the complement (guinea-pig serum) on impedance (upper part) and flash evoked potential amplitude (lower part). 1 µ guinea-pig serum was introduced into the lateral geniculate body of a cat (open symbols, solid line). The other lateral geniculate body was not injected and was used as a control (filled symbols, dashed line).

The effects described for the complement were not observed if the guinea-pig serum was introduced 1 h to 24 h following anti-synaptosomal fraction serum.

An anti-myosin serum or an absorbed anti-synaptosomal fraction did not prevent the changes induced by the complement.

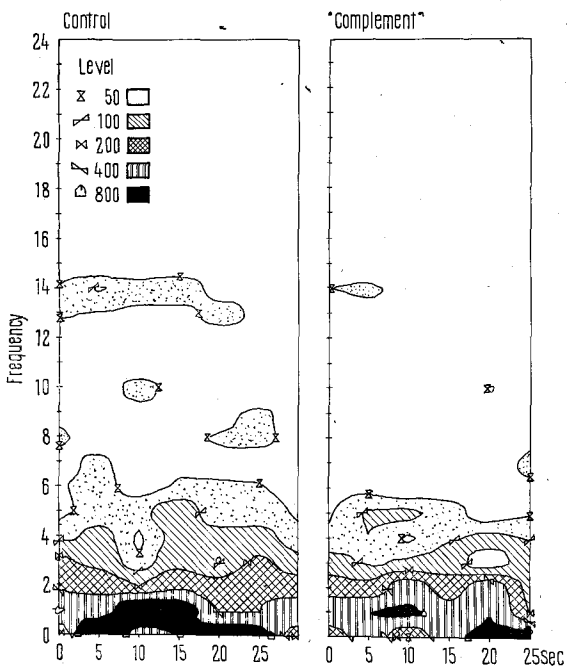

Fig. 3. Contour maps of serial autospectra of the EEG from the lateral geniculate body before (control) and 1 h after application of the complement. 6 consecutive EEG epochs of 5 sec have been combined in each contour map. These maps display in 3 dimension the power of each frequency. The abcissa is the time axis; the ordinate is the frequency axis and the shading represents power. The range for each shading is in $\mu V^2/Hz$; the darker shading represents higher power, the lighter shading corresponds to lower power.

The results suggest that the anti-synaptosomal serum has some specificity in producing changes in the impedance and EP as well as in preventing those elicited by the 'complement'. The usual role of the 'complement' is to cause lysis of the cells that have bound antibodies. If this were happening in the brain, one would expect a drop of impedance after antibodies and complement, but not a drop after 'complement' alone. Our observations are inconsistent with this simple model. Further studies of the changes in the ultrastructure are necessary in order to answer the question of whether those changes in EP and in the conductance of the extracellular space are determined by the state of its structural elements, including interdependent factors such as hydration and divalent cation binding[13], as well as by changes in its cross sectional area[14].

Résumé. L'introduction du «complément» (sérum de cobaye) dans le corps genouillé latéral du chat, produit une diminution des potentiels évoqués photiques et de l'impédance cérébrale. Ces effets sont prévenus par l'application locale d'un anti-serum réagissant spécifiquement avec les synaptosomes cérébraux.

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Tissue Ca Content of Intestinal Smooth Muscles and the Ca^{++}-Concentration of the Incubation Medium

Reports on the tissue Ca content of smooth muscles have supplied non-consistent values (for review see[1]). To see if this could be due to different experimental conditions, i.e. varying (Ca^{++}) of the incubation medium, smooth muscle pieces have been incubated in Tyrode solution of varying $[Ca^{++}]$ and the changes in tissue Ca content after stepwise changes of the $[Ca^{++}]$ of Tyrode solution have been determined.

The experiments were performed using isolated longitudinal muscle strips from guinea-pig ileum[2,3]. The strips were incubated at 30 °C in Tyrode solution of the following composition (m*M*): NaCl 137, KCl 2.7, $MgCl_2$ 1.0, $NaHCO_3$ 12, NaH_2PO_4 0.2, glucose 5.5 and $CaCl_2$ as indicated. The extracellular space amounted to 0.45 per 1.0 g wet wt. of muscle as determined by means of ^{14}C-sucrose. The wet weight of the smooth muscle pieces ranged from 20 to 50 mg. The Ca was determined by a fluorophotometric method according to ZEPF[4].

In the Table the equilibrium values for the tissue Ca content are summarized, as determined after 2 h of incubation in Tyrode solutions of various $[Ca^{++}]$. The tissue Ca content rose from 0.14 to 3.9 mmol/kg wet wt. by rising the $[Ca^{++}]$ of the Tyrode solution from 0.0 to 2.7 m*M*, whereas the ratio $Ca_{cell.} : Ca_{Tyr.}$ decreased simultaneously.

The adaptation of the tissue Ca upon a new $[Ca^{++}]$ of the Tyrode solution is depicted in the Figure. Both after increasing or decreasing the $[Ca^{++}]$, the new equilibrium of the tissue Ca content was reached in about 20 min. Taking into account the extracellular space, the half life time of the cellular adaptations process was calculated to be about 5 min for all conditions studied, which is similar as that found in atrial tissue[5].

The results reported clearly demonstrate the dependence of the tissue Ca content of smooth muscles on the $[Ca^{++}]$

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