blood cells. *Eperythrozoon wenyoni* was not observed to occur in connection with the reactions reported here.

The appearance presented by the discovered particles, as well as their manifestation in the affected animals during a limited period, their absence in the control animals and the positive result of subinoculation speak in favour of their being animate organisms. Reports can be found in the literature on interaction of platelets and some microorganisms, especially myxoviruses and mycobacteria. However, platelets are not the only type of cells affected by these agents. The agent concerned here may turn out to be the first recognized specific parasite of platelets.

Studies for further characterization of the agent are in progress. The first electron micrographs made in their course indicate resemblance to the eperythrozoon organisms[1].

*Zusammenfassung,* Bei Blutausstrichen (Giemsa-Färbung) eines splenektomierten Kalbes wurden an Thrombozyten angelagerte Partikel wahrgenommen, die Eperythrozoon glichen. Subinokulation eines zweiten splenektomierten Kalbes ergab dasselbe Erscheinungsbild. Es wird vermutet, dass diese Partikel einen bisher nicht beschriebenen Mikroorganismus darstellen.

J. Tuomi

*Department of Anatomy and Embryology, College of Veterinary Medicine and Department of Virology, University of Helsinki, Helsinki (Finland), March 1, 1966.*

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## Effect of Experimental Allergic Encephalomyelitis γ-Globulin Upon the Electrical Activity of the Brain

The experimental demyelinating disease is generally believed to be due to delayed hypersensitivity[1,2]. Paterson[3] has suggested that the circulating antibrain antibodies may play an important role in the pathogenesis of demyelination. Janković et al.[4] demonstrated recently that experimental allergic encephalomyelitis (EAE) can be passively produced by administration of serum from guinea-pigs with EAE directly into the lateral ventricle of the brain. In order to ascertain the activity of antibrain antibodies produced by diseased animals in a more delicate manner, the present experiment was undertaken to investigate electrical phenomena in various brain regions following the administration of EAE γ-globulin.

1 g of fresh rabbit spinal cord was homogenized in complete Freund's adjuvant, and chinchilla rabbits were injected with 0.4 ml of spinal cord-adjuvant mixture in the toe-pad of all 4 legs. The rabbits with clinical symptoms were bled. The sera were heated at 56 °C for 20 min and pooled, and γ-globulin fraction (EAE γ-globulin) was isolated[5] and lyophilized. γ-globulin from normal rabbit sera (normal γ-globulin) was prepared in a similar way. The pool of immune sera contained complement-fixing[6] and precipitating antibodies, while the pool of normal rabbit sera did not exert any antibody activity. Lyophilized normal and EAE γ-globulins were dissolved in distilled water (4.4–5.6 mg of protein per 0.2 ml). Gortical and deep electrodes were implanted[7] bilaterally into the frontal cortex, occipital cortex and caudate nucleus, and into the left hippocampus and left septum of normal rabbits[8,9]. The positions of the electrodes were verified histologically[10]. Cannula was inserted into the right lateral ventricle[11]. Animals were treated for 10 consecutive days with 0.2 ml of saline, and after this treatment 5 rabbits received through the cannula a single injection of 0.2 ml of EAE γ-globulin, and 4 animals were injected with 0.2 ml of normal γ-globulin. Electrical activity was recorded prior to the injection, immediately thereafter, and after 2, 3, 4, 5, 12 and 24 h.

The administration of EAE γ-globulin into the lateral ventricle of rabbit brain (Figure) caused a typical electroencephalographic inactivation: the appearance of high voltage slow activity in the frontal and occipital cortex, and acudate nucleus, and disorganization and irregularity in hypocampal θ-rhythm (rabbit No. 39). The high voltage slow waves in the frontal cortex and caudate nucleus were almost synchronous, but this activity was interrupted from time to time with short periods of faster rhythm which was similar to that seen before the injection. These bioelectrical abnormalities lasted for 3–4 h and were followed by corresponding behavioural inactivation. On the other hand, a single injection of normal γ-globulin, and multiple injections of saline, did not produce any apparent changes in electrical activity (rabbit No. 42).

The present results are in accordance with the reversible alterations in evoked bioelectrical responses of cultured cerebral cortex which occurred when the sera from rabbits with EAE and from patients with multiple sclerosis were added to the tissue culture medium[12]. In

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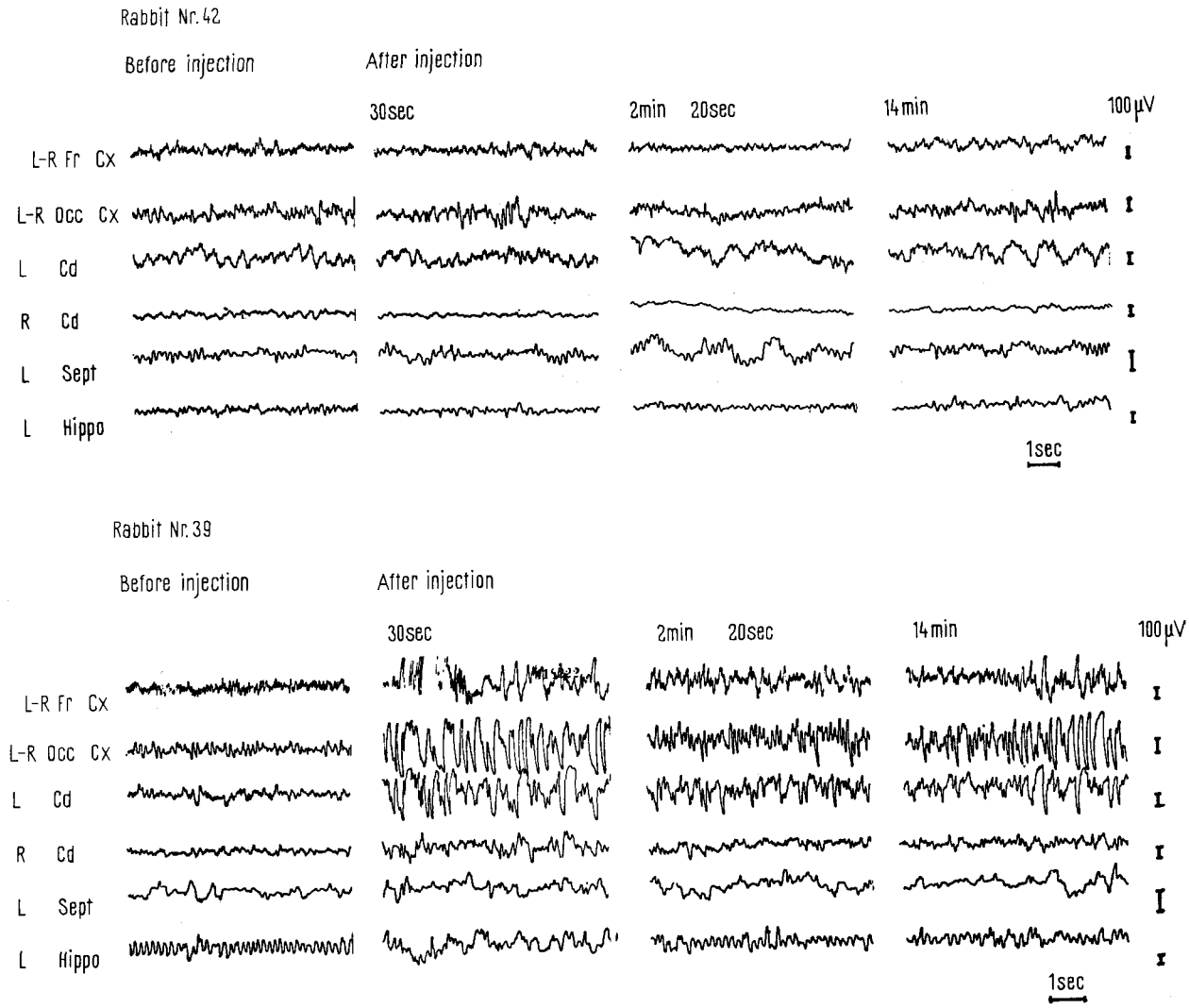


Rabbit No. 42 injected with normal rabbit $\gamma$-globulin. Records made before and after injection. Rabbit No. 39 injected with experimental allergic encephalomyelitis $\gamma$-globulin. Records taken prior and after EAE $\gamma$-globulin application. L = left; R = right; Fr Cx = frontal cortex; Occ Cx = occipital cortex; Cd = caudate nucleus; Sept = septum; Hippo = hippocampus.

addition, we reported earlier that antisera produced in rabbits against antigens from cat brain would affect the electrical response of the brain when introduced into the cerebral cavity[13]. It has also been shown by fluorescent antibody technique that the EAE globulins locate on neurological cell membrane and myelin sheaths[14]. Since Bornstein and Appel[14] demonstrated that sera from animals with EAE and patients suffering from multiple sclerosis are capable of producing demyelination of cultured fragments of central nervous tissue, one may speculate that EAE and multiple sclerosis occur when encephalogenic antibodies combine with some antigens in the central nervous system[15]. Although the present study does not offer evidence about the nature of the antibody responsible for bioelectrical abnormalities, and ordinary histological examination of nervous tissue of rabbits injected with EAE $\gamma$-globulin did not reveal any obvious changes when performed 24 h following the injection, it may be assumed that the effects exerted by EAE $\gamma$-globulin were most likely due to the activity of cytotoxic antibody[16]. All these data together strongly suggest that the activity of antibrain antibodies must be taken into account in any serious consideration of the pathogenesis of experimental demyelinating disease[17].

*Zusammenfassung.* Die direkte Wirkung eines $\gamma$-Globulin-Präparates aus Kaninchenserum (Injektion in laterale Hirnkammer des Kaninchens) auf die experimentelle allergische Encephylomyelitis wurde dargestellt und elektroencephalographisch eine Inaktivierung verschiedener Hirnregionen festgestellt.

B. D. Janković, L. M. Rakić, M. Janjić, J. Ivanuš, and K. Mitrović

*Microbiological Institute, Faculty of Pharmacy, and Biochemical Institute, Faculty of Medicine, University of Belgrade, and Immunology and Neurophysiology Units, Institute for Biological Research, Belgrade (Yugoslavia), March 22, 1966.*

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